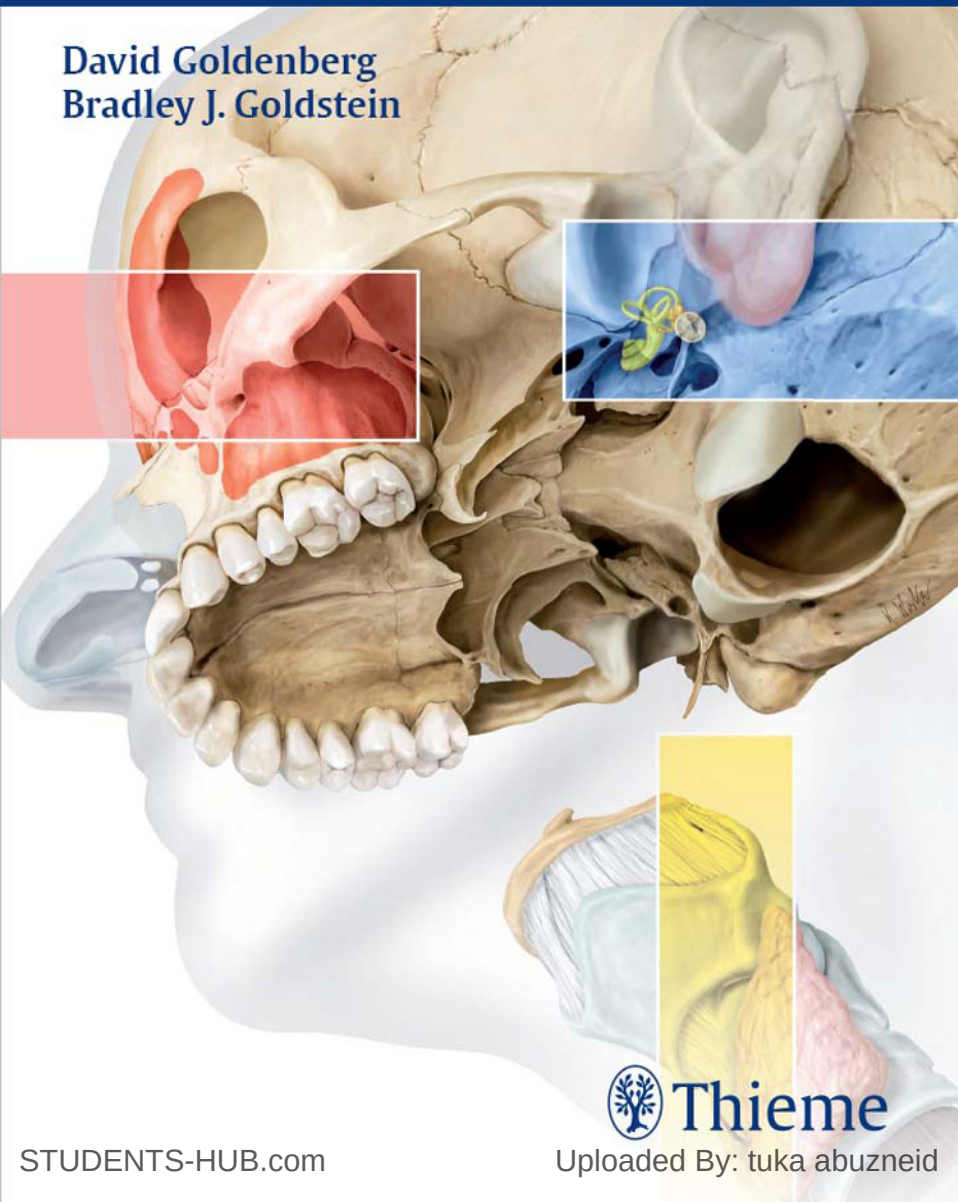
Handbook of Otolaryngology

Head and Neck Surgery

David Goldenberg
Bradley J. Goldstein



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Section 1 Perioperative Care and General Otolaryngology

Section 2 Otology

Section 3 Rhinology

Section 4 Laryngology and the Upper Aerodigestive Tract

Section 5 Head and Neck

Section 6 Pediatric Otolaryngology

Section 7 Facial Plastic and Reconstructive Surgery

Appendices

Index

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Handbook of Otolaryngology

Head and Neck Surgery

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To my parents for their support; to my wife, Renee Flax-Goldenberg, MD, who has been an invaluable partner in my life; and to my beloved children, Michael, Ellie, and Dana.

—David Goldenberg, MD, FACS

To my wife, Liz, and to my children, Ben and Eva, for their endless support, encouragement, and love. Also, thanks to my father-in-law, Thomas R. Van De Water, and my father, Jerome C. Goldstein, for guidance and mentorship from two of the best.

—Bradley J. Goldstein, MD, PhD, FACS

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Contents

Foreword by Charles W. Cummings xv
Preface xvii
Acknowledgments xix
Contributors xxi
Board of Review xxvii

Section 1 Perioperative Care and General Otolaryngology 1
1.0 Approach to the Otolaryngology–Head and Neck Surgery Patient 3
1.1 Preoperative Assessment 5
1.2 Airway Assessment and Management 7
1.3 Diagnostic Imaging of the Head and Neck 22
1.4 Anesthesia 29
1.4.1 Principles of Anesthesia 29
1.4.2 Regional Anesthesia Techniques 32
1.4.3 Anesthesia Drugs 36
1.4.4 Anesthetic Emergencies 47
1.5 Fluids and Electrolytes 48
1.6 Hematology for the Otolaryngologist 50
1.7 Common Postoperative Problems 58
1.8 General Otolaryngology 65
1.8.1 Obstructive Sleep Apnea 65
1.8.2 Odontogenic Disorders 69
1.8.3 Benign Oral Pathology 71
1.8.4 Temporomandibular Joint Disorders 73
1.8.5 Geriatric Otolaryngology 77
1.8.6 Lasers in Otolaryngology 82
1.8.7 Robotic-Assisted Head and Neck Surgery 86
1.8.8 Complementary and Alternative ENT Medicine 89

Section 2 Otology 93
2.0 Embryology and Anatomy of the Ear 95
2.1 Otologic Emergencies 102
2.1.1 Sudden Hearing Loss 102

STUDENTS-HUB.com Uploaded By: tuka abuzneid

2.1.2 Ear and Temporal Bone Trauma 104
2.1.3 Acute Facial Paresis and Paralysis 109
2.1.4 Ear Foreign Bodies 114
2.2 Otitis Media 116
2.2.1 Acute Otitis Media 116
2.2.2 Chronic Otitis Media 121
2.2.3 Complications of Acute and Chronic Otitis Media 127
2.2.4 Cholesteatoma 135
2.3 Otitis Externa 141
2.3.1 Uncomplicated Otitis Externa 141
2.3.2 Malignant Otitis Externa 145
2.4 Audiology 149
2.4.1 Basic Audiologic Assessments 149
2.4.2 Pediatric Audiologic Assessments 155
2.4.3 Objective/Electrophysiologic Audiologic Assessments 157
2.5 Hearing Loss 159
2.5.1 Conductive Hearing Loss 159
2.5.2 Sensorineural Hearing Loss 163
2.5.3 Hearing Aids 168
2.5.4 Cochlear Implants 171
2.5.5 Other Implantable Hearing Devices 174
2.6 Vertigo 176
2.6.1 Balance Assessment 176
2.6.2 Benign Paroxysmal Positional Vertigo 179
2.6.3 Ménière Disease 183
2.6.4 Vestibular Neuritis 186
2.6.5 Migraine-Associated Vertigo 189
2.7 Tinnitus 193
2.8 Cerebellopontine Angle Tumors 195
2.9 Superior Semicircular Canal Dehiscence Syndrome 201
2.10 Otologic Manifestations of Systemic Diseases 204

Section 3 Rhinology 209
3.0 Anatomy and Physiology of the Nose and Paranasal Sinuses 211
3.1 Rhinologic Emergencies 215
3.1.1 Acute Invasive Fungal Rhinosinusitis 215
3.1.2 Orbital Complications of Sinusitis 219
3.1.3 Intracranial Complications of Sinusitis 222
3.1.4 Cerebrospinal Fluid Rhinorrhea 225
3.1.5 Epistaxis 229
3.2 Rhinosinusitis 233
3.2.1 Acute Rhinosinusitis 233
3.2.2 Chronic Rhinosinusitis 237
3.3 Rhinitis 243
3.3.1 Nonallergic Rhinitis 243
3.3.2 Allergy 247
3.4 Inverted Papillomas 250
3.5 Anosmia and Other Olfactory Disorders 254

STUDENTS-HUB.com Uploaded By: tuka abuzneid

3.6	Taste Disorders	257
3.7	Rhinologic Manifestations of Systemic Diseases	259
Section 4	**Laryngology and the Upper Aerodigestive Tract**	**263**
4.0	Anatomy and Physiology of the Upper Aerodigestive Tract	265
4.1	Laryngeal and Esophageal Emergencies	272
4.1.1	Stridor	272
4.1.2	Laryngeal Fractures	275
4.1.3	Caustic Ingestion	278
4.1.4	Laryngeal Infections	280
4.2	Neurolaryngology	285
4.3	Voice Disorders	291
4.3.1	Papillomatosis	291
4.3.2	Vocal Fold Cysts, Nodules, and Polyps	295
4.3.3	Vocal Fold Motion Impairment	297
4.3.4	Voice Rehabilitation	300
4.4	Swallowing Disorders	305
4.4.1	Zenker Diverticulum	305
4.4.2	Dysphagia	308
4.4.3	Aspiration	311
4.5	Acid-Reflux Disorders	317
4.6	Laryngeal Manifestations of Systemic Diseases	319
Section 5	**Head and Neck**	**333**
5.1	Anatomy of the Neck	325
5.1.1	Neck Emergencies	328
5.1.1.1	Necrotizing Soft Tissue Infections of the Head and Neck	328
5.1.1.2	Ludwig Angina	331
5.1.1.3	Deep Neck Infections	333
5.1.1.4	Neck Trauma	337
5.1.2	Approach to Neck Masses	341
5.2	Head and Neck Cancer	346
5.2.1	Chemotherapy for Head and Neck Cancer	352
5.2.2	Radiotherapy for Head and Neck Cancer	355
5.2.3	Sinonasal Cancer	359
5.2.4	Nasopharyngeal Cancer	364
5.2.5	Oral Cavity Cancer	369
5.2.6	Oropharyngeal Cancer	378
5.2.7	Hypopharyngeal Cancer	383
5.2.8	Laryngeal Cancer	388
5.2.9	Dysphonia and Aphonia After Laryngectomy	398
5.2.10	Referred Otalgia in Head and Neck Disease	401
5.2.11	Neck Dissection	403
5.2.12	Skin Cancer of the Head, Face, and Neck	407
5.2.12.1	Basal Cell Carcinoma	407
5.2.12.2	Squamous Cell Carcinoma	411
5.2.12.3	Melanomas of the Head and Neck	416

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5.2.13 Malignant Neoplasms of the Ear and Temporal Bone 424
5.2.14 Lymphomas of the Head and Neck 428
5.2.15 Idiopathic Midline Destructive Disease 434
5.2.16 Paragangliomas of the Head and Neck 437
5.2.17 Peripheral Nerve Sheath Tumors 439
5.3 The Salivary Glands 442
5.3.1 Salivary Gland Disease 442
5.3.2 Benign Salivary Gland Tumors 447
5.3.3 Malignant Salivary Gland Tumors 451
5.4 Endocrine Surgery of the Head and Neck 458
5.4.0 Embryology and Anatomy of the Thyroid Gland 458
5.4.1 Physiology of the Thyroid Gland 461
5.4.2 Thyroid Evaluation 464
5.4.3 Thyroid Nodules and Cysts 467
5.4.4 Hyperthyroidism 470
5.4.5 Hypothyroidism 474
5.4.6 Thyroid Storm 478
5.4.7 Thyroiditis 479
5.4.8 Thyroid Cancer 483
5.4.9 Embryology, Anatomy, and Physiology of the Parathyroid Glands 494
5.4.10 Hyperparathyroidism 495
5.4.11 Hypoparathyroidism 500
5.4.12 Calcium Disorders 501

Section 6 Pediatric Otolaryngology 505
6.1 Pediatric Airway Evaluation and Management 507
6.2 Laryngomalacia 511
6.3 Bilateral Vocal Fold Paralysis 514
6.4 Laryngeal Clefts 517
6.5 Tracheoesophageal Fistula and Esophageal Atresia 521
6.6 Vascular Rings 525
6.7 Subglottic Stenosis 529
6.8 Pierre Robin Sequence 533
6.9 Genetics and Syndromes 537
6.10 Diseases of the Adenoids and Palatine Tonsils 545
6.10.1 Adenotonsillitis 545
6.10.2 Adenotonsillar Hypertrophy 549
6.11 Pediatric Hearing Loss 553
6.12 Infectious Neck Masses in Children 565
6.13 Hemangiomas, Vascular Malformations, and Lymphatic Malformations of the Head and Neck 569
6.14 Branchial Cleft Cysts 573
6.15 Congenital Midline Neck Masses 577
6.16 Congenital Midline Nasal Masses 581
6.17 Choanal Atresia 584
6.18 Cleft Lip and Palate 587

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Section 7 **Facial Plastic and Reconstructive Surgery** **595**
7.1 Craniomaxillofacial Trauma 597
7.1.1 Nasal Fractures 597
7.1.2 Naso-Orbito-Ethmoid Fractures 599
7.1.3 Zygomaticomaxillary and Orbital Fractures 602
7.1.4 Frontal Sinus Fractures 605
7.1.5 Midface Fractures 609
7.1.6 Mandible Fractures 614
7.2 Facial Reanimation and Eye Care 620
7.3 Facial Reconstruction 626
7.3.1 Skin Grafts 626
7.3.2 Local Cutaneous Flaps for Facial Reconstruction 629
7.3.3 Microvascular Free Tissue Transfer 636
7.3.4 Bone and Cartilage Grafts 639
7.3.5 Incision Planning and Scar Revision 643
7.3.6 Implants and Fillers 646
7.4 Cosmetic Surgery 649
7.4.1 Rhytidectomy 649
7.4.2 Brow and Forehead Lifting 653
7.4.3 Chemical Peels and Laser Skin Resurfacing 656
7.4.4 Blepharoplasty 660
7.4.5 Otoplasty 665
7.4.6 Rhinoplasty 668
7.4.7 Deviated Septum and Septoplasty 672
7.4.8 Liposuction of the Face and Neck 675
7.4.9 Hair Restoration 677

Appendix A Basic Procedures and Methods of Investigation 681
A1 Bronchoscopy 681
A2 Esophagoscopy 682
A3 Rigid Direct Microscopic Laryngoscopy with or without Biopsy 684
A4 Tonsillectomy 685
A5 Adenoidectomy 686
A6 Open Surgical Tracheotomy 687
A7 Cricothyroidotomy 688

Appendix B The Cranial Nerves 689

Appendix C Otolaryngologic Emergencies Requiring Immediate Diagnostic and Therapeutic Intervention 695

Index 697

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Foreword

The specialty of otolaryngology–head and neck surgery requires of its clinicians a breadth and depth of knowledge that continues to evolve and expand. The trainee and practitioner have available to them a variety of outstanding written and electronic resources, including comprehensive texts, excellent board review books, and detailed surgical atlases. Why, then, another book? During training, the editors of this handbook were introduced to a neurosurgery clinical manual while caring for skull base surgery patients. That pocket-sized volume was written to provide the clinician with information helpful for clinical patient care in a highly organized format and was readily available. It was apparent that there was no comparable clinical handbook for otolaryngology–head and neck surgery. This book was developed to fill that niche.

It is anticipated that this *Handbook of Otolaryngology–Head and Neck Surgery* will provide immediate access to clinically helpful and necessary information, compressing, as much as possible, the expenditure of valuable time. Decreasing the amount of time spent searching for needed information will accelerate clinical care.

With that goal in mind, this handbook is well organized, follows an established format, is thoroughly indexed, and in all areas is focused on providing only the information that is likely to be clinically most useful. A comprehensive discussion of each subject is neither practicable nor useful in a clinical handbook, and the reader should seek comprehensive texts for that purpose. It is hoped that this handbook will serve clinicians well to the benefit of the patient.

I endorse the content and concept of this clinical contribution, and applaud Dr. Goldenberg and Dr. Goldstein for their vision.

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Preface

The practice of medicine is a uniquely challenging and rewarding endeavor. As our knowledge base continues to expand and the real-world demands on physicians continue to evolve, one of our greatest challenges involves time. Today, the practicing otolaryngologist–head and neck surgeon or trainee may be facing time pressures that were unheard of not long ago, such as PQRI paperwork or mandated work-hour restrictions. However, we hold ourselves to a standard of excellence that must never be compromised. In the provision of excellent clinical care, it is often desirable or necessary to turn to texts or other references to help guide evaluation and management. Thus, in an effort to enable practitioners in our field to find clinical information in a highly time-efficient manner, we sought to produce a truly useful clinical handbook in otolaryngology–head and neck surgery.

There exist excellent comprehensive textbooks for our specialty. There are outstanding basic science review books. Many residents use the same board review books, which serve that purpose well. However, a highly detailed, thorough, well-organized clinical handbook will fill a unique niche.

In designing an ideal clinical handbook for otolaryngology–head and neck surgery, several factors were thought to be most important. One critical feature was that it must be pocket-sized, so that the clinician could have the resource readily available in a lab coat pocket at the time and place when information is sought. Also, it must be of sufficient detail to prove helpful in terms of patient care, while not becoming overwhelming with the nonclinical, basic science, or board review trivia. Furthermore, the organization must enable the user to find what is sought in a quick and efficient manner. With these goals in mind, we have prepared this *Handbook of Otolaryngology–Head and Neck Surgery*.

The 160 chapters follow a standard outline or template so that the reader can readily focus on the desired subject matter. Most include key features, a brief discussion or overview, and a section on epidemiology, followed by symptoms, signs, differential diagnosis, history, physical exam, imaging, labs, treatment options, and outcome and follow-up. This standard organization was chosen to allow the user to review a subject in the typical pragmatic manner in which one approaches a clinical problem. Furthermore, the fact that each brief chapter follows this same outline should enable the reader to become comfortable finding necessary subjects rapidly. When applicable,

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tables are also used to present and organize key information. The extensive index has been thoughtfully planned to as a quick and easy reference. We are confident that this handbook will serve clinicians well.

We will be pleased to receive any comments, or suggestions for the eventual second edition, at **handbookofotolaryngology@gmail.com**.

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Acknowledgments

The contributing authors are true experts in the topics at hand and have put forth great effort into preparing exceptional sections and chapters that are informative, readable, and concise. We would like to thank them for their willingness to participate. Also, we thank the people who provided us with our training – faculty, fellow residents, and patients. And we are indebted to our physician board of reviewers, who helped to carefully edit this text and whose feedback was invaluable: Dr. J. Walter Kutz, Jr., Dr. Elizabeth A. Leon, Dr. Amber Luong, Dr. Vicki L. Owczarzak, Dr. W. Cooper Scurry, Jr., and Dr. Melissa McCarty Statham.

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1 Perioperative Care and General Otolaryngology

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1.0 Approach to the Otolaryngology–Head and Neck Surgery Patient

This book is organized into brief chapters addressing specific clinical entities. The chapters generally follow a standard format that allows readers to readily focus on their information needs. The template includes key features; epidemiology; signs and symptoms; differential diagnosis; evaluation, including history, exam, imaging, and other testing; treatment options, including medical and surgical treatments; and follow-up care. Most chapters also include suggestions for further reading. In this first chapter, we review in detail the approach to an efficient and effective otolaryngologic history and physical examination, which should be especially useful to those new to the care of such patients.

◆ History

The generally accepted organization of the history and physical examination for a new patient is outlined in **Table 1.1**.

The History of Present Illness is the subjective narrative regarding the current problem. One should include a focused summary of the complaint, including location, time of onset, quality, severity, duration, associated problems, and previous testing or treatment.

Table 1.1 Organization of the History and Physical Exam for a New Patient

Chief complaint
History of present illness
Past medical history
Past surgical history
Current medications
Medication allergies
Social history
Family history
Review of systems
Physical examination
Impression
Plan

◆ Physical Exam

The physical examination in otolaryngology is typically a complete head and neck exam. This should include an evaluation of the following:

- The general appearance of the patient (i.e., well- or ill-appearing, acute distress)

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- Stridor, abnormal respiratory effort
- Cranial nerve function
- The pinnae, ear canals, tympanic membranes, including mobility
- 512-Hz tuning fork testing (Weber, Rinne)
- Description of any cutaneous lesions of the head and neck
- External nasal deformities
- Anterior rhinoscopy noting edema, masses, mucus, purulence, septal deviation, perforation
- Oral cavity and pharynx, noting any masses, mucosal lesions, asymmetries, condition of dentition
- Mirror exam of the nasopharynx, hypopharynx, and larynx
- Laryngeal exam should note vocal fold mobility, mucosal lesions, and masses as well as assess the base of the tongue, valleculae, epiglottis, vocal folds, and piriform sinuses
- Presence of hoarseness or phonatory abnormality
- Inspection and palpation of the parotid and submandibular glands
- Inspection and palpation of the neck for adenopathy or masses
- Inspection and palpation of the thyroid gland for enlargement or masses

Other more-specialized aspects of an examination are discussed in the various sections that follow, such as vertigo assessment or nasal endoscopy.

◆ Endoscopic Exam

If the mirror examination does not provide an adequate assessment of the nasopharynx, hypopharynx, or larynx, a flexible fiberoptic nasolaryngoscopy is performed. Usually, the nose is decongested with oxymetazoline (Afrin, Schering-Plough Healthcare Products Inc., Memphis, TN) or Neo-Synephrine (Bayer Consumer Health, Morristown, NJ) spray. Topical pontocaine spray may be added for anesthetic. Surgilube jelly (E. Fougera & Co., Melville, NY) is helpful to reduce irritation. Antifog is applied to the tip of the flexible laryngoscope. The patient is best examined sitting upright. The tip of the scope is inserted into the nostril, and under direct vision is advanced inferiorly along the floor of the nose into the nasopharynx. If there is septal spurring or other intranasal deformities, the other nostril may be used. The nasopharynx is assessed for masses or asymmetry, adenoid hypertrophy, and infection. One can comment of the presence of anteroposterior (AP) or lateral collapse in the sleep apnea patient. The scope is then guided inferiorly to examine the base of tongue, valleculae, epiglottis, piriform sinuses, arytenoids, and vocal folds. Again, mucosal lesions, masses, asymmetries, mobility are noted. Asking the patient to cough and to phonate will reveal vocal fold motion abnormalities.

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◆ Other Tests

Often, laboratory studies, audiograms, or imaging studies are reviewed. These are summarized in the note after the physical exam section. Whenever possible, radiology images (CTs, MRIs) should be reviewed to confirm that one agrees with the reports.

◆ Impression and Plan

In the documentation of the patient's visit, the note concludes with an impression and plan. Generally, a concise differential diagnosis is given, listing the entities that are considered most relevant. A plan is then discussed, including further tests to confirm or exclude possible diagnoses as well as medical or surgical treatments that will be instituted or considered. Timing of a return or follow-up visit, if needed, is noted.

A copy of one's note, or a separate letter, should always be sent to referring physicians.

1.1 Preoperative Assessment

The aims of the Preoperative Assessment are risk stratification and identification of medical conditions and their severity (**Table 1.2**). An additional goal is to ensure that medical management is optimized preoperatively. A review of medications, allergies, NPO (nothing by mouth) status (guidelines are summarized in **Table 1.3**), and previous experiences with anesthesia should be obtained. Laboratory and physiologic tests and screens can be ordered and reviewed prior to surgery. The American Society of Anesthesiologists (ASA) physical status classification (**Table 1.4**) stratifies patient risk associated with general anesthesia based on physical status and existing diagnoses (it is not differentiated by type of operation or patient age).

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Table 1.2 Preoperative Assessment

System	Assessment
Cardiac	Does the patient have hypertension, congestive heart failure, or coronary artery disease? What medications are currently prescribed? Does the cardiac status prohibit the patient from achieving 5 METS (up flight of stairs) without shortness of breath? Review of recent (past-month) ECG for patients older than 45 or those with symptoms? Review of cardiac notes and tests.
Respiratory	Does the patient have asthma, COPD, OSA, or reactive airway disease? Is the patient on medications, home oxygen, or a CPAP device? Is the patient a smoker? Does the patient have shortness of breath related to pulmonary conditions? Review of PFTs, sleep studies, and pulmonary notes
Metabolic	Does patient have diabetes mellitus or thyroid disease? Are these conditions optimized? What medications is the patient currently taking?
NPO status	Has the patient followed the ASA NPO guidelines? (see also Table 1.3)
Alcohol/substance abuse	Does the patient have substance abuse issues that will interact with the pharmacology of an anesthetic? Will postoperative pain management be complicated by existing opioid abuse? Has alcohol or drug abuse contributed to organ dysfunction?

Abbreviations: ASA, American Society of Anesthesiologists; CPAP, continuous positive airway pressure; COPD, chronic obstructive pulmonary disease; ECG, electrocardiogram; METS, metabolic equivalents (of oxygen consumption); NPO, nothing by mouth; OSA, obstructive sleep apnea; PFT, pulmonary function test.

Table 1.3 American Society of Anesthesiologists NPO Guidelines

Item	Time Frame
Clear liquids	2 hours
Infant formula	6 hours
Light meal	6 hours
Breast milk	4 hours
Nonhuman milk	6 hours
Medications	Take with $<$30 mL water $>$1 hour prior to surgery

Abbreviations: NPO, nothing by mouth.

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Table 1.4 American Society of Anesthesiologists (ASA) Physical Status Classification System

ASA Class	Definition
1	Patient has no organic, physiologic, biochemical, or psychiatric disturbance. The pathologic process for which the procedure is to be performed is localized and does not entail systemic disturbance.
2	Mild to moderate systemic disturbance caused either by the condition to be treated surgically or by other pathophysiologic processes.
3	Severe systemic disturbance or disease; variable degree of disability.
4	Severe systemic disorders that may be life threatening, not always correctable by operation.
5	Seriously ill patient who has little chance of survival, but is submitted to the operation in desperation.
6	Patients who have met brain death criteria and are undergoing organ procurement.

1.2 Airway Assessment and Management

All forms of surgery in the head and neck region require consideration of airway management, maintenance of ventilation with an adequate form of anesthesia, and prevention of concentrated oxygen in the operative field in the presence of cautery or laser. Various forms of airway management are discussed below, considering anatomy, innervation, indications, instrumentation and equipment, and clinical context. The specific situation of the difficult airway is then discussed.

◆ Airway Anatomy

There are two physiologic entry points to the airway: the nose and the mouth. The epiglottis, located at the base of the tongue, separates the oropharynx from the hypopharynx. The larynx is made up of a cartilaginous structure supported by muscles and ligaments.

Innervation

The sensory innervation above the epiglottis is provided by the trigeminal nerve (cranial nerve V [CN V]) and the glossopharyngeal nerve (CN IX); below the epiglottis, by the superior laryngeal and recurrent laryngeal branches of the vagus nerve (CN X). For more information on the cranial nerves, see Appendix B.

- Nasal mucosa: by the sphenopalatine ganglion branch of the middle division of the trigeminal nerve

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- Posterior pharynx (including uvula and tonsils): by the continued branches from the sphenopalatine ganglion
- Oropharynx and supraglottic area: by the glossopharyngeal nerve; branches of this nerve include the lingual, pharyngeal, and tonsillar nerves
- Trachea: by the recurrent laryngeal nerve
- Larynx: sensory and motor innervation from the vagus nerve
  - Sensory: above the vocal folds, innervation is supplied by the internal branch of the superior laryngeal nerve; below the vocal folds, by the recurrent laryngeal nerve.
  - Motor: all muscles are supplied by the recurrent laryngeal nerve except for the cricothyroid muscle, which is supplied by the external branch of the superior laryngeal nerve.

◆ Airway Equipment

Oral and Nasal Airways

In anesthetized patients, loss of airway tone allows the tongue and epiglottis to contact the posterior pharyngeal tissue leading to obstruction. Artificial airway devices can be placed in the nose or mouth to provide an air passage. Nasal airways carry a risk of epistaxis and should be avoided in anticoagulated patients. These devices should also be avoided in patients with basilar skull fractures to avoid intracranial penetration of the airway device. If an airway device is indicated in a lightly anesthetized patient, the nasal route is generally tolerated better.

Face Masks

The face mask is designed to contour and conform to a variety of facial features with the intention of creating an airtight seal capable of delivering gasses from the anesthesia equipment.

Laryngoscopes

The most commonly used intubating laryngoscopes by the anesthesiology team have curved (Macintosh) or straight (Miller) blades and an open-blade design. Newer fiberoptic video rigid laryngoscopes are useful, such as the GlideScope (Diagnostic Ultrasound Corporation, Bothell, WA). There are a variety of operative laryngoscopes that may be useful for intubation, such as the Holinger or Dedo laryngoscope.

◆ Airway Assessment

A complete airway examination looks at several aspects of the airway. However, the first priority is to identify patients who may be difficult to ventilate or intubate. This is critically important: induction of general anesthesia with the subsequent inability to adequately ventilate the patient is an acute life-threatening emergency. Thus identification of a patient with a potentially

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difficult airway before induction will enable the operative and anesthetic team to institute an appropriate plan, backup plans, and assemble and test airway equipment ahead of time.

- **Mallampati classification:** used to predict the ease of intubation by looking at the anatomy of the oral cavity while the patient is sitting upright in the neutral position (**Fig. 1.1**)
- **Thyromental distance:** measured from the upper edge of the thyroid cartilage to the chin with head in full extension. A short thyromental distance (<6 cm) equates with an anterior (superior) laryngeal position that is at a more acute angle, which makes the larynx difficult to visualize via direct laryngoscopy. It is a relatively unreliable test unless combined with other tests.
- **Mouth opening:** less than two finger-widths of mouth opening (trismus) suggest difficulty with intubation.
- **Cervical spine movements:** mobility of the atlantooccipital and atlantoaxial joints may be assessed by asking the patient to extend the head while the neck is in flexion. Extension of the head with atlantoaxial joint immobility results in greater cervical spine convexity, which pushes the larynx anteriorly and impairs laryngoscopic view.
- **Temporomandibular joint (TMJ) mobility:** assessed by asking the patient to protrude the jaw while sitting up in the neutral position. Decreased mobility suggests greater difficulty with intubation.
  - Grade A – lower incisors in front of upper incisors (good mobility)
  - Grade B – lower incisors up to upper incisors
  - Grade C – lower incisors cannot be protruded to touch upper incisors (poor mobility)

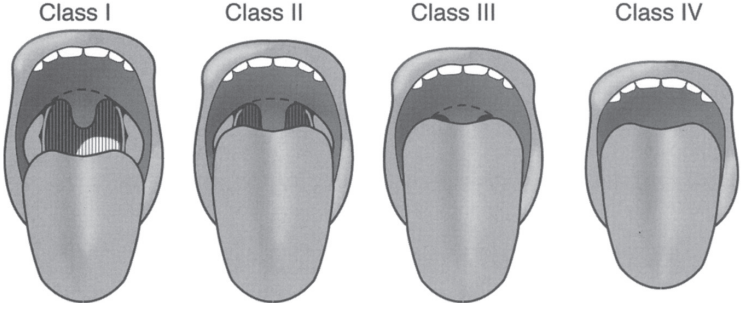

Fig. 1.1 The Mallampati classification of oral opening. Class 1: visualization of soft palate, hard palate, uvula, and tonsillar pillars. Class 2: visualization of soft palate, hard palate, and portion of uvula. Class 3: visualization of soft palate, hard palate, and base of uvula. Class 4: visualization of hard palate only. (From Goldenberg D, ed. Acute surgical management of the airway. Oper Tech Otolaryngol Head Neck Surg 2007;18(2):73. Reprinted with permission.)

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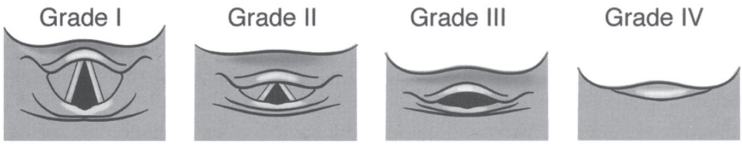

Fig. 1.2 Cormack & Lehane grades. Grade I: visualization of entire laryngeal aperture. Grade II: visualization of posterior part of the laryngeal aperture. Grade III: visualization of epiglottis only. Grade IV: not even the epiglottis is visible. (From Goldenberg D, ed. Acute surgical management of the airway. Oper Tech Otolaryngol Head Neck Surg 2007;18(2):73. Reprinted with permission.)

- **Cormack and Lehane grade:** used to classify the view on direct laryngoscopy. Previous documentation of laryngoscopy should include the grade of glottic view (**Fig. 1.2**).

◆ Preoperative Medication

Medications are often administered preoperatively to alleviate anxiety, to provide analgesia, or as aspiration prophylaxis (**Table 1.5**).

◆ Airway Management

Endotracheal Intubation

Indications for Endotracheal Intubation

- Protection from aspiration
- Altered level of consciousness (Glasgow Coma Score [GCS] score <8)
- Respiratory distress
- Severe pulmonary or multiorgan failure
- Facilitation of positive pressure ventilation
- Operative position other than supine
- Operative site involving the upper airway

Table 1.5 Common Preoperative Medications

Anxiolysis	Analgesia	Aspiration Prophylaxis
Diazepam Midazolam Lorazepam	Narcotics: fentanyl, morphine, meperidine Nonsteroidal antiinflammatory drugs COX II inhibitors Acetaminophen Gabapentin	Bicitra Metoclopramide Famotidine

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- Disease involving the upper airway
- One-lung ventilation

Confirmation of Endotracheal Intubation

- Direct visualization of endotracheal tube passing through the vocal folds
- Carbon dioxide present in exhaled gasses in at least four consecutive breaths (positive end-tidal CO_2)
- Bilateral breath sounds
- Absence of air entry during epigastric auscultation
- Condensation of water vapor in endotracheal tube (ETT) during exhalation
- Maintenance of arterial oxygenation
- Chest x-ray with tip of ETT between the carina and thoracic inlet

Endotracheal Tube Size Recommendations

- Endotracheal tube size (mm): Age/4 + 4 for patients >2 years old.
- Length of insertion: 12 + Age/2. Add 2 to 3 cm for nasal intubation.
- Pediatrics: Uncuffed ETTs are generally used in patients <8 years old.

Types of Endotracheal Tube

● Ring-Adair-Elwyn Endotracheal Tubes. Ring-Adair-Elwyn (RAE) endotracheal tubes are preformed to fit in the nose or mouth and are commonly used in oral or pharyngeal surgery, particularly adenoidectomy or tonsillectomy. The shape prevents obstruction of the surgical field and fits surgical retractors such as the Crow–Davis retractor.

● Armored Endotracheal Tubes. These tubes are commonly used in head and neck surgery. They prevent kinking of the tube when the head is manipulated. This tube works well through a tracheotomy as it can be curved inferiorly out of the surgical field without kinking and sutured in place temporarily.

● Laser-Resistant Endotracheal Tubes. Laser-resistant endotracheal tubes are used in laser surgery, particularly treatment of laryngeal lesions. By preventing an interaction of inhaled oxygen and the laser, these tubes help prevent airway fires.

● Nerve-Monitoring Endotracheal Tubes. For thyroid surgery, a tube with contact electromyographic electrodes positioned at the level of the vocal folds permits intraoperative monitoring of recurrent laryngeal nerve integrity.

Endotracheal Intubation Procedure

- Preoperative evaluation will help determine the route (oral vs. nasal) and method (awake vs. anesthetized) for tracheal intubation.
- Equipment: laryngoscope with working light, appropriate-sized endotracheal tubes, oxygen supply, functioning suction catheter, functioning intravenous (IV) line, and anesthetic medications.

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- Cricoid pressure: an assistant's thumb and forefinger depress the cricoid cartilage downward, compressing the esophagus against the underlying vertebral body. This prevents spillage of gastric contents into the pharynx during the period of time from induction of unconsciousness to placement of the endotracheal tube in the trachea.
- Induction of anesthesia can be achieved by using IV or inhaled agents.

Endotracheal Intubation Complications

During intubation, possible complications include aspiration, dental damage, laceration of lips/gingiva/palate, laryngeal injury, esophageal intubation, endobronchial intubation, bronchospasm, and activation of sympathetic nervous system (elevated heart rate [HR] and blood pressure [BP]).

After intubation, possible complications include aspiration, laryngospasm, transient vocal fold incompetence, pharyngitis, or tracheitis.

Orotracheal Intubation (Fig 1.3)

If there is no history of cervical spine instability, the patient's head is extended into a "sniffing" position. This position aligns the oral, pharyngeal, and laryngeal axes such that the passage from the lips to the glottic opening is a straight line. The height of the operating room (OR) table should be manipulated so that the patient's head is at the level of the clinician's xiphoid cartilage.

The laryngoscope is introduced into the right side of the mouth. Advancing the blade posteriorly and toward the midline, the tongue is displaced to

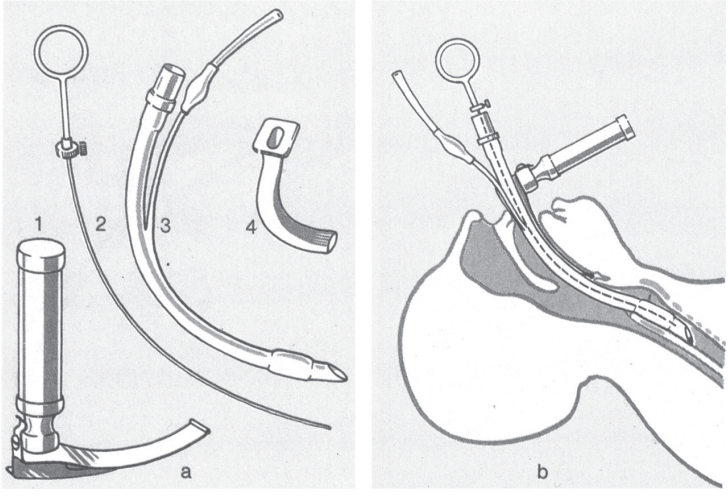

Fig. 1.3 Intubation. **(A)** Necessary instruments. 1, Mcintosh laryngoscope; 2, stylette; 3, tube with cuff; 4, Guedel tube. **(B)** Introduction of the tube. (From Becker W, Naumann HH, Pfaltz CR. Ear, Nose, and Throat Diseases: A Pocket Reference. 2nd ed. Stuttgart/New York: Thieme; 1994:450.)

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the left. Check that the lower lip is not pinched between the blade and the incisors. Placement of blade depends on which style has been selected:

- Macintosh (curved) blade: the tip of the blade is advanced until the tip enters the vallecula (the space between the epiglottis and the base of the tongue).
- Miller (straight) blade: the tip of the blade is passed below the laryngeal surface of the epiglottis, which is then lifted to expose the vocal folds.

Regardless of the blade selected, the laryngoscope is lifted upward and forward in the direction of the long axis of the handle. The upper incisors should *not* be used as a fulcrum for leverage as this may damage teeth.

The vocal folds should be visualized prior to advancement of the endotracheal tube. Passing the endotracheal tube from the right, little resistance should be encountered. The balloon cuff of the endotracheal tube should pass 1 to 2 cm past the vocal folds. Once the proper position of the endotracheal tube is confirmed, it should be secured in place.

Nasotracheal Intubation

Prior to instrumentation, a vasoconstrictor (i.e., oxymetazoline) should be applied to nasal mucosa. After induction of anesthesia and mask ventilation is established the endotracheal tube can be placed.

The naris and endotracheal tube should be generously lubricated. The tube can be softened by placing it in warm water prior to induction. The tube is placed in the nose parallel to the palate, aiming inferiorly to avoid skull base injury, until a loss of resistance is encountered consistent with entrance into the pharynx.

Placement under direct visualization can be performed using a laryngoscope and McGill forceps to direct the endotracheal tube past the glottic opening. Alternately, a fiberoptic bronchoscope can be placed through the tube and directed past the vocal folds.

Rapid-Sequence Intubation

Indications include aspiration risk (full stomach, history of gastroesophageal reflux disease [GERD], pregnancy, trauma) in a patient who does not appear to be a difficult intubation on physical examination.

Nonparticulate antacids, H2 blockers, and metoclopramide can be administered to decrease the acidity and volume of gastric contents. As with a standard intubation described above, instrumentation should be prepared and available. Preoxygenation with 100% O_2 by mask for 3 to 5 minutes or 4 maximal breaths over 30 seconds is sufficient. Once the paralytic and induction agents are administered, no further ventilation is given.

Induction is accomplished with any induction agent, and the procedure is followed immediately with the administration of a paralytic agent. As the medications are being delivered, cricoid pressure should be applied. Succinylcholine (1–1.5 mg/kg) because of its speed of onset is the drug of choice for rapid sequence induction. As soon as jaw relaxation is present, intubation should be performed. Cricoid pressure should continue until tracheal placement of the endotracheal tube is verified.

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Conscious Intubation

Indications include history of difficult intubation, acute infectious/inflammatory process that may involve the airway, mandibular fractures or other facial deformities, morbid obesity, and certain neoplasms involving the upper aerodigestive tract.

The indications and plan should be discussed with the patient. As with a standard intubation all necessary equipment should be available and checked prior to induction. A backup plan should be formulated should intubation be difficult, such as the creation of a surgical airway.

Preparing the airway by decreasing secretions with an antisialagogue (glycopyrrolate 0.2 mg 30 minutes before intubation) will help with visualization. If the nasal route is desired, administer 4 drops of 0.25% Neo-Synephrine to each naris to reduce bleeding. After standard monitors are placed, sedation with fentanyl, midazolam, or dexmedetomidine should be considered.

Topical anesthesia can be achieved using topical agents or nerve blocks. Topical agents include lidocaine jelly, nebulized lidocaine, nasal cocaine, or Cetacaine (Cetylite Industries, Pennshauken, NJ) spray. Regional anesthesia, either alone or in combination with topical agents, is useful in awake intubations. Each major nerve should be approached as described above.

Fiberoptic-Assisted Tracheal Intubation

Indications for a fiberoptic-assisted tracheal intubation include upper airway obstruction, mediastinal mass, subglottic edema, congenital upper airway abnormalities, immobile cervical vertebrae, and confirmation of proper double lumen ETT placement.

Nasal Technique

After vasoconstricting and anesthetizing the airway (see above), the ETT is advanced through the naris into the nasopharynx. The bronchoscope is then inserted into the ETT until the epiglottis and glottic opening are visualized. The scope is then passed through the opening until the carina is in view. While maintaining the carina in sight, the ETT is passed over the scope.

Oral Technique

A bite block should be inserted to protect the fiberoptic scope after anesthetizing the airway (see above). The tracheal tube is inserted into the oropharynx (8–10 cm) and the bronchoscope is inserted through the ETT. The epiglottis and glottic opening should be in view. The ETT is advanced over the scope as the carina is kept in view.

Transtracheal Ventilation

Transtracheal ventilation serves as a temporizing measure if mask ventilation and oxygenation become inadequate or impossible. A catheter (12- or 14-gauge) is inserted into the trachea through the cricothyroid membrane and connected to a jet-type ventilator capable of delivering gas at a pressure of 50 psi. The gas is then delivered using the hand-held jet ventilator.

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Ventilation is best assessed by observing chest rise and fall. It is advised that an inspiration:expiration (I:E) ratio of 1:4 is utilized.

Complications include catheter displacement (caused by high pressure), pneumothorax, and pneumomediastinum.

Laryngeal Mask Airway (LMA)

Indications for the LMA

- Can be used in place of a face mask or ETT
- Can be used in place of an ETT for controlled ventilation as long as the peak pressure does not exceed 30 cm H_2O
- Can aid in the management of difficult airways by providing ventilation or as a guide for fiberoptic intubation

Contraindications for the LMA

- Does not protect against gastric regurgitation and aspiration because it does not provide an airtight seal of the airway

Insertion of the LMA

- The backside of the LMA should be lubricated with a water-soluble lubricant.
- Unconsciousness is rendered with an induction agent of choice. Typically, propofol at doses of 2 to 3 mg/kg produces reliable jaw and pharyngeal muscle relaxation.
- With the LMA held like a pencil, it is placed into the midline of the mouth and advanced with pressure against the hard palate as it slides into the hypopharynx. When the upper esophageal sphincter is encountered resistance is felt. The LMA is then inflated.
- When placed properly, the black vertical line on the back of the LMA faces backward toward the head of the patient.
- The LMA is then removed upon emergence when the patient can follow commands.

Complications of the LMA

- Possible regurgitation/aspiration
- Negative pressure pulmonary edema can result when placed improperly in a spontaneously breathing patient.
- LMA malfunction in patients with pharyngeal or esophageal disease.
- Placement requires neck extension, which is often contraindicated with cervical spine disease.

The GlideScope

The GlideScope is a video laryngoscope that can be a useful alternative to the fiberoptic scope for placement of an endotracheal tube if a difficult airway is expected. The blade is curved like the Macintosh blade with a 60-degree

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curvature to match the anatomic alignment. The GlideScope has a digital camera incorporated in the blade, which displays a view of the vocal folds on a monitor. Under visualization on the monitor, an endotracheal tube is passed between the vocal folds.

Surgical Laryngoscopes

Closed cylinder-style rigid laryngoscopes with bright fiberoptic light guides such as the Dedo or Holinger are used by the otolaryngologist and have advantages that permit visualization of the glottis and intubation.

◆ The Difficult Airway

Among otolaryngology–head and neck surgery patients, a high percentage presents with a difficult airway (**Table 1.6**). By definition, a patient with a

Table 1.6 Conditions Associated with Difficult Airway and Intubation

Condition	Examples
Tumors	Cystic hygroma Hemangiomas Hematoma
Infections	Submandibular abscess Peritonsillar abscess Epiglottitis
Congenital anomalies	Pierre Robin syndrome Treacher Collins syndrome Laryngeal atresia Goldenhar syndrome Craniofacial dystocias
Trauma	Laryngeal fracture Mandibular/maxillary fracture Inhalational burn Cervical spine injury
Inadequate neck extension	Rheumatoid arthritis Ankylosing spondylitis Halo traction
Anatomic variations	Micrognathia Prognathism Large tongue Arched palate Short neck Prominent upper incisors
Obesity	
Airway foreign body	

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difficult airway potentially poses difficulty with ventilation or endotracheal tube placement. The goal of history, physical exam, and chart review is to identify patients with difficult airway before they arrive in the OR. Evaluation by the otolaryngologist and review of diagnostic studies can provide invaluable information to the anesthesiologist when a difficult airway is suspected. Conditions associated with difficult airway are discussed below.

The induction of anesthesia in otolaryngology patients should not be initiated until a plan is formulated between the surgical and anesthesia teams. As outlined in the Difficult Airway Algorithm of the American Society of Anesthesiologists (**Fig. 1.4**), the plan should address basic management choices: surgical versus nonsurgical approach, awake versus asleep, and spontaneous versus nonspontaneous ventilation. The discussion should address backup plans should the initial attempt to secure the airway fails or inability to ventilate ensues. A surgical airway is indicated when ventilation becomes inadequate despite utilization of a nonsurgical airway ventilation device [jet ventilation, LMA, Combitube (Tyco-Kendall, Mansfield, MA)] in a patient under general anesthesia.

If a difficult airway is suspected, instrumentation and plans for possible surgical airway should be available. Cricothyroidotomy is the preferred method in adults. Briefly, the neck is extended, and the cricothyroid membrane is palpated and incised with a scalpel. The airway is entered and an endotracheal or tracheotomy tube is placed to ventilate the patient. Details on cricothyroidotomy are below.

Clinical situations involving a patient with a difficult airway can be divided into two categories. The first is the acute or urgent problem and the second, an elective situation with a suspected or known difficult airway. Both of these scenarios are to be handled differently. A logical, strict approach can prevent a disastrous outcome in what is often a stressful situation. The otolaryngologist–head and neck surgeon should have particular expertise in ensuring an adequate airway and should be skilled in the use of laryngoscopy, bronchoscopy, and surgical approaches to the airway.

Specific techniques to be discussed here include cricothyroidotomy and awake tracheotomy.

Cricothyroidotomy

For a patient with impending or complete obstruction, rapid establishment of an airway is required — the OR is the safest place for this airway control though this is not always possible. If the situation requires establishment of an emergency surgical airway, a cricothyroidotomy is generally the preferred procedure (**Fig. 1.5**). This is because it is simpler and faster than a tracheotomy and has a lower complication rate, especially in less-experienced hands. If it is impossible to mask ventilate, intubate, or control the airway by any other technique such as a laryngeal mask or jet ventilation, or if these are not readily available, cricothyroidotomy should be performed by the most trained and skilled physician in attendance.

Contraindications to cricothyroidotomy include subglottic stenosis or mass, laryngeal trauma, with an inability to identify landmarks; in general, it should be avoided in the pediatric population. A cricothyroidotomy

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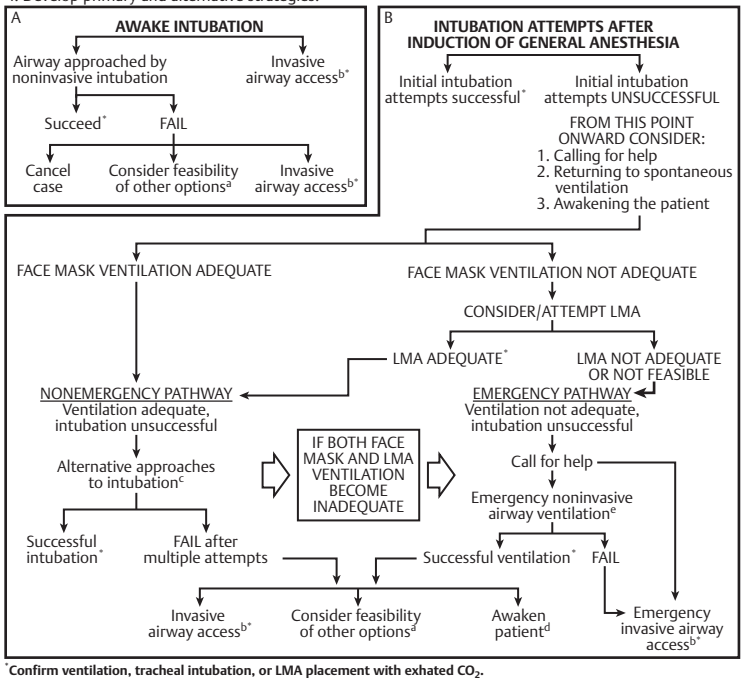
DIFFICULT AIRWAY ALGORITHM

1. Assess the likelihood and clinical impact of basic management problems:
   - A. Difficult ventilation
   - B. Difficult intubation
   - C. Difficulty with patient cooperation or consent
   - D. Difficult tracheotomy
2. Actively pursue opportunities to deliver supplemental oxygen throughout the process of difficult airway management
3. Consider the relative merits and feasibility of basic management choices:

A.	Awake intubation	vs.	Intubation attempts after induction of general anesthesia
B.	Noninvasive technique for initial approach to intubation	vs.	Invasive technique for initial approach to intubation
C.	Preservation of spontaneous ventilation	vs.	Ablation of spontaneous ventilation

4. Develop primary and alternative strategies:



*Confirm ventilation, tracheal intubation, or LMA placement with exhated CO_2.

[a] Other options include (but are not limited to): surgery utilizing face mask or LMA anesthesia, local anesthesia infiltration, and regional nerve blockade. Pursuit of these options usually implies that mask ventilation will not be problematic. Therefore, these options may be of limited value if this step in the algorithm has been reached via the Emergency Pathway.

[b] Invasive airway access includes surgical or percutaneous tracheotomy or cricothyrotomy.

[c] Alternative noninvasive approaches to difficult intubation include (but are not limited to): use of different laryngoscope blades, LMA as an intubation conduit (with or without fiberoptic guidance), fiberoptic intubation, intubating stylet or tube changer, light wand, retrograde intubation, and blind oral or nasal intubation.

[d] Consider repreparation of the patient for awake intubation or canceling surgery.

[e] Options for emergency noninvasive airway ventilation include (but are not limited to): rigid bronchoscope, esophageal-tracheal Combitube ventilation, and transtracheal jet ventilation.

Fig. 1.4 The American Society of Anesthesiologists Difficult Airway Algorithm. (From Practice guidelines for management of the difficult airway: an updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Anesthesiology 2003;98:1273. Reprinted with permission.)

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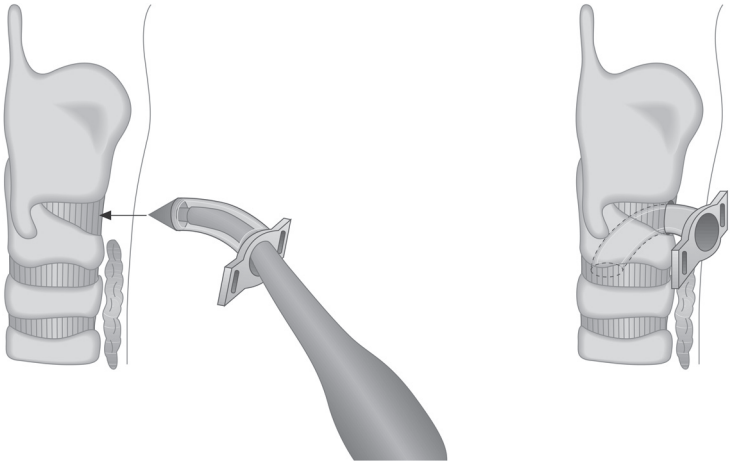

Fig. 1.5 Cricothyroidotomy. The airway is entered through an incision of the cricothyroid membrane. In this illustration, a trocar through a tracheotomy tube is used; a no. 15 blade will also suffice. (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:379.)

should be revised to a tracheotomy in the OR within 24 hours to prevent subsequent development of subglottic stenosis.

For needle cricothyroidotomy, a catheter is placed over a needle that penetrates the cricothyroid membrane, allowing ventilation by a pressurized stream of oxygen. Needle cricothyroidotomy is the preferred method of establishing an emergency airway in children younger than 10 to 12 years. (See the Cricothyroidotomy section, A7, in Appendix A).

Stable but Compromised Airway

A patient with supraglottitis, angioedema, or a laryngeal mass may have a stable but compromised airway. These patients maintain their oxygen saturation and do not require an emergency airway; however, the stability of the situation requires clinical judgment. In some cases, the patient can be managed with medical management and close monitoring. In other cases, in may be prudent to secure the airway.

Tracheotomy is a surgical procedure in which the tracheocutaneous airway is created through the anterior neck and the trachea. It may be reversible (**Table 1.7**). (For details on open surgical tracheotomy, see A6 in Appendix A.)

Awake Tracheotomy

In the unstable, poorly ventilated patient, an airway must be secured. If intubation is deemed impractical or dangerous, an awake tracheotomy can be performed. It is important to note that this is best done in a controlled

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Table 1.7 Indications for Tracheotomy

Upper airway obstruction with any of the signs or symptoms at right	Air hunger, stridor, retractions, obstructive sleep apnea, bilateral vocal fold paralysis, previous neck surgery or throat trauma, previous neck irradiation, prolonged or expected prolonged intubation
Pulmonary toilet	Inability of patient to manage secretions, including the following: aspirations and excessive bronchopulmonary secretions
Ventilatory support	Where prolonged ventilation or ventilatory problems are anticipated

environment, in the OR, or in the intensive care unit (ICU) with local anesthetic and a patient who can maintain spontaneous adequate ventilation. Clear communication between the otolaryngologist–head and neck surgeon, anesthetist, nurses, and technicians is vital. It is important to note that a life-threatening emergency may arise and this will necessitate close collaboration. All equipment should be open and ready.

For the awake tracheotomy, the patient should be placed in the semi-Fowler position enabling the anesthesiologist ready access to the airway. Every effort should be made to keep the patient comfortable and the general atmosphere should be as calm as possible. The patient is typically only minimally sedated and should not be paralyzed, maintaining spontaneous adequate ventilation.

Note that in the elective (nonurgent) tracheotomy the patient is intubated prior to the start of surgery.

● Awake Tracheotomy Technique. Surgical landmarks are palpated, and a horizontal or vertical skin incision is made between the cricoid and suprasternal notch. Subcutaneous tissue is bluntly spread. Next, lateral retraction of the strap musculature is performed. The thyroid isthmus is encountered and either retracted caudad, cephalad, or divided using a monopolar cautery. Once the trachea is in view, a tracheal hook may be used to provide superior retraction on the cricoid ring; next the trachea is entered, usually below the second ring, and a window or inferiorly based (Bjork) flap is created.

An appropriate tracheotomy tube is then placed while the anesthesiologist withdraws the endotracheal tube under direct visualization. After the tracheotomy tube is in place, the cuff is inflated, the patient is ventilated and CO_2 return and appropriate rise in oxygenation are confirmed. Then, the tracheotomy tube is sutured at four corners with suture, and a tracheotomy tie should be placed to secure the tracheotomy tube around the neck.

Complications of tracheotomy include bleeding, infection, crusting or mucus plugging, pneumothorax, and accidental dislodgment of the tracheotomy tube. Late postoperative complications include tracheoesophageal fistula, tracheal stenosis, tracheocutaneous fistula, and airway stenosis. A rare but deadly complication is a trachea–innominate artery fistula.

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Percutaneous Dilation Tracheotomy

A percutaneous dilation tracheotomy is *not* to be used in an emergent airway situation: this procedure is *only* to be performed on patients who are already intubated. Percutaneous bedside dilation tracheotomy has become a safe alternative to elective open tracheotomy in many ICUs. There are several different techniques all widely based on the Seldinger technique (needle and catheter over guide wire). Indications for a percutaneous dilation tracheotomy (PDT) are identical to those used for a routine open operative tracheotomy. The most common indication is prolonged intubation, mechanical ventilation. However, particular attention should be paid to potential contraindications. Some absolute contraindications for the performance of a PDT are the need for emergency tracheotomy (i.e., an emergent airway), or in a population of infants and children. Relative contraindications include poor neck landmarks, morbid obesity, previous neck surgery, limited neck extension, and severe coagulopathy that is not correctable. Other relative anesthetic contraindications include high positive end expiration pressure (PEEP >18 cm), high airway pressure (>45 cm), high FiO_2 requirement (>80%), retrognathic mandible with limited view of the larynx, and an inability to reintubate.

An appropriate percutaneous tracheotomy kit (i.e., Ciaglia or Griggs kit) and a video bronchoscope are needed to perform this procedure. At least two persons are needed: one to perform the tracheotomy, the other to manage the endotracheal tube and video bronchoscope.

● Percutaneous Dilation Tracheotomy Technique. A shoulder roll is placed beneath the patient's shoulders and a bronchoscopist should perform simultaneous bronchoscopy through the endotracheal tube during the procedure. An incision is made either horizontally or vertically between the cricoid and the suprasternal notch and an introducer needle is placed into the trachea at about the second ring; this is confirmed with bubbling of air and with simultaneous video bronchoscopy. In addition, transillumination of light from the bronchoscope can be used to help demarcate the best site for the introducer needle. The needle is withdrawn and the overlying plastic cannula is left in place. A J-tipped guide wire is then placed under direct visualization through the catheter into the trachea. Again, every step should be bronchoscopically visualized on the video monitor during the procedure. Dilation is then performed using the provided dilator over the guide wire. This is removed and once dilation of the anterior tracheal wall has been performed, a percutaneous tracheotomy appliance is placed over the guide wire and positioned in the airway. Following this, the bronchoscope should be withdrawn from the endotracheal tube and introduced via the tracheotomy tube to confirm placement in the airway. The placement is confirmed by visualization of the carina. The tracheotomy tube is connected to the anesthesia circuit to confirm bilateral breath sounds and end-tidal CO_2. It is then secured using four sutures on all corners of the flange and a tracheotomy tie is also used to secure the appliance around the neck. Only then can the orotracheal tube be fully removed.

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For percutaneous tracheotomy, the first tracheotomy tube change should *not* be performed sooner than 10 days postoperatively, to allow for formation of a safe tract.

Criteria for Extubation

- Respiratory rate (RR) <30 per minute
- Tidal volume (TV) >5 mL/kg
- Vital capacity (VC) >10 mL/kg
- Negative inspiratory force (NIF) > −20 cm H_2O
- Tidal volume/respiratory rate (TV/RR) >10
- PaO_2 >70 mm Hg (40% fraction of inspired oxygen [FiO_2])
- $PaCO_2$ <50 mm Hg
- Level of consciousness (LOC) stable or improving
- Ventilation, dead space/tidal volume ratio (Vd/Vt) <0.6

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1.3 Diagnostic Imaging of the Head and Neck

Many of the structures of the head and neck are deep and inaccessible to direct visualization, palpation, or inspection. Therefore, valuable information may be obtained by the use of various radiographic techniques. Technologic advances have replaced simple x-ray procedures with computed tomography (CT), magnetic resonance imaging (MRI), ultrasound, and positron emission tomography (PET). Other imaging modalities, such as angiography, are used for specific conditions such as vascular lesions.

◆ Computed Tomography

A contrast-enhanced CT scan is typically the first imaging technique used to evaluate many ear, nose, throat, and head and neck pathologies. The CT scan is an excellent method for staging of tumors and identifying lymphadenopathy. A high-resolution CT scan may be used in cases of trauma of the head,

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neck, laryngeal structures, facial bones, and temporal bone. Temporal bone CT is used to assess middle ear and mastoid disease; paranasal sinus CT is the gold standard test for assessing for the presence and extent of rhinosinusitis. A CT scan is superior to MRI in evaluating bony cortex erosion from tumor. A CT scan is also widely used for posttreatment surveillance of head and neck cancer patients.

Working Principle of CT

In CT, the x-ray tube continuously rotates around the cranio-caudal axis of the patient. A beam of x-rays passes through the body and hits a ring of detectors. The incoming radiation is continuously registered and the signal is digitized and fed into a data matrix taking into account the varying beam angulations. The data matrix can then be transformed into an output image (**Fig. 1.6**).

Recent advances have improved the quality of CT imaging. Multidetector scanners have several rows of photoreceptors, allowing the simultaneous acquisition of several slices. Helical techniques allow patients to move continuously through the scanner instead of stopping for each slice. These advances have significantly decreased scan times and radiation exposure while improving spatial resolution. Newer in-office flat plate cone beam scanners can rapidly acquire 1-mm slice thickness images of the sinus and temporal bone with very low radiation exposure.

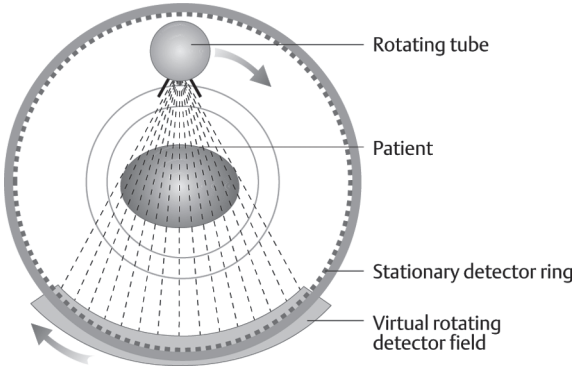

Fig. 1.6 Working principle of computed tomography. The x-ray tube rotates continuously around the longitudinal axis of the patient. A rotating curved detector field opposite to the tube registers the attenuated fan beam after it has passed through the patient. Taking into account the tube position at each time point of measurement, the resulting attenuation values are fed into a data matrix and further computed to create an image. (From Eastman GW, Wald C, Crossin J. Getting Started in Clinical Radiology: From Image to Diagnosis. Stuttgart/New York: Thieme; 2006:9. Reprinted with permission.)

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Table 1.8 Attenuation of Different Body Components

Body Component	Hounsfield Units (HU)
Bone	1000–2000
Thrombus	60–100
Liver	50–70
Spleen	40–50
Kidney	25–45
White brain matter	20–35
Gray brain matter	35–45
Water	−5 to 5
Fat	−100 to −25
Lung	−1000 to −400

Source: Eastman GW, Wald C, Crossin J. Getting Started in Clinical Radiology: From Image to Diagnosis. Stuttgart/New York: Thieme; 2006:9. Reprinted with permission.

Contrast Media

Contrast media is used in CT to visualize vessels and the vascularization of different organ systems (**Table 1.8**). Contrast material contains iodine or barium, which attenuates the scan radiation.

Computer-Assisted Surgical Navigation

CT scanning data can be utilized for computer-assisted surgical navigation. There are several systems in use. The axial CT image data, acquired at 1 mm slice thickness or less, is loaded onto the image-guidance system in the operating room. The machine uses a system, such as an infrared camera, to detect the patient's facial features or landmarks, and can also be registered to detect various surgical instruments. The location of an instrument tip is then displayed on the previous CT images in three planes. Most often, this is used in sinus surgery.

◆ Magnetic Resonance Imaging

MRI provides the physician with high definition imaging of soft tissue without exposing the patient to ionizing radiation. MRI is useful for mucosal tumors, neoplastic invasion of bone marrow, and at times perineural invasion of large nerves. MRI is valuable in assessing intracranial extension of tumors of the head and neck. Gadolinium-enhanced MRI of the brain with attention to the internal auditory canal is the gold standard test for diagnosis of vestibular schwannoma or meningioma, easily identifiable on postcontrast T1-weighted images. The disadvantages of MRI include limited definition of bony detail, and cost. Magnetic resonance angiography (MRA)

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Table 1.9 Definitions of Terms Used in Magnetic Resonance Imaging

Time to repetition	TR
Time to echo	TE
Time to inversion	Ti
Time to magnetize (regrowth); also known as spin lattice relaxation time	T1
Time to demagnetize (DK); also known as spin relaxation time	T2

is a useful modality for imaging vascular anatomy or vascular pathology without the intravascular infusion of iodine contrast medium, which is used in traditional angiography with x-ray fluoroscopy.

Working Principle of MRI

MRI is a technique that produces cross-sectional images in any plane without the use of ionizing radiation. MR images are obtained by the interaction of hydrogen nuclei (protons), high magnetic fields, and radiofrequency pulses. This is done by placing the patient in a strong magnetic field, which initially aligns the hydrogen nuclei in similar directions. The intensity of the MRI signal that is converted to imaging data depends on the density of the hydrogen nuclei in the examined tissue (i.e., mucosa, fat, bone) and on two magnetic relaxation times (**Table 1.9**).

Contraindications to MRI

- Implanted neural stimulators, cochlear implants, and cardiac pacemakers (MRI may cause temporary or permanent malfunction)
- Ferromagnetic aneurysm clips, foreign bodies with a large component of iron or cobalt that may move or heat up in the MRI scanner
- Metallic fragments within the eye
- Placement of a vascular stent, coil, or filter in the past 6 weeks
- Ferromagnetic shrapnel
- Relative contraindications include claustrophobic patients, critically ill patients, morbidly obese patients who cannot physically fit in the MRI scanner, metal implants in the region of interest

◆ Ultrasound

Ultrasound or ultrasonography is an inexpensive and safe method of gaining real-time images of structures of the head and neck. Neck masses can be assessed for size, morphologic character (i.e., solid, cystic, or combined solid and cystic also known as complex), and for association with adjacent structures. High-resolution ultrasound is used for head and

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neck anomalies such as thyroglossal duct cysts, branchial cleft cyst, cystic hygromas, salivary gland masses, abscesses, carotid body and vascular tumors, and thyroid masses.

Ultrasound combined with fine-needle aspiration biopsy (FNAB) and cytology is helpful in both providing a visual description as well as an aid for sampling of a mass for cytologic evaluation. Until recently, ultrasounds were performed mainly by radiologists. However, many otolaryngologists are now performing their own in-office ultrasounds and ultrasound-guided FNABs.

Working Principle of Ultrasound

An alternating electric current is sent through piezoelectric crystals; it vibrates with the frequency of the current, producing sound waves of that frequency. In medical ultrasound, typical frequencies vary between 1 and 15 MHz. Ultrasound gel acoustically couples the ultrasound transducer to the body, where the ultrasound waves can then spread. Inside the body the sound is absorbed, scattered, or reflected. Fluid-filled (cystic) structures appear dark and show acoustic enhancement behind them. Bone and air appear bright because they absorb and reflect the sound, showing an "acoustic shadow" behind them (**Fig. 1.7**). Linear transducers with a width of 7.5 to 9 cm and frequencies of 10 to 13 MHz are typically used for evaluating the neck and thyroid.

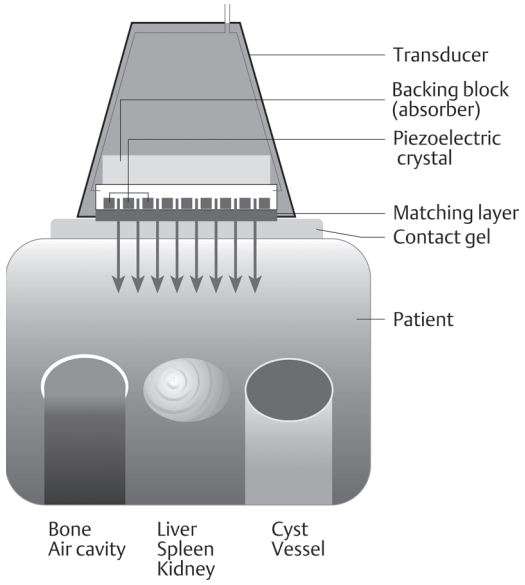

Fig. 1.7 Working principle of ultrasonography. (From Eastman GW, Wald C, Crossin J. Getting Started in Clinical Radiology: From Image to Diagnosis. Stuttgart/New York: Thieme; 2006:11. Reprinted with permission.)

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◆ Barium Esophagram

An esophagram (also known as a barium swallow) is designed to evaluate the pharyngeal and esophageal mucosa; it is distinct from a modified barium swallow (MBS), which evaluates laryngotracheal aspiration and is usually performed in conjunction with a speech pathologist. These techniques are performed utilizing fluoroscopy. Fluoroscopy with intraluminal contrast is invaluable for studying the functional dynamics of the pharynx and esophagus.

An MBS evaluates the coordination of the swallow reflex. It is most often used to determine the etiology and severity of airway aspiration. A speech pathologist is usually in attendance and administers barium suspensions of varying thickness (thin liquid, thick liquid, nectar, paste, and solid) while the radiologist observes fluoroscopically in the lateral projection. One can also assess for esophageal motility/dysmotility, Zenker diverticulum, stricture, mass, hiatal hernia, or obvious free reflux.

Contrast Media

Barium suspension is the most commonly used fluoroscopic contrast agent. If a perforation of the hypopharynx or esophagus is suspected there is a risk for barium extravasation into the soft tissues of the neck and/or chest. Therefore, in these cases, water-soluble contrast agents are used (such as Gastrografin, Bracco Diagnostics, Inc., Princeton, NJ). It is important to note that these agents may cause a chemical pneumonitis or severe pulmonary edema if aspirated into the airway.

◆ Nuclear Medicine Imaging

Positron Emission Tomography with Computed Tomography

PET-CT is essentially a positron emission tomography scan performed and superimposed upon a simultaneous computed tomography scan to allow precise correlation between increased function (enhanced cellular activity) and anatomic evaluation provided by the CT. The PET scanning portion is a functional imaging technique that measures metabolic activity through the use of tagged nuclear isotopes such as a glucose precursor. ^{18}F-fluoro-deoxyglucose (FDG) is the most commonly used radiotracer and has a half-life of ~110 minutes.

Working Principle of PET-CT

After being emitted from the atom, the positron travels in the tissue for a short distance until it encounters an electron and forms a positronium, which immediately annihilates (converts its mass to energy) forming two photons. These annihilation photons travel away from each other at 180 degrees and are picked up by the detectors placed around the patient. Simultaneous detection of these photons relates them to the same annihilation event and allows spatial localization. Annihilation detection is accomplished by dedicated PET scanners, which yield spatial resolution

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and sensitivity. The spatial resolution of the final reconstructed images is limited by the number of collected events.

FDG is taken up by glucose transporters. Normally, glucose enters into a cell, is phosphorylated by hexokinase, and then enters directly into either the glycolytic or glycogenic pathway. FDG, a glucose analogue, is subsequently unable to continue into the usual glucose metabolic pathways (due to the presence of fluorine) and is essentially trapped in the cell as FDG-phosphate. Because neoplastic cells have higher rates of glycolysis and glucose uptake, localized areas of intracellular activity on a PET scan may represent neoplastic disease.

^{18}F is cyclotron produced and has a half-life of 110 minutes. Tumor concentration of FDG generally peaks at 30 minutes, remains constant for 60 minutes, and then declines.

Note that FDG can also accumulate nonspecifically in other cells that have active glycolysis such as areas of active inflammation and infection. This may lead to a false-positive PET-CT scan. Other activities that may cause false-positive findings include muscular activity, foreign bodies, and granulomas. False-negatives in PET-CT scans may occur when the tumor threshold is too small ($<$0.5 cm in diameter). In PET scanning, quantification of FDG uptake intensity is generally expressed on an arbitrary scale as standard uptake values (SUVs).

Thyroid Scintigraphy

Thyroid scintigraphy renders, at one point in time, information about the global and regional functional status of the thyroid. It is observer-independent and reproducible with low inherent radiation exposure. Scintigraphic imaging of the thyroid helps determine whether solitary or multiple nodules are functional when compared with the surrounding thyroid tissue. Findings for a nodule may be normal functional (warm), hyperfunctional (hot), or hypofunctional (cold). Scintigraphy can also help determine whether cervical masses contain thyroid tissue, and it can demonstrate whether metastases from well-differentiated thyroid cancer concentrate iodine for the purpose of radioiodine therapy. For thyroid scintigraphy the following radionuclides are in use: technetium-99m (^{99m}Tc), ioflupane (^{123}I), and iodine-131 (^{131}I).

Working Principle of Thyroid Scintigraphy

The technique of thyroid scintigraphy is based on the principle that functional active thyroid cells incorporate iodine.

Parathyroid Scintigraphy

Several radiotracers are available for parathyroid scintigraphy. At present, the radiotracer of choice is ^{99m}Tc-sestamibi (aka sestamibi, methoxyisobutylisonitrile, or MIBI). ^{99m}Tc-sestamibi is a lipophilic cation that is taken up in the mitochondria of the cells. Of note, this radiotracer can be used with a wide variety of imaging techniques including planar MIBI, single proton emission computed tomography (SPECT), and fused SPECT-CT.

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Working Principle of Parathyroid Scintigraphy

Sestamibi accumulates in the thyroid and parathyroid tissues within minutes after IV administration, but it has a different washout rate from these two tissues. It is released faster from the thyroid than from the parathyroid. The presence of large numbers of mitochondria-rich cells in parathyroid adenomas is thought to be responsible for their slower release of ^{99m}Tc-sestamibi from hyperfunctioning parathyroid tissue versus the adjacent thyroid tissue. Thus, on 2- to 3-hour washout images, after thyroid uptake has dissipated, the presence of a retained area of activity allows one to identify and localize a parathyroid adenoma. Overall, ^{99m}Tc-sestamibi parathyroid scintigraphy has good sensitivity for the detection and localization of a single adenoma in patients with primary hyperparathyroidism. Correlation with ultrasound findings can also be helpful.

Single Proton Emission Computed Tomography

SPECT scanning for parathyroid disease allows increased accuracy of routine sestamibi scanning by ~2 to 3%. SPECT scanning can be performed within the first several hours after a patient is injected with the sestamibi. During the scan, multiple images are taken of the patient's head and neck. These images are assimilated to provide a three-dimensional picture. SPECT scanning is typically used when ordinary planar sestamibi scans are inconclusive.

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1.4 Anesthesia

1.4.1 Principles of Anesthesia

◆ Modes of Anesthesia

- **General anesthesia:** characterized by total loss of consciousness with blunted (or absent) protective upper airway reflexes and the use of an endotracheal tube or supraglottic device (e.g., laryngeal mask airway).
- **Sedation:** aims to maintain protective upper airway reflexes in patients with iatrogenic altered levels of consciousness. Supplemental oxygen is often required (e.g., nasal cannula), with or without the assistance of an oral/nasopharyngeal airway. It can range from minimal (i.e., patient is responsive to verbal command) to deep (i.e., no response to procedural stimulation).

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- **Regional anesthesia:** involves the use of local anesthetics in conjunction with sedation or general anesthesia to improve analgesia, expedite recovery, and reduce overall IV/inhalational anesthetic requirements. It can be used as a sole anesthetic for minor or superficial procedures.

◆ Factors of Anesthesia

An ideal anesthetic strikes a balance between the following four essential factors, which in turn are influenced by independent patient risk factors, unique surgical requirements, and circumstances under which recovery is to occur.

- **Amnesia/anxiolysis:** management of preoperative anxiety and control of intraoperative awareness is a cornerstone of anesthetic care. Various oral, IV, and/or inhalational agents may be used, depending on the desired effect.
- **Analgesia:** multimodal therapy is the preferred method of achieving intra- and postoperative pain control with a combination of narcotics, nonsteroidal antiinflammatory drugs (NSAIDs), local anesthetics, and/or adjutants [e.g., gabapentin, Ketamine (Pfizer Pharmaceuticals, New York, NY), clonidine].
- **Muscle relaxation:** depolarizing and/or nondepolarizing muscle relaxants facilitate optimal airway conditions for laryngoscopy and surgical manipulation.
- **Antiemetics:** postoperative nausea and vomiting (PONV) is a strongly undesirable complication in patients following head and neck surgery. High-risk procedures include otology surgery, adenotonsillectomy, thyroidectomy, as well as head and neck cancer surgery. PONV influences patient satisfaction, length of stay, recovery, and surgical outcome. Multimodal and prophylactic therapy improves outcomes. Emesis may predispose development of wound hematoma, which can become an airway emergency in the presence of a neck wound.

◆ Phases of Anesthesia

- **Preinduction:** begins in the preoperative area and continues to the point where a patient is positioned on the operating room table with standard monitors (electrocardiogram [ECG], noninvasive blood pressure, pulse oximetry, capnography) and has been adequately preoxygenated.
- **Induction:** general anesthesia in patients with established IV access is initiated with rapid-acting parenteral drugs to facilitate airway control. Otherwise, inhalational anesthetics are administered via a face-mask; once an adequate depth of anesthesia is achieved, IV access is established and is followed by the placement of an endotracheal tube or supraglottic device (unless a very brief procedure is planned). For sedation cases, IV agents are titrated to effect while maintaining spontaneous ventilation. For difficult airway situations, awake fiberoptic nasotracheal intubation or awake tracheotomy may be indicated to secure the airway, prior to induction of general anesthesia.

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Table 1.10 Key Areas in Each Phase of Anesthesia

Preinduction	Induction	Maintenance	Emergence
Airway assessment Intravenous access Anxiolysis Aspiration precautions Positioning for safety and patient comfort Baseline vital signs Preoxygenation	Adequate depth of anesthesia (to prevent awareness or laryngospasm) Aspiration precaution (cricoid pressure, rapid sequence induction) Positioning for optimal view of the airway upon direct laryngoscopy Securing the airway (confirmed by auscultation, inspection, and capnography)	Maintenance of end organ perfusion Adequate fluid administration Normothermia Appropriate anesthetic depth Procedural analgesia Facilitation of optimal surgical field (e.g., controlled hypotension)	Extubation upon meeting criteria (spontaneous ventilation, reversal of neuromuscular blockade, suctioning of airway)

- **Maintenance:** global amnesia is maintained during surgery with inhalational agents and/or parenteral drug infusions. Surgical analgesia is maintained either with a well-established regional block, generous local anesthetic infiltration, intermittent opioid boluses, and/or IV narcotic infusion. Duration of required muscle paralysis depends on several factors, and may be just long enough to facilitate endotracheal tube placement. In some instances (e.g., cases involving extensive neuromonitoring), anesthesia may be maintained through the predominant or exclusive use of parenteral infusions (total intravenous anesthesia [TIVA]). TIVA is sometimes useful to reduce vasodilation and bleeding, i.e., in endoscopic sinus surgery.
- **Emergence:** extubation upon meeting criteria

Table 1.10 provides an overview of the key aspects of each phase of anesthesia.

◆ Stages of Anesthesia

- **Stage 1:** the period between initial delivery of induction agents and loss of consciousness. Patients experience analgesia without amnesia and can often carry on a conversation during this time.
- **Stage 2:** more commonly known as the "excitement stage," follows loss of consciousness and is characterized by delirious, uninhibited, and often spastic activity. Cardiovascular and respiratory patterns are erratic. There is also an increased risk for aspiration. The goal of any anesthetic induction is thus to minimize time spent in this stage.

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- **Stage 3:** also known as "surgical plane," where procedural stimulation causes minimal, if any, cardiovascular and/or respiratory changes.
- **Stage 4:** massive anesthetic overdose that causes severe depression of brainstem activity leading to respiratory and/or cardiovascular collapse. This stage should never be reached as it may be lethal even with appropriate cardiovascular and/or respiratory support.

Further Reading

Barash PG, Cullen BF, Stoelting RK, et al. Clinical Anesthesia. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009
Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York: McGraw-Hill Medical; 2005

1.4.2 Regional Anesthesia Techniques

◆ Benefits of Regional Anesthesia

When combined with a general anesthetic, regional nerve blocks reduce narcotic requirements and their subsequent side effects such as nausea, vomiting, somnolence, and respiratory depression. Time to discharge is reduced.

◆ Contraindications to Regional Anesthesia

Contraindications to regional anesthesia include lack of patient consent and interference with surgical field/technique. Relative contraindications include coagulopathy, infection at the skin site, and presence of neurologic disease.

◆ Complications Common to All Nerve Blocks

Complications common to all nerve blocks include local anesthetic complications (intravascular injection, overdose, and allergic reaction), nerve damage (needle trauma, intraneural injection), and hematoma.

◆ Blocks of the Scalp and Face

Supraorbital and Supratrochlear Nerve

Indications: closure of lacerations, forehead, and ear procedures

The forehead and anterior scalp can be rendered insensate by blocking the supraorbital and supratrochlear branches of the ophthalmic division of the trigeminal nerve where they exit from their respective foramina along the brow line.

A skin wheal is placed over the glabella. A 25-gauge needle, bent to aid in superficial placement, is inserted through the anesthetizing wheal and

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advanced laterally along the brow. A total of 8 mL of local anesthetic is applied from the glabella to the lateral edge of each brow.

Greater and Lesser Occipital Nerves

Indications: closure of lacerations

By blocking the greater and lesser occipital nerves, the posterior scalp can be anesthetized. By placing a track of local anesthetic from the mastoid process to the inion (i.e., external occipital protuberance) along the highest nuchal line from each side, both the greater and lesser occipital nerves will receive a dose of local anesthetic and the posterior scalp will become anesthetized.

A large skin wheal is placed over the mastoid process on each side using a 27-gauge needle. Then through this wheal, a wheal is placed from the mastoid process to the inion using a 25-gauge Quincke needle that is bent to facilitate a superficial injection.

Infraorbital Nerve

Indications: closure of lacerations, facial surgery

The maxillary division of the trigeminal nerve innervates the midface, from the inferior portion of the orbit to the mandible. This area includes the area overlying the zygoma, the maxilla, and most of the nose, as well as the philtrum and the hard and soft palate.

The infraorbital foramen is palpable 2 to 3 mm below the rim of the orbit, just medial to the equator of the orbit. A small-gauge needle is used to inject local anesthesia just outside the foramen. Avoid injection into the foramen as the nerve is located in a confined space.

A small amount (2–4 mL) of local anesthetic is sufficient and it should be injected based on which area is to be anesthetized, i.e., emphasize *above* the foramen for lower lid work, *medial* to the foramen for lateral nasal work, and *inferomedial* to the foramen for work on the philtrum.

◆ Blocks of the Neck

Superficial Cervical Plexus

The cervical plexus is composed of four nerve roots, C_1–C_4, and terminates in four branches: lesser occipital, great auricular, transverse cervical, and supraclavicular nerves. The terminal branches emerge superficially at the posterior border of the sternocleidomastoid (SCM) muscle along the midportion of the muscle.

This is a purely cutaneous nerve block; there is no motor block with the superficial cervical plexus block. Neuromonitoring and stimulation of the recurrent laryngeal nerve is not compromised when using this block.

The patient is positioned in the seated position. A line connecting the insertion of the SCM at the midpoint of the clavicle to the mastoid process along the posterior muscle border designates the path in which subcutaneous local anesthetic should be injected. Initially, 3 to 5 mL of local anesthetic is injected at the midpoint of the SCM using a 27-gauge needle. Using a

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25-gauge Quincke point spinal needle, subcutaneous injections are then performed from this initial injection site in caudad and cephalad directions along the posterior edge of the SCM. Infiltration along these paths should require 6 to 8 mL of anesthetic in each direction. Aspiration prior to injection is important to avoid intravascular injection.

Deep Cervical Plexus

The deep cervical plexus (DCP) is the collection of the C_2–C_4 nerve roots as they exit the "gutter" formed by the transverse process of the respective vertebrae. By injecting prior to the division of the cervical roots into dorsal and ventral spinal nerves, a more complete blockade of the ipsilateral neck is achieved—including both sensory and motor elements. This is not commonly used for otolaryngology procedures.

The patient is seated upright in a high Fowler position with a small towel behind the shoulders. The above-mentioned line is drawn between the mastoid process and the anterior tubercle of C_6, which is palpable in the vast majority of patients. A line parallel to this is drawn 1.5 cm behind the first, and the posterior tubercle is palpated on C_2, C_3, and C_4. Deep palpation can be uncomfortable and a light touch is indicated. After a small skin wheal with a 27-gauge needle, a short (2.5 cm) blunt needle is advanced to the posterior process of each of the three vertebrae and then "walked" laterally and anterior to the posterior tubercle. An advance of 1 mm beyond the bony tubercle will suffice. It is not uncommon for the patient to describe a light paresthesia in the dermatome of the root being blocked. After careful aspiration, 4 to 5 cc of local anesthetic (with epinephrine) is injected at each of the three levels. (C_2, C_3, C_4). The proximity to the spinal column and major vascular structures increases the risk of intrathecal or intravascular injection.

Specific Nerve Blocks for the Upper Airway

Maxillary Division of the Trigeminal Nerve (Sphenopalatine Ganglion)

- The transnasal topical approach to the sphenopalatine ganglion involves application of local anesthetic to the mucous membranes surrounding the ganglion.
- Position the patient supine with neck extension. A local anesthetic (typically 80 mg of 4% cocaine) is applied to each nostril. Cotton-tipped applicators soaked in 4% cocaine are gently swirled and advanced into the nares. Each applicator is advanced a little further than the one prior, and once placed the applicator is left there as successive applicators are introduced. Each nostril should receive four to seven applicators as the opening allows. The applicators should remain in the nares for at least 20 minutes allowing the local anesthetic to diffuse through the mucosa overlying the ganglion.
- The sphenopalatine ganglion can also be approached through the greater palatine foramen located at the posterior portion of the hard palate. In this approach, the patient is positioned in the supine position with the

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neck extended. The foramina can be palpated medial to the gumline of the third molar. A small-gauge needle is advanced <2.5 cm through the foramen in a superior and slightly posterior direction. To avoid an intravascular injection, aspirate prior to injection.

Glossopharyngeal Nerve

- The glossopharyngeal nerve (CN IX) exits the skull at the jugular foramen and passes between the internal jugular vein and the internal carotid artery. It descends just dorsal to the styloid process before curving forward and anterior to innervate the palatine tonsil, the mucous membranes of the fauces and the base of the tongue. This nerve has motor, sensory, and autonomic components, and supplies lower motor neurons to the stylopharyngeus and parasympathetic innervation of the parotid and mucous glands.

Superior Laryngeal Branch of the Vagus Nerve

- The superior laryngeal nerve can be blocked as it passes into the thyrohyoid membrane inferior to the greater cornu of the hyoid bone and superior to the greater cornu of the thyroid cartilage. This block will provide anesthesia to the glottis above the vocal folds.
- With the patient seated in an upright (high Fowler position) with a towel roll transversely laid behind the shoulders, the thyroid cartilage is palpated. It can be helpful to lightly displace the thyroid cartilage toward the side of the block. Using a small-gauge needle, 2 to 3 mL of local anesthetic is injected near the cartilaginous greater cornu. Aspiration prior to injection will confirm that the needle has not entered the supraglottic air column. This procedure is then repeated on the other side.

Topical Anesthesia of the Subglottic Airway

- The recurrent laryngeal branch of the vagus nerve pierces the subglottic trachea to innervate all the laryngeal muscles other than the cricothyroid muscle as well as provide sensory innervation to the subglottic mucosa.
- The patient is positioned in a high Fowler position with a towel roll laid transversely behind the shoulders. Moderate neck extension is helpful. After skin disinfection, the thyroid cartilage is identified. The next palpable cartilage inferiorly is the cricoid cartilage. The palpable gap between these two structures overlies the cricothyroid membrane. A 22-gauge needle containing local anesthetic (2 to 4 mL 3% chloroprocaine or 2 to 4 mL 4% lidocaine) is advanced perpendicular to the skin while gentle aspiration is applied to the syringe plunger. Air will be freely aspirated when the needle penetrates the cricothyroid membrane, entering the trachea. The patient should be alerted that the injection will induce coughing. The local anesthetic should be rapidly administered and the needle withdrawn. The patient will cough and should be encouraged to do so several times to enhance spread of the anesthetic.

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1.4.3 Anesthesia Drugs

◆ Opioids

Opioids work by binding to specific receptors located throughout the central nervous system (CNS) and other tissues (**Table 1.11**). The pharmacodynamic effect of the administered opiate depends on which receptor is bound, the affinity of the binding, and whether the receptor is activated or inhibited. There are four opioid receptors: mu, delta, kappa, and sigma (**Table 1.12**).

Morphine

Because morphine is a hydrophilic compound it has a slower onset with a longer clinical effect. Morphine can lead to hypotension secondary to histamine-induced vasodilation as well as decreased sympathetic tone. Morphine is metabolized in the liver to morphine 3-glucuronide and morphine 6-glucuronide, metabolites that are excreted by the kidneys. Patients with renal failure can have prolonged duration of action given that between 5 and 10% of morphine is excreted unchanged in the urine. Morphine 6-glucuronide can lead to respiratory depression and narcosis.

Dosing: Preoperatively, postoperatively 5–15 mg intramuscular (IM)/IV; 0.05–0.2 mg/kg IV

Fentanyl

Fentanyl is a synthetic compound with a higher lipid solubility; fentanyl has a rapid onset and short duration of action. Because of fentanyl's high lipid solubility it can accumulate in adipose tissue creating a "reservoir." Prolonged respiratory depression is seen after large doses or prolonged fentanyl infusions.

Dosing: 0.5–1 μg/kg IV, 1–5 μg/kg per hour infusion. Larger doses may be used during general anesthesia.

Remifentanil

Remifentanil is an ultrafast acting narcotic with an elimination half-life of less than 10 minutes. The ester structure of this drug renders it susceptible to metabolism by plasma and tissue esterases. Remifentanil is metabolized so efficiently that during long infusion times the drug is not allowed to accumulate in adipose or other tissues. The length of infusion time therefore

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Table 1.11 Opioid Effects on Organ Systems

Organ System	Physiologic Response
Cardiovascular	In general, opioids do not impair cardiovascular function. At high doses opioids can lead to bradycardia. Meperidine, structurally similar to atropine, can cause tachycardia. Hypotension seen with opioid use is secondary to bradycardia and in the case of morphine and meperidine histamine-induced vasodilatation.
Respiratory	Opioids depress ventilation, particularly respiratory rate. The hypoxic drive, the body's ventilatory response to CO_2, is decreased. The result is an increase in $PaCO_2$ and decreased respiratory rate. The apneic threshold, the highest $PaCO_2$ at which a patient will remain apneic, is increased. In patients susceptible to histamine-induced reactive airway disease, morphine and meperidine can lead to bronchospasm. Chest wall rigidity, severe enough to prevent adequate ventilation, can be seen with fentanyl and remifentanil.
Central nervous system	Opioids reduce cerebral oxygen consumption, cerebral blood flow and intracranial pressure. Minimal changes are seen on EEG except for meperidine, which can cause an increase in EEG frequency. Even at high doses opioids do not reliably produce amnesia. The high doses necessary to establish unconsciousness can lead to physical dependence.
Gastrointestinal	Opioids slow peristalsis resulting in decreased gastric emptying and constipation. Contraction of the biliary sphincter leading to biliary colic is also common.
Endocrine	Opiates block the release of catecholamines, ADH, and cortisol associated with surgical stress.
Drug interactions	Combined with opiates barbiturates, benzodiazepines, and other CNS depressants have a synergistic effect on level of sedation and respiratory depression.

Abbreviations: ADH, antidiuretic hormone; CNS, central nervous system; EEG, electroencephalogram.

has no effect on wake-up time. Because the biotransformation of remifentanil is extrahepatic, narcotic toxicity can be avoided in patients with hepatic dysfunction.

Dosing: 0.025–0.2 μg/kg per minute infusion

Meperidine

Meperidine (Demerol, Sanofi-Aventis Pharmaceuticals, Paris, France) is *N*-demethylated in the liver to normeperidine, an active metabolite that is associated with myoclonus and seizures. Patients with renal disease are particularly susceptible as this metabolite is renally cleared. Meperidine

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Table 1.12 Opioid Receptors

Receptor	Physiological Characteristics
Mu	μ1 receptor is responsible for producing analgesia, miosis, nausea/vomiting, urinary retention, and pruritus. The endogenous stimulus for μ1 receptors are enkephalins. μ2 receptor activation leads to euphoria, respiratory depression, sedation, bradycardia, ileus, and physical dependence.
Delta	Activation leads to analgesia and contributes to physical dependence. These receptors are highly selective for endogenous enkephalins.
Kappa	Activation leads to analgesia, sedation, dysphoria, and psychomimetic effects. Pure Kappa agonists do not lead to respiratory depression. Stimulation leads to vasopressin release and subsequent diuresis.
Sigma	Activation leads to dysphoria, hallucinations, tachypnea, and mydriasis.

is useful in the postanesthesia care unit (PACU) setting in the treatment of shivering. Note that postoperative oral meperidine use/prescription is discouraged due to adverse side effects.

Dosing: Preoperatively; postoperatively 50–150 mg IM/IV

◆ Reversal of Opioids

Naloxone

Naloxone is a competitive antagonist at opioid receptors, particularly the mu class. There is no significant agonist activity. Naloxone is indicated in cases of narcotic overdose. Respiratory depression secondary to narcotic overdosage is rapidly reversed with naloxone (1–2 minutes). Care should be taken to titrate low doses as abrupt reversal of analgesia can result in abrupt sympathetic stimulation and acute withdrawal symptoms in those who are opioid dependent. Naloxone has a short duration of action (30–40 minutes) and redosing is usually required when reversing long-acting opioids.

Dosing: 0.5–1 μg/kg every 5–10 minutes; maximum total dose of 10 mg

◆ Benzodiazepines

Benzodiazepines interact with specific receptors in the CNS that enhance the action of specific neurotransmitters (**Table 1.13**). In particular, the action of gabapentin (GABA), an inhibitory neurotransmitter, is enhanced. As a result, benzodiazepines produce amnesia, anxiolysis, sedation, and prevent seizures. Benzodiazepines have no direct analgesic properties. Like barbiturates, benzodiazepines are highly protein-bound and rely on redistribution for their offset of action. Biotransformation of benzodiazepines occurs in the liver and metabolites are excreted mainly in the urine. Duration of action may be prolonged in patients with renal failure as metabolites are pharmacologically active.

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Table 1.13 Benzodiazepine Effects on Organ Systems

Organ System	Physiologic Response
Cardiovascular	Minimal depression of the cardiovascular system even at high doses. Slight decreases in arterial blood pressure, cardiac output, and peripheral vascular resistance are often observed. Midazolam decreases arterial blood pressure and peripheral vascular resistance more than lorazepam or diazepam. Variability in heart rate during administration suggests that Midazolam has vagolytic properties.
Respiratory	Ventilatory response to CO_2 is reduced with benzodiazepine administration. This response is particularly pronounced when administered with other respiratory depressants, such as opioids.
Neurologic	Benzodiazepines are very effective in seizure prophylaxis. Cerebral oxygen consumption and cerebral blood flow are reduced.
Drug interactions	When administered with opiates, benzodiazepines work synergistically to depress ventilation. When administered with heparin, diazepam is displaced from its protein binding sites causing an increased free drug concentration. The MAC of volatile anesthetics is reduced up to 30%. CNS depressants such as alcohol and barbiturates potentiate the sedative effects of benzodiazepines.

Abbreviations: CNS, central nervous system; MAC, minimum alveolar concentration.

Diazepam

Diazepam is insoluble in water and requires propylene glycol, which may cause venous irritation. Diazepam has a long duration of action secondary to slow hepatic extraction and a large volume of distribution. The elimination half-life is nearly 30 hours.

Dosing: 5–20 mg orally (PO), 2–10 mg IV

Midazolam

At low pH, midazolam is water soluble. At physiologic pH, midazolam becomes more lipid soluble resulting in fast onset of action. Midazolam has the shortest elimination half-life (2 hours) because of a high hepatic extraction ratio. Midazolam's high potency and steep dose response curve require careful titration and close monitoring of respiration as even small doses can lead to apnea.

Dosing: Adult 3–5 mg IM, 0.5–5 mg IV; pediatric 0.025–0.1 mg/kg IV; 0.25–0.5 mg/kg PO 30 minutes before procedure

Lorazepam

Like diazepam, lorazepam is insoluble in water and requires propylene glycol, which may cause venous irritation when administered. Because of

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its moderate lipid solubility lorazepam has a slower onset of action secondary to slower brain uptake. The lower lipid solubility of lorazepam limits its volume of distribution and decreases its elimination half-life (15 hours) despite the same hepatic extraction ration of diazepam.

Dosing: Adult 1–4 mg PO/IM/IV; pediatric 0.05 mg/kg PO/IM/IV preoperatively

◆ Benzodiazepine Reversal

Flumazenil

Flumazenil is a specific benzodiazepine receptor antagonist that reverses most of the effects of benzodiazepines. Onset is rapid (<1 minute) for the hypnotic effects of benzodiazepines. The amnestic effect is less reliably reversed. This agent is a competitive inhibitor at the benzodiazepine receptor. Elderly patients in particular are prone to resedation and should be observed for respiratory depression after flumazenil administration.

Dosing: 0.2 mg (titrate every minute until desired degree of sedation reversal is observed). Sedation reversal typically requires 0.6–1.0 mg. Infusions at 0.5 mg/h are indicated for overdoses of long-acting benzodiazepines.

◆ Alpha-2 Agonists

Dexmedetomidine

Dexmedetomidine (brand name Precedex, Hospira, Inc., Lake Forest, IL) is an $\alpha 2$ agonist with 8 to 10 times greater receptor affinity than clonidine. It has sympatholytic, analgesic, and sedating properties. The effect on the cardiovascular system is to lower heart rate and blood pressure, blunting the typical surgical response. $\alpha 2$ Agonists have minimal respiratory depression. Head and neck surgeons will find this drug useful for conscious sedation cases, augmented sleep studies, and fiberoptic intubations and tracheotomy placement. Also of interest to the otolaryngologist who employs the use of topical cocaine intraoperatively, recent research has suggested dexmedetomidine to be an effective treatment for the dangerous cardiovascular symptoms of cocaine intoxication.

Dosing: Loading dose of 1 μg/kg over 10 minutes followed by IV infusion at 0.2–0.7 μg/kg per hour

◆ Anesthesia Induction Medications

Propofol

Propofol is a rapidly acting induction agent, which produces unconsciousness within 30 seconds of dosing and effects last between 2 to 8 minutes (**Table 1.14**). Propofol enhances the inhibitory action of GABA. Propofol is metabolized in the liver; however, offset of action results from redistribution, which is rapid secondary to high lipid solubility. Compared with other induction agents, propofol provides a faster recovery with less "hangover"

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Table 1.14 Effects of Propofol on Organ Systems

Organ System	Physiologic Response
Cardiovascular	Increased peripheral vasodilatation, decreased cardiac contractility, and decreased preload combine to cause hypotension. The normal vagal response to hypotension is also impaired. The hypotension with propofol is greater than that produced by barbiturates.
Respiratory	Respiratory depression
Central nervous system	Reduced intracranial pressure by reducing cerebral blood flow

than barbiturates or etomidate. Additionally, this agent has antiemetic, antipruritic and anticonvulsion properties. At low (subhypnotic) doses (10–15 mg) propofol can ameliorate nausea and vomiting. Propofol does not provide analgesia, but does enhance the analgesic effects of narcotics. Careful titration is advised in hypovolemia or coronary vascular disease, as propofol can lead to a profound decrease in blood pressure secondary to decreased systemic vascular resistance. Venous irritation with administration can be avoided with concomitant administration of lidocaine (20–80 mg). Because propofol is an emulsion it should be avoided in patients with disorders in lipid metabolism.

Dosing: Induction 2–2.5 mg/kg IV (pediatric dosing: 2.5–3.5 mg/kg); infusion 100–200 μg/kg per minute; sedation 25–75 μg/kg per minute

Etomidate

Etomidate depresses the reticular activation system by binding to the GABA receptors and enhancing the inhibitory effects of this neurotransmitter (**Table 1.15**). Etomidate is dissolved in propylene glycol leading to pain on injection. This can be reduced by injecting lidocaine prior to induction. Myoclonic movements are common after etomidate induction. Etomidate is characterized by a rapid onset secondary to high lipid solubility at physiologic pH. Etomidate is metabolized into inactive end products by hepatic microsomal enzymes and plasma esterases. Metabolites are excreted in the urine. Etomidate has very little effect on the cardiovascular system and is therefore the induction agent of choice in cardiovascular disease and severe hypovolemia.

Dosing: induction 0.2–0.6 mg/kg

Ketamine

Ketamine has multiple effects through the CNS and has been demonstrated to be an *N*-methyl-D-aspartate (NMDA) antagonist (**Table 1.16**). By effectively "disconnecting" the thalamus from the limbic system, a state of "dissociative anesthesia" results. In this state the patient appears conscious,

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Table 1.15 Effects of Etomidate on Organ Systems

Organ System	Physiologic Response
Cardiovascular	Slight decrease in peripheral vascular resistance leads to slight decrease in arterial blood pressure. Myocardial contractility and cardiac output are unchanged.
Respiratory	Ventilation is reduced less than with other induction agents. Even at induction doses, apnea usually does not occur unless opioids are also administered.
Central nervous system	Cerebral metabolic rate, cerebral blood flow, and intracranial pressure are reduced. Due to cardiovascular stability, cerebral perfusion pressure is maintained. Etomidate lacks analgesic properties.
Endocrine	Transient inhibition of enzymes responsible for cortisol and aldosterone synthesis occurs with intubation doses. Long-term infusions lead to adrenocortical suppression.

but is unable to process or respond to sensory stimulation. Ketamine is a structural analogue to phencyclidine (PCP), and as with PCP, hallucinations can ensue even at low doses. Ketamine is metabolized in the liver, resulting in pharmacologically active metabolites (norketamine). Products of hepatic metabolism are renally excreted.

Dosing: 1–2 mg/kg IV; 3–5 mg/kg IM

Table 1.16 Effect of Ketamine on Organ Systems

Organ System	Physiologic Response
Cardiovascular	Increased cardiac output, heart rate, and blood pressure secondary to increased sympathetic outflow
Respiratory	Minimally effects ventilatory drive Ketamine is a potent bronchodilator and has benefits to patients with asthma. Increased salivation can be resolved by pretreatment with anticholinergic medications.
Central nervous system	Increased cerebral blood flow, cerebral oxygen consumption, and intracranial pressure Hallucinations, delirium, and disturbing dreams are decreased in children and those who receive benzodiazepines prior to ketamine. Ketamine produces analgesia, amnesia, and unconsciousness.

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Barbiturates

Barbiturates have several sites of action resulting in suppression of excitatory neurotransmitters and activation of the inhibitory effects of GABA. The result is inhibition of the reticular activation system. Methohexital and thiopental are the commonly used barbiturates used for induction. As more titratable induction agents have come into use, barbiturates have fallen out of favor. Barbiturates have no analgesic properties and cause dose-related depression of the respiratory, cardiac, and central nervous systems. Thiopental has a short duration of action secondary to a high rate of redistribution from the brain to inactive tissues, which secondary to a high lipid solubility. Barbiturates are contraindicated in patients with intermittent porphyria. Side effects include venous irritation, myoclonus, and hiccupping.

Dosing: Thiopental 3–6 mg/kg IV; methohexital 1–2 mg/kg IV

◆ Inhaled Anesthetics

In the OR, general anesthesia is commonly maintained with inhaled anesthetics. These agents also provide some analgesia, amnesia, and muscle relaxation. In pediatric patients in whom there is no IV access, anesthesia may be induced by inhalation. All of the inhaled anesthetics, with the exception of nitrous oxide, are bronchodilators and may be useful in those with reactive airways. Most inhaled agents reduce blood pressure. The onset of anesthetic induction as well as emergence from anesthesia is based on the lipid solubility characteristics of the inhaled anesthetic: the more insoluble the anesthetic agent, the faster the induction of anesthesia. The agents with high lipid solubility prolong the emergence from anesthesia.

Minimum alveolar concentration (MAC) is defined as the alveolar concentration at which 50% of test subjects will not respond to a surgical stimulus. Dosing of inhaled agents is based on the MAC of each particular agent. The MAC of inhaled agents depends on the individual gas properties of each agent. MAC is additive such that ½ MAC isoflurane combined with ½ MAC nitrous oxide is equivalent to one MAC of sevoflurane.

Isoflurane

Compared with other inhaled anesthetics (sevoflurane, desflurane), isoflurane has a relatively high lipid solubility, leading to increased induction and emergence time. Isoflurane causes minimal cardiac depression and decreased blood pressure secondary to decreased systemic vascular resistance. Like other volatile anesthetics, isoflurane causes respiratory depression with a decrease in minute ventilation (**Table 1.17**). Despite its ability to cause airway irritation, isoflurane induces bronchodilation.

Desflurane

Other than the substitution of a fluoride atom for a chloride atom, the structure of desflurane is very similar to that of isoflurane. This composition makes desflurane highly insoluble. Because of its low lipid solubility,

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Table 1.17 Physiologic Effects of Isoflurane

Organ System	Physiologic Response
Cardiovascular	Decreased systemic vascular resistance leads to lower arterial blood pressure.
Respiratory	Can cause airway irritation and cause bronchodilation Respiratory depression
Neurologic	At high concentrations, increased cerebral blood flow and intracranial pressure can develop. Decreased cerebral metabolic oxygen requirements may provide cerebral protection.
Neuromuscular	Skeletal muscle blood flow is increased.
Renal	Decreases renal blood flow, glomerular filtration rate, and urinary output.

induction and emergence from anesthesia are rapid. The time required for patients to awaken is approximately half as long as that observed following isoflurane administration. Desflurane has cardiovascular and cerebral effects similar to those of isoflurane. Like isoflurane this agent is irritating to the airway making gas induction difficult.

Sevoflurane

Sevoflurane is the primary inhaled anesthetic agent used in anesthesia induction when an IV induction cannot be performed, such as in pediatric patients. Nonpungency and a rapid increase in alveolar anesthetic concentration make it an excellent choice where inhalational induction is necessary. The blood solubility of sevoflurane is slightly greater than that of desflurane. Sevoflurane mildly depresses myocardial contractility and systemic vascular resistance. Arterial blood pressure declines slightly less than with isoflurane or desflurane. Like isoflurane and desflurane, sevoflurane causes slight increases in cerebral blood flow and intracranial pressure.

Nitrous Oxide

The uptake and elimination of nitrous oxide are relatively rapid compared with other inhaled anesthetics. This is the result of its low blood–gas partition coefficient. Nitrous oxide produces analgesia, amnesia, mild myocardial depression, and mild sympathetic nervous system stimulation. It does not significantly affect heart rate or blood pressure. Nitrous oxide is a mild respiratory depressant, although less so than the volatile anesthetics. The elimination of nitrous oxide is via exhalation.

◆ Muscle Relaxation

Neuromuscular blocking agents are used most commonly for facilitation of endotracheal intubation and when patient movement is detrimental to

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the surgical procedure. Prior to administration, ventilation must be ensured by the anesthesiologist. Ventilation can be achieved with a mask until the endotracheal tube is placed in the trachea. Neuromuscular blockers have no intrinsic sedative or analgesic properties and must be used in concert with anesthetic agents. Inadequate sedation and hypnosis while using neuromuscular blockers can produce recall by patients causing long-term side effects. There are two classifications of neuromuscular blocking agents: depolarizing and nondepolarizing.

Depolarizing Muscle Relaxants

Depolarizing agents have a similar chemical structure to acetylcholine. They induce paralysis by binding to acetylcholine receptors at the skeletal muscle neuromuscular junction causing depolarization. Paralysis ensues because these agents have a higher affinity for the postsynaptic receptor preventing the reestablishment of its ionic gradient. Clinically, fasciculations are seen prior to relaxation after dosing. The only medication in this class that is still in use today is succinylcholine (**Table 1.18**).

Nondepolarizing Muscle Relaxants

Nondepolarizing muscle relaxants induce paralysis by binding to the postsynaptic receptor at the skeletal muscle neuromuscular junction. Essentially, these medications compete with acetylcholine for binding sites

Table 1.18 Depolarizing Muscle Relaxant

Agent	Intubating Dose, Onset, Duration	Clinical Considerations
Succinylcholine	Dose: 1–1.5 mg/kg Onset: 30–60 s Duration: <10 min Maximum: 150 mg total dose	Succinylcholine is contraindicated for routine intubation in pediatric patients because of the risk of cardiac arrest with hyperkalemia in those with undiagnosed myopathies. Agent of choice for rapid sequence induction. Bradycardia can follow dosing, particularly in pediatric patients. Fasciculations with receptor activation typically occur and can result in myalgias postop. Potassium release with succinylcholine-induced depolarization can increase by 0.5 mEq/dL. Avoid in hyperkalemic patients. Transient increases in intracranial and intraocular pressure Avoid in patients with a history of malignant hyperthermia Metabolized by pseudocholinesterase

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Table 1.19 Commonly Used Nondepolarizing Muscle Relaxants

Agent	Intubating Dose, Onset, Duration	Clinical Considerations
Rocuronium	Dose: 0.5–0.9 mg/kg IV Onset: 1–2 min Duration: 40–90 min	At doses of 0.9–1.2, rocuronium can have rapid onset (60–90 s) and substituted for succinylcholine for rapid sequence induction. Rocuronium undergoes no metabolism and is eliminated in the bile and slightly by the kidneys. Severe hepatic failure and pregnancy can prolong duration of action.
Vecuronium	Dose: 0.1 mg/kg IV Onset: 2–3 min Duration: 25–30 min	Vecuronium has a short duration of action. Hepatic metabolism with renal excretion Lacks hemodynamic side effects Can be administered as an infusion (1–2 μg/kg/min)
Cisatracurium	Dose: 0.2 mg/kg IV Onset: 1–2 min Duration: 50–60 min	Cisatracurium undergoes organ-independent Hoffman degradation at physiologic pH and temperature. May be safely administered to patients with renal or liver failure Lacks hemodynamic side effects
Pancuronium	Dose: 0.1 mg/kg IV Onset: 5 min Duration: 60–100 min	Provides long duration of action Renal elimination Has vagolytic effects causing tachycardia, particularly after bolus administration

at the receptor. Unlike depolarizing blockade, the postsynaptic receptors are not activated and fasciculations do not occur. Nondepolarizing blockade can be reversed by increasing the acetylcholine concentration at the neuromuscular junction. This is achieved by administration of medications such as neostigmine, which prevent the breakdown of acetylcholine. The commonly used nondepolarizing agents are summarized in **Table 1.19**.

Further Reading

Barash PG, Cullen BF, Stoelting RK, eds. Clinical Anesthesia. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009

Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York: McGraw-Hill Medical; 2005

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1.4.4 Anesthetic Emergencies

Potential anesthetic emergencies are numerous. Emergencies related to management of the airway are covered in Chapters 1.2 and 6.1. Importance of prevention, early recognition, and prompt appropriate management are common to all potential anesthetic emergencies. In particular, airway fire and malignant hyperthermia are conditions with the potential for high morbidity or mortality and are thus reviewed here.

◆ Airway Fires

The prevention of airway fires is crucial. Lower oxygen concentration to 21 to 40% FiO_2 as tolerated when using cautery intraorally (i.e., tonsillectomy) or a laser. Laser precautions such as the use of a laser-safe endotracheal tube and packing and covering surrounding field with wet gauze or towels must be standard. Be sure there is not an endotracheal tube cuff leak. Turn off oxygen temporarily when using cautery during facial procedures requiring sedation and nasal cannula. Communication between the anesthetist and the surgeon is crucial. If there is a fire, turn off the oxygen, turn off the nitrous oxide, immediately remove the endotracheal tube, extinguish and remove any burning material, and reintubate the patient.

◆ Malignant Hyperthermia

Malignant hyperthermia (MH) is a hyperdynamic crisis caused by an uncontrolled increase in skeletal muscle metabolism (**Table 1.20**). The underlying pathophysiology is an inability of the sarcoplasmic reticulum to control intracellular calcium causing prolonged activation of muscle contractile units. This results in heat production, oxygen consumption, and acidosis. MH is an autosomal dominant myopathy that is triggered by inhalational anesthetics and depolarizing muscle relaxants (e.g., succinylcholine). "Triggers" should be avoided in those who have a history of MH or a family history of the disorder. Patients with a history of MH can be safely anesthetized with a "nontriggering anesthetic," which includes IV agents (TIVA) and nondepolarizing agents. Complications from MH are

Table 1.20 Signs of Malignant Hyperthermia

Unexplained tachycardia, tachypnea, and/or rigidity
Increased end tidal carbon dioxide
Hyperthermia
Hypoxia/cyanosis
Arrhythmias
Hyperkalemia

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Table 1.21 Treatment of Malignant Hyperthermia

1. Stop anesthesia and surgery
2. Hyperventilate with 100% oxygen
3. Administer dantrolene 2 mg/kg every 5 min to 10 mg/kg
4. Treat hyperthermia: institute cooling measures
5. Treat hyperkalemia: insulin (10 units) with glucose (50 mL D50)
6. Treat acidosis: bicarbonate 2–4 mEq/kg slow intravenous push
7. Treat arrhythmias
8. Invasive monitoring: Foley catheter, arterial line, central venous catheter
9. Administer high volumes of crystalloid, as tolerated

severe: they include cardiac arrest, cerebral or pulmonary edema, renal failure, diffuse intravascular coagulation (DIC), and death. Treatment is outlined in **Table 1.21**.

Further Reading

Barash PG, Cullen BF, Stoelting RK, et al. Clinical Anesthesia. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009

Morgan GE, Mikhail MS, Murray MJ. Clinical Anesthesiology. 4th ed. New York: McGraw-Hill Medical; 2005

1.5 Fluids and Electrolytes

◆ Key Features

- Appropriate fluid balance perioperatively can be estimated from established formulas.
- Attention to fluids and electrolytes is especially important in the patient who is not capable of maintaining normal oral intake.

Surgical patients, especially head and neck cancer patients, may be incapable of adequate oral intake. Often, IV fluids are needed for a short period before postoperative oral intake can resume. In other cases, longer-term NPO status is required, such as when a pharyngeal closure must heal, or a fistula is resolving. An important rule is that if the gut is available and functional, it should be used, i.e., if oral swallowing is not functional, one should provide intake via a nasogastric (NG) tube or percutaneous endoscopic gastrostomy (PEG) tube rather than by total parenteral nutrition (TPN) or peripheral IV nutrients. A summary of important considerations for fluid management is provided here.

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◆ Functional Compartments

Total Body Water (TBW)

60% (adult males), 50% (adult females) of ideal body weight (IBW)

Intracellular Fluid (ICF)

Comprises 35% of IBW or 60% of TBW
This is the principle potassium-containing space.

Extracellular Fluid (ECF)

25% of IBW or 40% of TBW; subdivided into interstitial fluid (ISF) and blood volume (BV is ~8% of total volume weight)
This is the principle sodium-containing space.

◆ Daily Electrolyte Requirements

Sodium: 2–3 mEq/kg per day
Potassium: 1–2 mEq/kg per day
Chloride: 2–3 mEq/kg per day
See **Table 1.22**.

◆ Perioperative Fluid Management

Intraoperatively, there are four aspects of fluid management that should be considered: maintenance requirement, fluid deficit, third space loss, and blood loss.

Maintenance Requirement

(Rough estimate, based on weight)

- First 10 kg: 4 mL/kg per hour
- Second 10 kg: add 2 mL/kg per hour
- Above 20 kg: add 1 mL/kg per hour (Note: for patients above 20 kg, add 40 to weight to get maintenance rate in mL/kg per hour).

Table 1.22 Electrolyte Composition of Commonly Used Perioperative Fluids

Fluid	Glu	Na	Cl	K	Ca	HCO_3	Kcal/L
D5W	50						170
NS		154	154				
D5¼NS	50	38.5	38.5				170
LR		130	110	4	3	27	<10

Abbreviations: D5W, 5% dextrose in water; NS, normal saline; LR, lactated Ringer's solution; D5¼NS, 5% dextrose in ¼ normal saline.

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Fluid Deficit

Replaces NPO deficit: hourly maintenance rate × number of hours NPO
Administer half of the deficit over the first hour intraoperatively and the remaining half over the next 2 hours.

Third Space (Redistribution Loss)

Made up of third space (redistribution) and evaporative losses; amount based on tissue trauma.

- Minimal: 0–2 mL/kg per hour
- Moderate: 2–4 mL/kg per hour
- Severe: 4–8 mL/kg per hour

Blood Loss (see also Chapter 1.6)

Replace each milliliter of blood loss with 3 mL of crystalloid, 1 mL colloid solution, or 1 mL packed red blood cells (PRBCs). Transfusion of RBCs as necessary to maintain desired hematocrit (Hct).

◆ Calcium Disturbances

- Normal plasma concentration is 8.5–10 mg/dL with 50% free ionized and 40% protein bound
- Normal free ionized concentration is 4.5–5 mg/dL
- Corrected calcium = measured calcium / [0.6 + (total protein) / 8.5]
- Ionized calcium increases 0.16 mg/dL for each decrease of 0.1 unit in plasma pH

Further Reading

Silbernagl S, Despopoulos A. Color Atlas of Physiology, 6th ed. Stuttgart/New York: Thieme; 2009
Sircar S. Principles of Medical Physiology. Stuttgart/New York: Thieme; 2008

1.6 Hematology for the Otolaryngologist

◆ Key Feature

- Screening for, and correction of, hematologic disorders may be required for head and neck surgery patients.

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An overview of blood components, disorders, and transfusion complications is provided in this chapter.

◆ Blood Loss Management

Estimated Blood Volume (EBV)

- 95–100 mL/kg for premature infant
- 85–90 mL/kg for full-term infant
- 80 mL/kg for infants up to infants up to 12 months
- 70–75 mL/kg for adult males
- 65–70 mL/kg for adult females

$$\text{Allowable blood loss} = [\text{EBV} \times (\text{Hct-target Hct})]/\text{Hct}$$

Replace every 1 mL blood loss with 3 mL crystalloid, 1 mL colloid, or 1 mL PRBC

PBRC Transfusion Guidelines

- One unit PRBC increases Hct ~3% and hemoglobin (Hb) ~1 g/dL in adults
- 10 mL/kg PRBC increases Hct ~10%

Compatibility Testing

- **Type specific:** ABO-Rh typing only; 99.80% compatible
- **Type and screen:** ABO-Rh and screen; 99.94% compatible
- **Type and crossmatch:** ABO-Rh screen, and crossmatch; 99.95% compatible. Crossmatching confirms ABO-Rh typing, detects antibodies to the other blood group systems and detects antibodies in low blood titers.
- **Screening donor blood:** hematocrit is determined; if normal, the blood is typed, screened for antibodies, and tested for hepatitis B, hepatitis C, syphilis, human immunodeficiency virus-1 (HIV-1), HIV-2, and human T cell lymphotropic viruses 1 and 2.

Blood Component Therapy

The archaic perioperative axiom of transfusing patients to maintain Hb of 10 and a hematocrit of 30 has fallen by the wayside. Although these are indeed safe guidelines for patients with coronary artery disease, transfusions are currently guided by hemodynamics, intraoperative blood loss and laboratory values such as the arterial blood gas.

- **Whole blood:** 40% hematocrit; used primarily in hemorrhagic shock
- **PRBCs:** each unit has a volume 250–300 mL with a hematocrit of 70–85%.
- **Platelets:** a normal platelet count is 150,000–400,000/mm^3. Thrombocytopenia is defined as <150,000/mm^3. Intraoperative bleeding increases

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with counts between 40,000 and 70,000/mm^3, and spontaneous bleeding can occur with counts <20,000/mm^3.

During most surgeries, platelet transfusions are probably not needed unless the count is less than 50,000/mm^3. One unit of platelets will increase platelet count 5000–10,000/mm^3. The usual dose is 1 unit of platelets per 10 kg body weight.

Platelets are stored at room temperature; ABO compatibility is not necessary.

- **Fresh frozen plasma (FFP)**: acute reversal of warfarin requires 5–8 mL/kg of FFP. ABO compatibility is mandatory. A 250 mL bag contains all coagulation factors except platelets. 10–15 mL/kg will increase plasma coagulation factors to 30% of normal. Fibrinogen levels will increase by 1 mg/mL of plasma transfused.
- **Cryoprecipitate:** Indications include hypofibrinogenemia, von Willebrand disease, and hemophilia A. ABO compatibility is not necessary. Each 10–20 mL/bag contains 100 units of factor VIII-C, 100 units von Willebrand factor (vWF), 60 units factor XIII, and 250 mg fibrinogen.

◆ Massive Transfusions

A massive transfusion is defined as the replacement of a patient's total blood volume in less than 24 hours. It also applies to the acute administration of more than half the patient's estimated blood volume per hour.

◆ Universal Donor Blood

Group O, Rh-negative blood should be reserved for patients close to exsanguination. If time permits, crossmatched or uncrossmatched type-specific blood should be administered. Group O, Rh-negative blood should not be given as whole blood. The serum contains high anti-A and anti-B titers, which may cause hemolysis of recipient blood.

If more than 4 units of group O, Rh-negative whole blood is administered, type-specific blood should not be given subsequently because the potentially high anti-A and anti-B titers could cause hemolysis of the donor blood.

Patients administered up to 10 units of group O, Rh-negative PRBCs may be switched to type-specific blood, since there is an insignificant risk of hemolysis from the small volume of plasma administration with PRBCs.

◆ Complications of Transfusions

Immune Reactions (Hemolytic versus Nonhemolytic)

Hemolytic Reactions

● Acute Hemolytic Reaction. An acute hemolytic reaction occurs when ABO-incompatible blood is transfused, resulting in acute intravascular hemolysis; the severity of a reaction often depends on how much incompatible

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blood has been given. Symptoms include fever, chills, chest pain, anxiety, back pain, dyspnea; in anesthetized patients, the reaction may present with fever, tachycardia, hypotension, hemoglobinuria, and diffuse oozing in the surgical field. Free Hb in plasma or urine is evidence of a hemolytic reaction.

Risk of a fatal hemolytic transfusion reaction: 1:600,000 units.

- Delayed Hemolytic Reaction. Typically, this reaction is delayed 2 to 21 days after the transfusion. The symptoms are generally mild and may include malaise, jaundice, and fever; treatment is supportive. A delayed hemolytic reaction is caused by incompatibility of minor antigens (i.e., Kidd, Duffy, Kelly, etc.) causing hemolysis.

Nonhemolytic Reactions

- Febrile Reaction. A febrile reaction is the most common nonhemolytic reaction (0.5–1.0% of RBC transfusions and up to 30% of platelet transfusions). The reaction is the result of the action of recipient antibodies against donor antigens present on leukocytes and platelets; treatment includes stopping or slowing the infusion and antipyretics.

- Urticarial Reaction. A urticarial reaction occurs in 1% of transfusions; it is thought to be due to sensitization of the patient to transfused plasma proteins. It is characterized by erythema, hives, and itching without fever. Treat with antihistamine drugs.

- Anaphylactic Reaction. Anaphylactic reactions are rare (1:500,000). Patients with IgA deficiency may be at an increased risk because of the transfused IgA reaction with anti-IgA antibodies.

Transfusion-Related Lung Injury

Transfusion-related lung injury (TRALI) is due to a transfusion of antihistocompatibility leukocyte antigen (HLA) antibodies that interact with, and cause, the patient's white cells to aggregate in the pulmonary circulation. Risk: 1:6000. Treatment is supportive, using a similar therapy for managing acute respiratory distress syndrome (ARDS).

Graft-versus-Host Disease

Graft-versus-host disease is most commonly seen in immunocompromised patients. Cellular blood products contain lymphocytes capable of mounting an immune response against the compromised host.

Posttransfusion Purpura

Due to the development of platelet alloantibodies; the platelet count typically drops dramatically one week after the transfusion.

Immune Suppression

Transfusion of leukocyte-containing blood products appears to be immunosuppressive. Blood transfusions may increase the incidence of

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serious infections following surgery or trauma. Blood transfusions may worsen tumor recurrence and mortality rate following resections of many cancers.

Infectious Complications

Viral Infections

- **Hepatitis**: risk of hepatitis B virus (HBV) is 1:137,000; risk of hepatitis C virus (HCV) is 1:1,000,000
- **HIV/acquired immunodeficiency syndrome (AIDS)**: risk of HIV is 1:1,900,000
- **Cytomegalovirus (CMV) and Epstein–Barr virus**: common and usually cause asymptomatic or mild systemic illness

Bacterial Infections

Gram-positive and gram-negative bacteria contamination of blood is rare. Specific bacterial reactions transmitted by blood include syphilis, brucellosis, salmonellosis, yersiniosis, and various rickettsioses.

Parasitic Infections

Malaria, toxoplasmosis, and Chagas disease are very rare.

◆ Other Complications Related to Blood Transfusion

Metabolic Abnormalities

- Decreased pH secondary to increased hydrogen ion production
- Increased potassium: due to cell lysis; (increases with length of storage)
- Decreased 2,3 DPG: consumed by RBCs; P50 decreases to 18 mm Hg after 1 week and 15 mm Hg after 3 weeks.
- Citrate toxicity: citrate metabolism to bicarbonate may contribute to metabolic alkalosis; binding of calcium by citrate could result in hypocalcemia and the ability of the liver to metabolize citrate to bicarbonate.

Microaggregates

Microaggregates consisting of platelets and leukocytes form during the storage of whole blood. Micropore filters may help remove these particles.

Hypothermia

The use of blood warmers (except for platelets) greatly decreases the likelihood of transfusion-related hypothermia.

Coagulopathies

Coagulopathies occur after massive transfusions (>10 units).

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Dilutional Thrombocytopenia

Dilutional thrombocytopenia is a common cause of abnormal bleeding in the setting of massive transfusion. It typically responds quickly to platelet transfusion.

Factor Depletion

Factors V and VIII are very labile in stored blood and may decrease to levels as low as 15 to 20% of normal (this is usually enough for hemostasis).

Disseminated Intravascular Coagulation

Disseminated intravascular coagulation (DIC) is a hypercoagulable state caused by activation of the clotting system leading to deposition of fibrin in microvasculature causing secondary fibrinolysis, leading to consumption of platelets and factors.

◆ Treatment of Hemolytic Transfusion Reactions

1. Stop the transfusion.
2. Check for error in patient or donor identification.
3. Send donor unit and newly obtained blood sample to blood bank for re-cross match.
4. Treat hypotension with fluids and pressors as needed.
5. If transfusion is required, use type O-negative PRBC and type AB FFP.
6. Maintain the urine output at a minimum of 75 to 100 mL/h by generously administering IV fluids; consider mannitol, 12.5 to 50 g or furosemide 20–40 mg.
7. Monitor for signs of DIC clinically or with laboratory tests.
8. Send patient blood sample for direct antiglobulin (Coombs) test, free Hb, haptoglobin; send urine for Hb.

◆ Coagulation Studies

Partial Thromboplastin Time (PTT)

- By adding particulate material to a blood sample, which activates the intrinsic coagulation system, the PTT can be determined.
- PTT measures the clotting ability of all factors in the intrinsic and common pathways except factor XIII.
- PTT is abnormal (prolonged) if there are decreased amounts of coagulation factors, patient is on heparin, or if there is a circulating anticoagulant present.
- Normal values are between 25 and 37 seconds.

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Prothrombin Time (PT)

- Performed by measuring the time needed to form a clot when calcium and a tissue extract are added to plasma.
- PT evaluates the activity of fibrinogen, prothrombin, and factors V, VII, and X; evaluates the intrinsic coagulation system and vitamin K-dependent factors.
- Normal PT is 10–12 seconds (depending on control).

International Normalization Ratio (INR)

INR is a means of standardizing PT values: it is the ratio of the patient's PT to the control PT value. INR values are commonly used as a guide for Coumadin therapy.

Activated Clotting Time (ACT)

The ACT is a simple test that provides a global measurement of activator of hemostatic function and is measured after the whole blood is exposed to a specific activator of coagulation. The time for clot formation after whole blood is exposed to Celite (diatomaceous earth) is defined as the ACT.

There is a linear relationship between ACT and heparin dose; provides a convenient method to measure response to heparin anticoagulation. Normal values are 90 to 120 seconds. The ACT lacks sensitivity to clotting abnormalities.

Platelet Function

Detailed evaluation of possible platelet aggregation disorders requires complex hematology testing. However, a screening platelet function test (via the use of the PFA-100 analyzer) is available and can rapidly screen for platelet function abnormality, without identifying specific cause (e.g., recent aspirin use, von Willebrand disease). Thus, abnormal PFA-100 test should prompt detailed workup, whereas a normal test is reassuring in the setting of planned elective surgery such as tonsillectomy.

◆ Hematologic Disorders

Sickle Cell Anemia

Sickle cell anemia is a hereditary hemolytic anemia resulting from the formation of an abnormal Hb (HbS); sickle Hb has less affinity for oxygen and decreased solubility. Patients are typically asymptomatic. Vigorous physical activity, high altitude, air travel in unpressurized planes and anesthesia are potentially hazardous.

Clinical features include signs and symptoms of anemia (Hb levels 6.5–10 gm/dL), obstructive or hemolytic jaundice, lymphadenopathy, hematuria, epistaxis, priapism, finger clubbing and skeletal deformities.

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The disease manifests as periodic exaggeration of symptoms or sickle crises. There are four types of crises:

- Vasoocclusive crisis: caused by sickled cells blocking the microvasculature; characterized by sudden pain frequently without a precipitating event.
- Hemolytic crisis: seen in patients with sickle cell disease and G6PD deficiency; manifests as sudden hemolysis.
- Sequestration crisis: red blood cells are sequestered in the liver and spleen leading to splenohepatomegaly and acute fall in hematocrit. This can progress to circulatory collapse.
- Aplastic crisis: characterized by transient episodes of bone marrow depression; often occurs after viral illness.

◆ Management of Sickle Cell Anemia

The practice of transfusion to an end point of 70% HbA and less than 30% HbS preoperatively is controversial. Patients should be well oxygenated and hydrated intraoperatively to lessen the chance of sickling. Acidosis and hypothermia can also trigger a crisis and should also be avoided.

Factor Deficiencies

Factor VIII Deficiency (Hemophilia A)

The half-life of factor VIII is 8 to 12 hours. Treatment includes lyophilized factor VIII, cryoprecipitate, or desmopressin. Infusion of 1 unit of factor VIII per kg will increase the factor VIII activity by 2%. Levels of 20 to 40% are desirable prior to surgery.

Factor IX Deficiency (Hemophilia B; Christmas disease)

The half-life of factor IX is 24 hours. Therapy consists of factor IX concentrates of FFP. To ensure surgical hemostasis, activity levels of 50 to 80% are needed. Infusion of 1 unit per kg of factor IX will increase the factor IX level by 1%.

Von Willebrand Disease (VWD)

VWD is the most common of all the inherited bleeding disorders. It occurs in ~1 of every 100 to 1000 people. VWD affects both males and females, with three forms. Von Willebrand factor (VWF) is involved in platelet adhesion and in carrying factor VIII.

- Type 1 VWD: most common, usually mild; low VWF, possibly low factor VIII.
- Type 2 VWD: abnormal function of VWF; various forms.
- Type 3 VWD: rare, severe. Usually no VWF and low factor VIII.

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Testing for VWD includes platelet function assay, VWF levels, ristocetin cofactor assay to assess VWF function, factor VIII levels, VWF multimers.

Management of Von Willebrand Disease

The goal of therapy is to correct the defect in platelet adhesiveness (by raising the level of effective VWF) and the defect in blood coagulation (by raising the factor VIII level). Desmopressin (1-diamine-8-D-arginine vasopressin, DDAVP) is used to increase VWF/factor VIII release. Most experience in treating individuals with VWD is with intravenous infusion, with which the response is rapid (i.e., peak VWF levels within ~45–90 minutes of infusion). Doses may be repeated at intervals of 12 to 24 hours for continued bleeding or for postoperative use. Pediatric dosage is 0.3 μg/kg IV.

VWF/factor III infusion can be given for more severe cases. Plasma-derived factor VIII concentrates that contain VWF in high molecular weight can be used, and cryoprecipitate contains multimeric VWF. Humate-P (Centeon, L.L.C., King of Prussia, PA) and Alphanate (Grifols, S.A., Barcelona, Spain) are other forms of factor VIII/VWF. Aminocaproic acid (Amicar, Pfizer Pharmaceuticals, New York, NY) inhibits fibrinolysis via inhibition of plasminogen activator substances and, to a lesser degree, through antiplasmin activity. Amicar (pediatric dosing 100 mg/kg/dose PO every 4–6 hours) can be useful in cases of mucosal bleeding.

Further Reading

Theml H, Diem H, Haferlach T. Color Atlas of Hematology: Practical Microscopic and Clinical Diagnosis. 2nd ed. Stuttgart/New York: Thieme; 2004

1.7 Common Postoperative Problems

A deviation from expected recovery requires prompt and appropriate evaluation. Such deviations may present with a change in exam findings, a subjective complaint from the patient, or with laboratory test or vital sign anomalies. Commonly encountered postoperative problems are reviewed here.

◆ Fever

Generally, an elevated temperature ≥38.5°C requires workup. Timing is important, as postoperative fever within the first 24 hours suggests atelectasis, possibly an early wound infection, or a urinary tract infection. Other considerations for fevers, especially after 24 hours postoperatively, include drug reactions, wound abscess, sepsis, pneumonia, or an IV or central line infection. Also, a possible transfusion reaction, a deep vein thrombosis, or an infected decubitus ulcer should be considered.

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Workup

A bedside examination includes taking vitals with pulse oximetry and checking the wound for erythema, edema, fluctuance, drainage, and warmth. You should auscultate for rales or diminished breath sounds, examine IV sites for redness, and check the patient's legs for calf tenderness. If the patient has a tracheotomy, look for increased and discolored sputum. Consider possible sepsis if the patient has tachypnea, tachycardia, or hypotension. Consider ordering a chest x-ray, blood cultures, sputum cultures, and/or a urinalysis with cultures. For atelectasis/pneumonia, empiric treatment may include a chest physical exam, supplemental oxygen, and respiratory therapy with incentive spirometry, mucolytics, nebulized bronchodilators, and empiric antibiotics (determined after the results of culture specimens have been received). For the treatment of pneumonia, cephalosporin and clindamycin are recommended and for a suspected urinary tract infection (UTI), treat the patient with sulfa or fluoroquinolone. Adjust antibiotics based on culture results. Gentle IV hydration may be useful. Treat with an antipyretic such as Tylenol (Ortho-McNeil-Jansson Pharmaceuticals, Titusville, NJ), a 650-mg dose for adults (15 mg/kg/dose for children). A wound abscess will require opening the wound, draining it, initiating a gram stain with culture, and changing the packing. If other vital signs are abnormal, you may want to transfer to the patient to a monitored bed, with continuous pulse oximetry and arterial blood gas assessment.

◆ Confusion (Mental Status Change)

This is one of the most common calls in otolaryngology–head and neck surgery regarding postoperative patients. Although the possible causes for a mental status change are many, it is prudent to consider the cause to be hypoxia until proven otherwise.

Resist the request for benzodiazepine to "calm him or her down"; instead instruct the nurse to obtain a full set of vitals, including pulse oximetry. Personally visit the patient.

The differential diagnosis includes hypoxia (can be due to tracheostoma occlusion, crusting, mucus plugging, underlying severe chronic obstructive pulmonary disease [COPD]—common in heavy smokers—atelectasis, pneumonia, aspiration, overmedication with narcotics, pneumothorax, pulmonary embolism, or acute postoperative pulmonary edema), cardiac arrhythmia, alcohol withdrawal, delirium from medications, stroke, meningitis, hypoglycemia or severe hyperglycemia, sepsis, anxiety, or psychosis.

Workup

The bedside exam should include taking a full set of vitals with pulse oximetry; auscultation; an examination of the tracheostoma, if present; and a quick neurologic exam looking for focal deficits, and the patient's orientation to person, place, and time. For ancillary tests, start with arterial blood gases (ABGs), a 12-lead ECG, a fingerstick glucose test, and a portable chest

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x-ray. Acute respiratory insufficiency in the head and neck patient often presents with a low PaO_2 and elevated PCO_2; however, a PCO_2 below 40 may be seen with compensatory overventilation. Typically, the patient has an underlying chronic lung disease and has had inadequate tracheopulmonary toilet, allowing for accumulation of secretions and mucus plugging. Thus, treat with humidified supplemental oxygen and aggressive suctioning. If there is an inadequate response and other tests are normal, consider testing the patient for a pulmonary embolism (PE).

Pulmonary Embolism

In the case of a possible PE, currently a spiral CT is commonly obtained, although ventilation/perfusion (V/Q) scans can be performed and laboratory testing for D-dimer may be useful. Patients with proven PE and an otherwise stable cardiovascular status are often managed with supplemental oxygen and anticoagulation, using an IV heparin bolus of 10,000 units, with a drip at 800 to 1200 units per hour maintaining an activated partial thromboplastin time (aPTT) at 1.5 to 2.0 times normal. The patient is transferred to the ICU with continuous monitoring; if the patient becomes unstable with cyanosis, low PaO_2, cardiac arrhythmia, hypotension, and low urine output, consider intubation with ventilatory support as well as prescribing an inotropic agent.

Acute Postobstructive Pulmonary Edema

Acute postobstructive pulmonary edema (APOPE) should be suspected in patients with postoperative acute respiratory failure. The development of hypoxia, bradycardia, and pink frothy sputum is characteristic in patients with APOPE. Type I APOPE occurs with acute airway compromise usually following extubation. This develops from inspiration against a closed glottis due to laryngospasm or other obstruction. Type II APOPE develops after relief of chronic upper airway obstruction. This may occur in children or adults following surgery to correct severe obstructive sleep apnea. In both cases, a sudden decrease in intrathoracic pressure leads to increased pulmonary venous return and transudation from the capillary bed into the interstitium.

The treatment of APOPE includes intensive care monitoring and a low threshold for immediate reintubation. Positive end expiratory pressure may be necessary for adequate ventilation. Diuretics and steroids may be considered. In patients who are stable, medical management with oxygen supplementation, diuretics, and close observation may be appropriate. Careful restriction of intravenous crystalloids may also be an option.

Other Causes of Mental Status Change

If the hypoxia workup is normal, other testing may reveal an obvious cause. The ECG should rule out the possibility of cardiac arrhythmia, ST changes, or signs of cardiac ischemia or infarction. If the ECG is positive, transfer of the patient to the ICU and a consult with cardiology is mandatory. The fingerstick glucose test is a rapid way to exclude a common cause for mental

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status change, especially in known diabetics. Glucose below 40 should be treated with an ampule of IV D50 and repeated. If the neurologic exam suggests focal deficit, a brain CT scan to screen for stroke should be considered, although an MRI scan is much more sensitive. Mental status change in a patient with a high fever should prompt consideration of meningitis, especially if the patient has had skull base or otologic surgery. Workup includes a lumbar puncture, then treatment with empiric antibiotics followed by the transfer of the patient to an ICU setting (see Chapter 2.2.3 for further details regarding meningitis).

Alcohol Withdrawal

Many head and neck patients have a history of alcohol abuse, often underreported. Thus, in the absence of other obvious causes for mental status change, attempting to obtain an honest alcohol use history is important. Unrecognized delirium tremens can have up to an 8% mortality rate. Thus, the postoperative alcoholic patient must be handled properly. Typically, diazepam 5 to 10 mg every 4 hours may be needed. IV fluids may contain D10, instead of the typical D5, and also should be supplemented with a 10 mL multivitamin solution per bag. The patient should also be given thiamine daily. Check serum magnesium levels, which are typically low, and replete.

Psychiatric Disorders

Severe anxiety, delirium, or psychosis can be seen in the postoperative patient. There are many predisposing factors, such as sleep deprivation, elderly state, ICU stay, drug dependency, or metabolic alterations. It is important to exclude hypoxia or other obvious medical causes for mental status change or agitation. Psychiatric consultation, if available, may be helpful. Haldol (Ortho-McNeil-Jansson Pharmaceuticals, Titusville, NJ) 5 mg IV as needed is a reasonably safe drug to use short term, as it will decrease agitation while having little to no influence on cardiovascular status.

◆ Wound Problems

A variety of wound problems can arise following head and neck surgery, including hematoma, seroma, infection, dehiscence, development of a pharyngocutaneous fistula, exposure of the carotid artery leading to rupture or "carotid blowout," chyle leak, or reconstructive flap complications such as venous edema or arterial ischemia. As with most situations, prevention is helpful: proper preoperative assessment, management of identified risk factors such as malnutrition or coagulopathy, and meticulous surgical technique. Preoperative radiation therapy is a common issue that greatly increases the risk of healing problems.

Assessment and Management of Wound Problems

Hematoma and Seroma

In cases of hematoma and seroma, the wound will be swollen, usually fluctuant but possibly tense or discolored. A fluid collection can lead to

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infection, dehiscence, or decrease skin flap viability. A small fluid collection may resorb, or it may be simply aspirated. With a rapidly expanding or large fluid collection, the patient should be taken to the OR, the wound should be opened, irrigated, and explored to maintain hemostasis. **A hematoma following thyroid surgery is an emergency.** Most commonly, this is seen the day of surgery and presents with an expanding mass and discomfort, which may progress to dyspnea, stridor, and severe airway compromise. This is due to venous back-pressure causing the rapid development of laryngeal edema. The wound is opened immediately at the bedside and the patient is then taken to the OR to wash out the wound and establish hemostasis. Intubation may be difficult, but opening the wound should facilitate intubation. If this remains difficult, a tracheotomy should be simple to perform because the thyroidectomy has exposed the subglottic trachea.

Wound Infection

An infected neck wound is typically warm, red, swollen, and tender; it may present with purulent drainage, a fluctuant collection, or abscess formation. Complications may lead to fistula, flap necrosis, or carotid exposure. Management includes opening the wound, culture of drainage, appropriate antibiotics, and packing change. Empiric antibiotic coverage should be broad spectrum, such as Unasyn (Pfizer Pharmaceuticals, New York, NY), or cefuroxime and clindamycin. Simple wet to dry saline gauze changes twice a day may be sufficient. Clorpactin or acetic acid gauze packing have antimicrobial properties and promote granulation tissue formation. However, if there is carotid exposure, it is prudent to perform surgery to cover the carotid with vascularized tissue, such as a pectoralis flap.

Pharyngocutaneous Fistula

Salivary drainage increasing in suction drains or draining via an incision indicates development of a fistula. One can test drainage for amylase to confirm. Most commonly, poor wound healing leads to a fistula following salvage surgery in a patient with persistent or recurrent disease after radiation therapy. Again, this is managed with packing changes, antibiotics, and consideration of surgery to bring in vascularized tissue if this fails to heal. One should check prealbumin and thyroid-stimulating hormone (TSH) levels to assess for malnutrition and hypothyroidism, and correct. Using the gut when it works is a sound principle, so the patient who must be kept NPO to reduce fistula output should be fed via an NG tube or PEG. Only if this cannot be accomplished should one use TPN.

Chylous Fistula

The incidence of chylous fistula after neck dissection is reported to be between 1 and 5.8%. Chylous fistula results from injury to the thoracic duct, which is encountered in level 4 on the left. However, 25% of chyle

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leaks occur on the right. This complication is best treated if recognized intraoperatively with suture ligature or oversewing local muscle flaps. Chylous fistula is suspected postoperatively in the face of increased drain outputs. The quality of output may change from serosanguineous to a more milky/turbid consistency. This may occur with the onset of PO intake. High-output chyle loss may lead to electrolyte disturbances, hypovolemia, hypoalbuminemia, immunosuppression, chylothorax, peripheral edema, and local skin breakdown. If there is a question about the diagnosis of a chylous fistula, obtain a drain output analysis for triglycerides and chylomicrons. First-line treatment involves conservative management. This is aimed at reducing chyle flow. The patient may be started on a medium-chain fatty acid diet. If there is no improvement, a trial of NPO with TPN should be considered. Local pressure dressing should be placed. Surgical management is indicated if chylous drainage is in excess of 600 mL per day, persistent low-output drainage for an extended period of time, and electrolyte disturbances. Treatment involves wound reexploration and ligation of lymphatic channels. Alternatively, thoracic ligation or percutaneous lymphangiography-guided embolization of the thoracic duct may be useful.

◆ Carotid Artery Blowout

A carotid artery blowout is a devastating complication and efforts are aimed at prevention. If postoperative wound problems result in exposure of the carotid, it can rapidly desiccate and then rupture, either externally or into the trachea, depending upon the wound situation. Maintaining healthy vascularized tissue between the carotid and the external environment can generally prevent the problem. Preoperatively, correcting malnutrition, hypothyroidism, and stopping tobacco use are important, especially in the previously irradiated neck. Surgically, if the sternocleidomastoid muscle can be preserved, without oncologic compromise, this will help cover and protect the carotid. Avoiding placement of an incision trifurcation directly over the carotid is best. If there is postoperative wound breakdown that may threaten the carotid, prompt management with packing and prevention of desiccation is critical. It is prudent to consider "carotid precautions": maintain large-bore IV access, have a type and cross order in place with the blood bank, and keep an emergency instrument kit in the room. Moreover, if there appears to be any evidence of carotid exposure, one should proceed with surgery to bring in vascularized tissue coverage, rather than hope for healing. A pectoralis flap is ideal; a microvascular free flap is another option.

If a carotid blowout occurs, this can present first with a relatively minor sentinel bleed, which stops. Again, one should proceed with flap coverage if there has been a sentinel bleed. If there is a true blowout, there will be a profuse hemorrhage. One should establish direct firm pressure, treat with bolus IV fluids and proceed directly to the OR for hemostasis. This will involve establishment of proximal and distal control of the vessel, with a risk of stroke. Transfusion will likely be necessary,

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and if the patient can be saved, wound coverage should be performed. If the blowout occurs into a tracheotomy stoma in a laryngectomee, one may use an endotracheal tube to intubate a mainstem bronchus distal to the site of rupture, tightly inflate the cuff, and then tightly pack the stoma opening to achieve tamponade while ventilating on one lung on the way to the OR.

◆ Gastrointestinal and Genitourinary Problems

Renal Failure

Low urine output is a common postoperative issue. The problem may be considered as prerenal, renal, or postrenal. Low urine output is generally defined as less than 30 mL per hour, for the 70 kg patient. A prerenal problem means the kidney is underperfused. Usually, this is from hypotension and/or hypovolemia. One should treat with IV hydration. If there is no cardiac failure, give 500 mL of normal saline as an IV bolus. If cardiac failure exists, this must be treated to correct renal perfusion. Diuretics should be used carefully. Acute renal failure of parenchymal cause may be due to glomerulonephritis, nephrotoxicities, or acute tubular necrosis. Urine may show casts; urine osmolality is equal to plasma; and urine sodium is elevated. One must follow daily fluid intake and output carefully, and follow laboratories closely. Electrolytes and creatinine will guide the need for dialysis. Postrenal problems refer to obstructive uropathy. One must identify and correct the source of obstruction. The placement of a Foley catheter may be all that is needed; or imaging studies such as an IV pyelogram may be indicated. In any patient with low urine output or possible renal failure, one must be cautious administering potassium. If hyperkalemia exists, watch for ECG changes. Potassium >6 should be lowered. A Kayexalate (Sanofi-Aventis Pharmaceuticals, Paris, France) 15 g enema may be given; also IV glucose can be given, along with 10 units of regular insulin to rapidly lower plasma potassium.

Diarrhea

The main concern in hospitalized patients with diarrhea is the possibility of *Clostridium difficile* colitis, also known as pseudomembranous colitis. This is an anaerobic spore-forming bacteria that is highly transmissible, especially by health care workers with poor hand washing. Stool samples should be sent for the *C. difficile* toxin assay in triplicate. Typically, colonic flora has been reduced by antibiotic use, leading to the *C. difficile* infection. Thus, it is important to stop antibiotics whenever possible, and to use them appropriately. Patients with *C. difficile* are treated with Flagyl (Pfizer Pharmaceuticals, New York, NY) 500 mg PO or IV TID. There are resistant strains, which may require the use of vancomycin given orally. Proper handwashing is critical. New highly virulent strains of *C. difficile* have resulted in fatal infections, and other cases have been treated with colectomy: this is a problem to be taken seriously.

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Electrolytes

Hypocalcemia may be seen on the head and neck service, following thyroid or parathyroid surgery. Inadequate parathyroid gland function rapidly results in low serum calcium. One may follow total serum calcium, correlated to albumin level, or may follow ionized calcium. As total calcium drops below ~7.5 to 8.0, the patient may become symptomatic with tingling hands or twitching; with severe hypocalcemia, tetany may ensue. Chvostek sign is twitching of the corner of the mouth in response to tapping over the facial nerve trunk, and tends to correlate with a calcium level lower than ~8.0. For mild hypocalcemia, the patient may be treated with oral calcium carbonate, 1 g TID, along with Rocaltrol 0.25–0.5 mcg PO daily. In more symptomatic patients, IV correction is necessary using 10% calcium gluconate given 20 ml IV over 15 to 30 minutes. For severe hypercalcemia, a rare condition, treat with massive IV hydration.

For further information on hypercalcemia and hypocalcemia, see Chapter 5.4.12.

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1.8 General Otolaryngology

1.8.1 Obstructive Sleep Apnea

◆ Key Features

- A cessation of breathing during sleep
- Sleep apnea may be central, obstructive, or mixed
- Obstructive sleep apnea: caused by upper airway collapse and narrowing
- Cardinal symptoms: disruptive snoring, witnessed apneas, daytime sleepiness

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Obstructive sleep apnea (OSA) is characterized by repetitive episodes of complete (apnea) or partial (hypopnea) upper airway obstruction occurring during sleep. Apnea is a cessation of breathing for at least 10 seconds. Hypopnea is a transient reduction of breathing for 10 seconds. Hypopneas must be associated with oxygen desaturation of at least 4%, whereas apneic events are not required to be associated with a decrease in oxygen saturation.

◆ Epidemiology

The prevalence of sleep-disordered breathing is estimated to be 2 to 4% of middle-aged women and 49% of middle-aged men. The prevalence increases with age and may remain undiagnosed in the majority of patients.

◆ Clinical

Signs and Symptoms

The signs and symptoms of sleep apnea include witnessed nocturnal apnea events, snoring, daytime sleepiness, headaches, depression, and decreased libido.

Differential Diagnosis

The diagnosis is laryngospasm related to GERD and dyspnea secondary to pulmonary edema or cardiac disease. Sleep apnea can also be caused by nonobstructive alveolar hypoventilation, asthma, chronic obstructive pulmonary disease, congestive heart failure, narcolepsy, periodic limb movement disorder, sleep deprivation, and medication, drug, and alcohol use.

◆ Evaluation

History

The history taken from the patient and his or her bed partner will include reports of snoring, witnessed apneas or gasping events at night, daytime sleepiness, decreased libido or sexual dysfunction, and motor vehicle or work accidents.

Physical Exam

- Body habitus (weight, body mass index [BMI])
- Congenital craniofacial abnormalities
- Oral exam: large tongue base, elongated soft palate, enlarged uvula, narrow oropharyngeal inlet
- Nasal exam: deviated septum, hypertrophic turbinates, polyposis nasi
- Hypertension

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Imaging

Consider a chest x-ray to rule out right-sided heart failure.

Labs

Laboratory testing is not necessary.

Other Tests

Nocturnal polysomnography (PSG): the gold standard for diagnosing obstructive sleep apnea; multiple physiologic parameters are measured while the patient sleeps in a laboratory. Data are collected in the presence of a qualified technician. Typical parameters in a sleep study include eye movement observations (to detect rapid-eye-movement [REM] sleep), electroencephalography (EEG; to determine arousals from sleep), chest wall monitors, nasal and oral air flow measurements, an ECG, an electromyogram (to look for limb movements that cause arousals), and oximetry.

Apnea–hypopnea index (AHI): the most common summary measure used to describe respiratory disturbances during sleep. AHI is the total number of episodes of apnea and hypopnea during sleep divided by the hours of sleep time. AHI values can be computed for different sleep stages. An AHI >5 is required for the diagnosis of OSA; values >15 and >30 indicate moderate and severe levels of sleep apnea, respectively.

Respiratory disturbance index (RDI): a similar measure to AHI, which refers to the number of times per hour that blood O_2 saturation falls >3%.

Arousal index (AI): with an EEG, an AI can be computed, which is the number of arousals per hour of sleep. The AI may be correlated with AHI or RDI, but ~20% of apneas and desaturation episodes are not accompanied by arousals, or other causes of arousals are present.

Some facilities use portable monitors that measure only heart rate, pulse oximetry, and nasal airflow to diagnose OSA.

◆ Treatment Options

Medical

Mild OSA may be managed conservatively by weight loss or avoidance of alcohol and certain medications. Weight loss is the simplest treatment for obstructive sleep apnea in obese patients. Even a modest 10% weight loss may eliminate apneic episodes by reducing the mass of the posterior airway.

Continuous positive airway pressure (CPAP) treatment is used in most patients who have obstructive sleep apnea. CPAP improves upper airway patency by the application of positive pressure to the collapsible upper airway. Effective pressures typically range from 3 to 15 cm H_2O. The most common problem with CPAP is noncompliance. CPAP failure may be caused by perceived discomfort, claustrophobia, and panic attacks.

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Oral appliances may be useful—they are designed to advance the mandible or, at the very least, prevent mandible retrusion with sleep.

Surgical

There are a variety of procedures that may improve OSA in properly selected patients. The challenge is identifying the likely anatomic site of obstruction to be corrected. Many surgeons advocate a potentially staged approach to treat additional sites if improvement is not achieved with the initial procedure. Sites of obstruction may include the nasal airway (septum, turbinates, nasal valve), the palatine tonsils and soft palate/uvula, the base of tongue/lingual tonsils, and the mandible. Common procedures include:

- Septoplasty with or without inferior turbinate reduction may improve nasal breathing and reduce mouth-breathing. It may enable CPAP to be used at a lower pressure with a nasal-only device.
- Uvulopalatopharyngoplasty (UPPP) involves the removal of part of the soft palate, the uvula and redundant peripharyngeal tissues, and the tonsils.
- Laser-assisted uvulopalatoplasty (LAUP) can be performed in a series of office treatments. Its effectiveness has not yet been clearly defined.
- Tongue base reduction procedures include a midline glossectomy, lingual tonsillectomy, radiofrequency treatment of base of tongue (i.e., coblation)
- Jaw advancement or base of tongue suspension involves mandibulomaxillary advancement or hyoid suspension.
- Tracheotomy is the most effective therapeutic maneuver for obstructive apnea but is a procedure of last resort. It bypasses the site of obstruction during sleep. It is indicated for patients who are most severely affected by OSA or obstructive sleep hypopnea.

◆ Outcome and Follow-Up

Prognosis for improvement is excellent with proper treatment. Polysomnography is repeated ~3 months following surgical treatment, or following weight loss achievement. It is important to note that surgical success is often defined as at least a 50% reduction in AHI. This may not be a cure, and either retitration of CPAP or additional staged treatment may be required.

OSA is frequently undiagnosed and untreated and can have long-term sequelae, including poorly controlled hypertension and heart failure. The adverse effects of hypersomnolence may include loss of employment, motor vehicle accidents, and sexual dysfunction.

◆ ICD-9 Codes

780.57 Sleep apnea, regardless of type
780.53 If hypersomnia is present, assign code
780.51 Sleep apnea with hyposomnia or insomnia is classified to code

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1.8.2 Odontogenic Disorders

◆ Key Features

- Localized or regional jaw pain
- Periapical radiolucency
- Tenderness on percussion
- Possible fistula formation at gingival surface

The periapical granuloma forms as a result of pulpal necrosis that can result from dental caries invading the tooth pulp or from physical or chemical injury to the pulp. Subsequent to pulpal inflammation and necrosis, an apical inflammatory process is established that progresses to a localized zone of bone destruction and associated discomfort manifests radiographically as a radiolucency on routine dental radiographs.

◆ Epidemiology

Periapical granuloma or apical periodontitis is a very common form of dental pathology and is responsible for three-fourths of all dental cysts and dental extractions in the young. Most occur in the anterior segment of the maxilla and represents the precursor lesion for the most common dental cyst, the radicular cyst. The epithelial lining of the latter arises from epithelial remnants within the periapical portion of the periodontal ligament.

◆ Clinical

Signs and Symptoms

Symptoms are usually minimal and are often noted incidentally. Radiographically a localized radiolucent area at the apex of a nonvital tooth contiguous with the periodontal ligament and lamina dura is present.

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Size ranges from a few millimeters to 1.5 cm in most instances. Occasionally, a mild degree of labial or buccal alveolar bony expansion may be noted in addition to root apex resorption.

Differential Diagnosis

The differential diagnosis resides between the periapical cyst and the radicular cyst, whereas rarely there may be mimicry with an odontogenic tumor, other forms of odontogenic cyst, primary bone disease, a giant cell lesion, and metastatic disease. All of the latter considerations present with vital teeth.

◆ Evaluation

Histologic evaluation of the periapical granuloma reveals a mixed chronic to subacute inflammatory cell infiltrate within a loose and highly vascular connective tissue stroma that also is likely to contain foamy macrophages. The radicular or periapical cyst is lined by nonkeratinized stratified squamous epithelium within a variably inflamed supportive stroma with scattered erythrocytes, hemosiderin, plasma cells, and Russell bodies.

The clinical and radiographic examination reveals the presence of a nonvital tooth and a characteristic periapical radiolucency. Alveolar bone expansion may be present, but not invariably so.

◆ Treatment Options

Management ranges from tooth extraction to curettage of the periapical region. Preservation of the tooth requires endodontic treatment. In cases of a chronic or persistent problem not responsive to routine endodontic treatment, an apical curettage (apicoectomy) procedure will be required.

◆ Complications

In cases in which apical curettage was inadequate or not performed, the risk of recurrence is present in the form of a residual cyst or the initial pathology, a periapical granuloma. In such cases, continued destruction of alveolar bone can follow, with larger lesions compromising the strength and integrity of the mandible or maxilla.

◆ Outcome and Follow-Up

Long-term success is measured by bony fill-in within the periapical region and the reestablishment of function secondary to tooth replacement by removable or fixed bridgework or by dental implant placement and restoration.

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1.8.3 Benign Oral Pathology

◆ Key Features

- Focal epithelial hyperplasia presents as discrete, well-defined, minimally elevated painless, pinkish white lesions of the oral mucosa. It is associated with a wide ethnic incidence.
- The lesions exhibit benign behavior, may be very numerous, and are transmissible.
- Squamous papilloma is the most common oral mucosal papillary lesion (2.5% of all oral lesions).
- A viral etiology is present in many but not necessarily all squamous papillomas.

Focal epithelial hyperplasia (Heck disease) usually arises in childhood, with a large percentage of children in some populations affected; in immunosuppressed individuals the incidence is increased. These well-circumscribed lesions are sessile in nature and are predominantly over the buccal and labial mucosa.

Squamous papilloma (oral wart) is a broad term connoting epithelial verrucous and papillary growths with a minimal amount of supportive connective tissue.

◆ Etiology

A viral origin of focal epithelial hyperplasia has been shown, in particular human papillomavirus (HPV) 13 and 32 using in situ hybridization techniques. Earlier suggestions that this was a lesion where genetic factors were at play have been refuted.

Most if not all squamous papillomas are due to infection with HPV-2, 6, 11, and 57.

◆ Clinical

Signs and Symptoms

For focal epithelial hyperplasia, widely distributed lesions over the tongue, buccal and labial mucosa, and alveolar gingiva are characteristic, as is a

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white, well-defined periphery with a finely granular surface quality. Lesions are asymptomatic and are often incidentally discovered. They may be clustered in areas or widespread in distribution.

For squamous papilloma, a predilection for the hard and soft palate and the uvula has been noted, but lesions may be noted anywhere from the vermilion portion of the lips as well as any other intraoral site. Lesion size ranges from a few millimeters to 1 cm in diameter with an exophytic and cauliflower-shaped outline and generally white to pink in color. Lesions are asymptomatic and generally solitary, although in the immunosuppressed patient they may be multiple.

Differential Diagnosis

Multiple squamous papillomas and verruca vulgaris are the most likely lesions to show similarity to focal epithelial hyperplasia, whereas the lesions of Cowden disease, pyostomatitis vegetans, and Crohn disease may also be considered.

Solitary lesions must include the verruciform xanthoma, papillary hyperplasia, and condyloma acuminatum.

◆ Evaluation

The clinical presentation is characteristic; however, the associated histopathology of a representative excised lesion will confirm the diagnosis.

For focal epithelial hyperplasia, surface acanthosis and parakeratosis are present in association with prominent clubbing and fusion of epithelial ridges. Characteristic is the presence of enlarged, ballooning epithelial cells within the more superficial layers, with a koilocytic change within the surface cells. Clear areas within the surface nuclei in association with pyknosis are typical as is the presence of granular cytoplasmic alterations and nuclear fragmentation.

For squamous papillomas, a pronounced growth of normal squamous epithelium covers a well-vascularized connective tissue stromal component that projects from the mucosal surface. Superficial cell layers often contain koilocytic cells where the nuclei are pyknotic or crenated and surrounded by an optically clear zone.

◆ Treatment Options

Generally, no treatment is indicated for focal epithelial hyperplasia, as many cases resolve spontaneously. If few lesions are present or if esthetic considerations apply, conservative excision or cryoablation may be performed. Advanced cases may be managed with a combination of laser and interferon α-2b therapy.

The treatment of choice for a squamous papilloma is surgical excision or laser ablation, with recurrence noted only rarely. In those infected with or demonstrating AIDS, the risk of recurrence is not uncommon.

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◆ Outcome and Follow-Up

No complications are expected, though recurrence may be noted. Malignant transformation should not be a concern.

◆ ICD-9 Code

528 Diseases of the oral soft tissues, excluding lesions specific for gingiva and tongue

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1.8.4 Temporomandibular Joint Disorders

◆ Key Features

- After toothache, temporomandibular joint (TMJ) disorders are the most common cause of facial pain
- Most patients respond to conservative management.
- Surgical treatments can repair or replace severe damage.

Disease of the TMJ is often classified as myogenous or arthrogenous, involving a primary problem of muscle tension versus joint anatomy. Patients present with pain, headache, discomfort with chewing, and popping of the temporomandibular joint.

◆ Anatomy and Physiology

The TMJ connects the jaw and skull at the condyle of the mandible and the squamous portion of the temporal bone. The articular surface of the temporal bone is formed by the articular eminence anteriorly and a concave articular fossa posteriorly. The articular surface of the mandible consists

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of the top of the condyle. The surfaces are separated by an articular disk, the meniscus. The joint meniscus is made of fibrocartilage and is important for smooth joint function. The meniscus has a thick anterior band, a thin intermediate zone, and a thick posterior band; pathologic changes can contribute to TMJ disorders.

◆ Epidemiology

TMJ disease is more common in women, with a male-to-female ratio of 1:4. The highest incidence is among young adults, aged 20 to 40 years. The disorder is seen more frequently in Caucasians than in persons of African descent.

◆ Clinical

Signs and Symptoms

These include TMJ pain, popping, crepitus, tenderness, and spasm of the mastication muscles. Headache, neck ache, and tooth sensitivity are common. A history of poor sleep or other sleep disorders is common. Many patients may carry a diagnosis of migraine, which is a misdiagnosis.

Differential Diagnosis

For patients presenting with complaints in the TMJ region, important considerations, in addition to TMJ disorders, include disease involving the ear, such as otitis externa or otitis media, and sources of referred otalgia such as malignancy of the larynx or pharynx. TMJ disorders may be related to rheumatoid arthritis, degenerative joint disease, ankylosis, dislocations, infections, congenital anomalies, and neoplasm. Dental malocclusion, jaw clenching, bruxism, personality disorders, increased pain sensitivity, and stress and anxiety should be considered.

◆ Evaluation

History

As in other areas, a careful history is important to guide the clinical assessment. Particular attention to prior dental problems, dental procedures, and head and neck injuries is important. Prior diagnosis of psychologic disorders, chronic pain, migraine, or other headache disorders should be noted. A detailed accurate medication list is required, with attention to chronic pain or anxiety medication use.

Physical Exam

A full head and neck examination is performed. One must assess for evidence of active otologic disease, which could be a source of pain. Also, it is important to visualize the pharynx and larynx to exclude an obvious lesion that could be a source of referred pain. Assess for dental malocclusion, abnormal dental wear, absent teeth, visible clenching or spasm of the ipsilateral neck muscles. Evaluate jaw motion, which may be reduced in TMJ disease.

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Normal range of motion for opening is 5 cm. The joint should be palpated, inferior to the zygomatic arch 1 to 2 cm anterior to the tragus, in both open and closed positions. The examiner should feel for muscle spasm, muscle or joint tenderness, and joint capitation.

Imaging

CT and MRI can provide detailed evidence of bony and soft tissue derangements. Imaging is recommended prior to consideration of surgery.

Pathology

Two categories of TMJ disease exist:

1. Myogenous temporomandibular disease: muscular hyperactivity and dysfunction due to dental malocclusion. Psychological factors often are contributory, such as anxiety leading to habitual clenching of the jaw. Factors contributing to muscle spasm include malocclusion, jaw clenching, bruxism, increased pain sensitivity, personality disorders, stress and anxiety, and a history of trauma.
2. Articular temporomandibular disease: joint dysfunction related either to (a) displacement of the meniscus disk, or (b) diseases causing degenerative changes to the joint anatomy. Meniscus displacement is the most common cause. Abnormal anterior displacement of the posterior band between the condyle and the eminence leads to signs and symptoms. Muscular hyperactivity/spasm in this situation is secondary. Conditions causing degenerative changes to the joint anatomy include rheumatoid arthritis, degenerative joint disease, ankylosis, dislocations, infections, trauma, congenital anomalies, and neoplasm.

◆ Treatment Options

Medical

Most patients can be managed with conservative treatment, involving joint rest, antiinflammatories, muscle relaxants, dental occlusal splints/nightguards. Joint rest is achieved with the use of a soft diet and the avoidance of chewing gum. Nightguard splints can be fashioned using dental impressions and will likely reduce nighttime bruxism and masticator muscle clenching.

The main goal of physical therapy is to stabilize the joint and restore mobility, strength, endurance, and function. Modalities include relaxation training, friction massage, and ultrasonic treatment.

Relevant Pharmacology

- Antiinflammatory medication for 4 weeks then taper:
  - Ibuprofen 200–400 mg PO every 4–6 hours; not to exceed 3.2 g/d
  - Naproxen 500 mg PO, followed by 250 mg every 6–8 hours; not to exceed 1.25 g/d

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- Muscle relaxants:
  - Cyclobenzaprine (Flexeril, Ortho-McNeil-Jansson Pharmaceuticals, Titusville, NJ) 20–40 mg/d PO divided into two or four doses; not to exceed 60 mg/d
  - Diazepam (Valium, Roche Pharmaceuticals, Nutley, NJ) Mild spasms: 5–10 mg PO every 4–6 hours as needed
  - Adverse effects include sedation, depression, and addiction.
- Other:
  - Botox has been injected to reduce muscle spasm.

Surgical

Absolute indications: tumor, severe ankylosis
Relative indications: failed conservative management

Arthrocentesis

Arthrocentesis is a minimally invasive procedure, usually performed under either IV sedation or general anesthesia. A 22-gauge needle is inserted in the superior joint space and a small amount of saline is injected to distend the joint space, after which the fluid is withdrawn and evaluated. With reinjection, the joint is then lavaged; steroids and/or local anesthetics can be injected into the joint space.

Arthroscopic Surgery

Indications include internal derangements, adhesions, fibrosis, and degenerative disk changes. Arthroscopic lysis and lavage can be a minimally invasive alternative to open procedures.

Arthroplasty

Arthroplasty refers to open TMJ surgery, including disk repositioning, discectomy, and joint replacement. Disk repositioning is used when the meniscus has become displaced. Under general anesthesia, an incision is made to access the joint and the displaced disk is repositioned with or without plication. Repair of surrounding ligaments may be required.

Discectomy is indicated for TMJ internal derangement involving disk position and integrity; it is typically done as an open procedure under general anesthesia.

In cases of articular eminence anatomic derangement, a procedure to reduce and smooth the articular eminence, articular eminence contouring, may be indicated.

If TMJ damage cannot be repaired, the patient may be a candidate for removal and replacement of part or all of the TMJ components. Conditions requiring surgery may include severe degenerative disease, congenitally deformed TMJs, and advanced rheumatoid arthritis. Options for a partial joint replacement are (a) articular fossa replacement, and (b) mandibular condyle replacement using either autologous bone such as rib, or using a metal prosthesis. In a total joint procedure, the condyle and fossa are both replaced using prosthetic components.

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◆ Complications

A mandible dislocation may occur iatrogenically – during intubation or endoscopy. Typically, the jaw can be relocated as described. Support the head. If the patient is awake, give Valium and consider morphine. Place your thumbs behind the last molar on either side of the mandible and grasp the inferior surface of the mandible with your fingers on each side. Exert downward pressure on the lower molars to free the condyle from its entrapped position anterior to the articular eminence. Ease the mandible posteriorly to return it to its anatomic position. The teeth should close rapidly.

◆ Outcome and Follow-Up

In most cases the prognosis is good, and patients respond to conservative management. Surgical outcomes tend to be favorable in properly selected cases.

◆ ICD-9 Code

524.60 Temporomandibular joint disorders

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1.8.5 Geriatric Otolaryngology

◆ Key Features

- The elderly population is expanding rapidly.
- The elderly often present with multiple comorbidities.
- Disease presentation in the elderly may be atypical.
- For safe performance of surgery in the elderly, thorough preoperative management is important.

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Table 1.23 General Approach to the Elderly Patient

History of present illness: patient may be a poor historian; obtain primary care records if possible
Medications: obtain accurate current medication list; consider polypharmacy issues
Social: DNR status; end-of-life care wishes, advance directives; tobacco and alcohol use; functional status; elder neglect and abuse
Preoperative: careful consideration of risk factors. Testing including CBC, PT, PTT, BMP, chest x-ray, 12-lead ECG. Consider cardiac echo or stress test if risk factors. Anesthesia: consider local, local with IV sedation, versus general
Examination: ancillary testing to consider: audiology, VNG study, videostroboscopy, barium swallow

Abbreviations: DNR, do not resuscitate; CBC, complete blood count; PT, prothrombin time; PTT, partial thromboplastin time; BMP, basic metabolic panel; ECG, electrocardiogram; IV, intravenous; VNG, videonystagmographic.

The proportion of the U.S. population aged 65 and older is predicted to expand from 12.4% in 2000 to 19.6% in 2030, or 71 million persons. Moreover, the over-80 age group is expected to expand from 9.3 million persons to 19.5 million by 2030. Diseases affecting the elderly often require otorhinolaryngologic care. Comorbidities such as cardiovascular disease, renal disease, and polypharmacy make caring for these patients medically, and safely administering surgical care, complex.

The clinical approach to the geriatric otolaryngology patient should follow the same general organization as is used for other patients. However, certain areas of the evaluation require specific attention (**Table 1.23**).

◆ History

Obtaining a good history from a debilitated, chronically ill elderly patient, or a patient suffering from dementia, may be difficult. If possible, input from a close relative or friend may be helpful. In addition, the primary care provider (PCP) and/or nursing home records should be obtained. For instance, the patient may be unable to state how long a lump has been present, but knowledge of the duration or growth rate of a mass will have direct impact upon how the mass will or will not be evaluated. Previous medical or surgical treatments for the current problem might be unknown to the patient, but are of great relevance. The same is true of previous testing or evaluation, such as laboratories or imaging. The general past medical and surgical history should be thorough; again PCP records should be sought. It is not uncommon for the elderly patient to forget to mention important facts, such as the presence of a pacemaker, that are crucial to consider if planning surgery or an MRI scan.

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Medication History

The importance of obtaining an accurate current medication list cannot be overstated. Medication reconciliation, or ensuring that pretreatment, current treatment, and posttreatment medication lists are accurate, has become an issue of great importance to regulation bodies such as the Joint Commission, because of emerging data regarding the consequences and frequency of medication errors. Fortunately, the growing use of electronic medical records is making the availability of legible and detailed medication lists more rapidly available. In the elderly, polypharmacy is a widespread problem. Complaints such as dysequilibrium, xerostomia, dysphagia, dyspepsia, and parosmias can be a consequence of medication side effects. This should be considered during the assessment of the elderly. Also, when surgical therapy is being considered, the accurate medication list is critical. The use of cardiac medications such as β-blockers must be considered perioperatively in accordance with current (rapidly evolving) guidelines. Medications such as coumadin, aspirin, or Plavix (Bristol-Myers Squibb, New York, NY) must be considered regarding potential risks associated with holding medications preoperatively. Herbs, nutritional supplements, and any complementary and alternative medicine treatments should not be overlooked.

◆ Social History

A key factor to consider when treating elderly patients involves a consideration of end-of-life care wishes. Often the otolaryngologist is not directly involved in these time-consuming and emotional conversations, which may involve multiple family members, close friends, and clergy. Ideally, the PCP can and should address these issues, with clearly written documentation. When considering surgical intervention in the elderly, one should be sure that advanced directives have been discussed.

◆ Physical Exam

The head and neck exam in the elderly patient does not, in general, differ. There are some generalizations to consider. The incidence of presbycusis or other forms of hearing loss is such that most geriatric patients should be considered for screening audiologic assessment. The incidence of hyposmia in the geriatric group, especially the over-80 group, is exceedingly high. The use of videostroboscopy in the geriatric patient with voice complaints can be of great value in elucidating stiffness, atrophic or other subtle vocal fold changes, thus facilitating appropriate voice therapy choices. Although the otolaryngologist does not generally focus on the physical exam outside of the head and neck, the ability to briefly examine for obvious signs of congestive heart failure such as peripheral edema or jugular venous distention (JVD) may prompt appropriate preoperative testing.

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Table 1.24 Conditions Used to Identify Patients at Risk for Increased Perioperative Morbidity and Mortality

Cardiovascular: coronary artery disease, congestive heart failure, presence of arrhythmias, peripheral vascular disease, severe hypertension, recent MI
Respiratory: smoking history >20 pack-years, morbid obesity, preexisting pulmonary disease, PO_2 <60 or PCO_2 >50
Renal: renal insufficiency (BUN >50, creatinine >3.0)
Hepatic: cirrhosis, hepatitis
Endocrine: diabetes mellitus, hyperthyroidism or hypothyroidism, chronic steroid therapy
Hematologic: coagulopathy, anemia, thrombocytopenia
General: age >70, bedridden/debilitated, malnutrition, emergency surgery

Abbreviations: MI, myocardial infarction; BUN, blood urea nitrogen.

◆ Review of Systems

This is especially important in stratifying preoperative risk. Angina, dyspnea on exertion, chronic renal disease, liver failure, or severe COPD all must be factored in when considering surgery. General surgeons traditionally have used classifications such as the Goldman criteria to consider major surgical risk factors (**Table 1.24**).

◆ Diseases

Several aspects of otolaryngology–head and neck surgery are directly impacted by aging. Age-related changes may affect hearing, balance, head and neck neoplasms, voice, swallowing, nutrition, olfactory dysfunction, sleep disorders, and cosmetic concerns. A consideration of some common conditions, by area, follows.

Otology

- Ceruminosis
- Serous otitis
- Presbycusis
- Tinnitus
- Balance dysfunction
  - Benign paroxysmal positional vertigo (BPPV)
  - Ménière disease
  - Medication side effects
  - Nonvestibular dizziness

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Rhinology

- Presbyosmia
- Rhinitis
- Epistaxis
- Sinusitis

Laryngology and Oropharyngeal

- Presbylarynx
- Neurolaryngeal disorders
- Xerostomia
- Dysphagia
- Aspiration
- Reflux

Endocrine

- Hyperparathyroidism with osteopenia
- Thyroid disorders or malignancy

Head and Neck

- Upper aerodigestive tract malignancy
- Parotid neoplasm
- Cutaneous malignancies of the head and neck

It has been stated that "there is nothing like an operation or an injury to bring a patient up to chronological age." The elderly patient with potentially limited reserve, impaired organ function, and/or polypharmacy requires particular attention, especially when considering surgical intervention. The reader is directed to resources from the recently formed American Society of Geriatric Otolaryngology (ASGO); for mission, meeting, membership and other information about ASGO, go to www.geriatricotolaryngology.com. Also, consult the online e-book from the American Academy of Otolaryngology–Head and Neck Surgery Foundation, *Geriatric Care Otolaryngology* at www.entnet.org. As the aging population expands, and economic and physician workforce issues evolve, there is likely to be increasing need for practicing otolaryngologists to provide appropriate and efficient geriatric care.

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1.8.6 Lasers in Otolaryngology

◆ Key Features

- The carbon dioxide (CO_2) laser is the most commonly used laser in otolaryngology–head and neck surgery and facial plastic surgery.
- Complications of laser surgery include eye damage, airway fire, and infection via smoke plume.

Surgical management of the head and neck has some distinctive features. Access to specific areas may be restricted and highly vascular. Lasers are very adaptable to certain surgical procedures. The energy in the laser beam exists as radiation, heat, and photoacoustic or mechanical form; heat is what is commonly used for surgery (for vaporization, cutting, and coagulation).

◆ Laser Biophysics

The word "laser" was originally an acronym for light amplification through stimulated emission of radiation. Lasers provide a spatially coherent beam of light at a uniform wavelength (frequency). As such, they provide a source of relatively uniform power with predictable effects. When applied to tissues, lasers have certain effects, which depend on several variables including the specific targeted chromophore, competing chromophores, the pulse duration, pulse frequency, pulse pattern, beam diameter, fluence, and depth of penetration. Furthermore, associated tissue cooling techniques can also alter the effects of the laser.

A chromophore is a substance that absorbs a specific wavelength of light. Some of the chromophores directly targeted by current laser technology include water, melanin, oxyhemoglobin, and deoxyhemoglobin. Lasers can affect a selected chromophore but may also affect a competing chromophore (something that is not targeted, but also absorbs that particular wavelength). Depending on the goals of treatment, this may give added benefit or may be undesirable.

When using lasers it is important to consider how far the laser travels inside the treated tissues. This depth effect is important to consider because a laser could potentially not reach its target, or reach well beyond its target. The actual depth of laser penetration depends on multiple factors including but not limited to the laser wavelength, the beam diameter, and the amount of scatter. Relative scatter can also vary depending on the diameter of the laser beam.

In addition to the depth of penetration, the extent of associated tissue interactions is also related to adjacent thermal effects. Adjacent thermal effects can alter the effects of the laser treatment, beneficially (e.g., such as by stimulating neocollagen production in the heated tissues) or not. These effects can extend laterally within the horizontal plane of the treated tissues as well as deeper to these

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tissues. Adjacent thermal effects can vary depending on pulse duration, number of pulses, rate of energy absorption, sequencing, beam diameter, any associated cooling techniques, and whether the beam is passing through previously lasered tissues with secondarily altered characteristics, such as residual "char." For some lasers, residual char can act as a heat sink, increasing adjacent changes.

Fluence is the measure of the laser's energy per unit of area (usually reported as joules per square centimeter). This is an important measure for use in medicine. Typically, there is a certain amount of fluence that is necessary to achieve a given effect. For some procedures, subthreshold fluence may provide no benefit. However, at other times "low-energy" lasers may be beneficial. Alternatively, excessive fluence can result in undesirable excessive thermal effects.

◆ Laser Applications

Specific laser applications in otolaryngology–head and neck surgery include:

- **General otolaryngology:** treatment of benign oral lesions, sleep medicine
- **Otology:** middle ear surgery, stapedotomy
- **Rhinology:** treatment of nasal polyposis, reduction of turbinate hypertrophy, management of bleeding from Kiesselbach's plexus in chronic recurrent nasal bleeding
- **Laryngology:** treatment of exudative lesions of Reinke's space, intracordal mucosal or epidermal cysts, sulcus vocalis, webs, treating recurrent respiratory papilloma, management of stenosis and some laryngeal cancers
- **Facial plastic surgery:** treatment of vascular anomalies of the skin, facial skin rejuvenation, hair removal

Laser selection for a specific application will vary depending upon the desired effects and the laser characteristics. Optimal device selection for any application can be challenging because lasers are an evolving technology. As with all procedures in medicine, risk/benefit ratios should be considered. Only well-constructed studies can determine if there is an optimal device for a given procedure.

◆ Types of Laser

The types of lasers commonly used in otolaryngology–head and neck surgery and facial plastic surgery are described below.

Carbon Dioxide Laser

The CO_2 laser is the most commonly used laser; it emits at 10.6 μm, in the (invisible) mid-infrared region. A visible red helium-neon (HeNe) laser is accurately superimposed on the path of the CO_2 beam and acts as an aiming beam. The CO_2 laser has excellent tissue cutting properties with very little lateral tissue damage. The CO_2 laser integrates well with operating microscopes; however, it cannot be propagated through a traditional fiberoptic cable. There are some new fibers, such as Omniguide, that can be used to deliver a CO_2 laser in a flexible modality.

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Holmium:Yttrium-Aluminum-Garnet (Ho:YAG) Laser

The Ho:YAG laser is a pulsed infrared laser with output at 2.1 μm (2100 nm). Its lasing medium is a yttrium-aluminum-garnet crystal doped with holmium. The laser is excited by a xenon arc flash lamp. It is used in nasal surgery and tonsillectomy. The Ho:YAG laser may be propagated through a fiberoptic cable.

Neodymium:Yttrium-Aluminum-Garnet (Nd:YAG) Laser

The Nd:YAG laser emits in the near infrared at 1064 nm; it is capable of vaporizing large volumes of tissue and is well suited for hemostasis by coagulation. The Nd:YAG may be propagated through a fiberoptic cable.

Potassium Titanyl Phosphate (KTP) Laser

A potassium titanyl phosphate (KTP) laser is a Nd:YAG laser that is frequency-doubled by passing the beam through a potassium titanyl phosphate (KTP) crystal. Doubling the frequency halves the wavelength (from 1064 nm to 532 nm). The visible green light appears to be continuous, but is in fact rapidly pulsed. A KTP laser integrates well with an operating microscope. A KTP laser may be propagated through a fiberoptic cable.

Erbium:Yttrium-Aluminum-Garnet (Er:YAG) Laser

The Er:YAG laser's lasing medium is a yttrium-aluminum-garnet crystal doped with erbium; it emits at 2940 nm (a wavelength that is strongly absorbed by water). It is used for facial skin rejuvenation.

Erbium,Chromium:Yttrium-Scandium-Gallium-Garnet (Er,Cr:YSGG) Laser

The Er,Cr:YSGG laser emits at 2791 nm.

Diode Laser

The diode laser is a semiconductor electronic device that produces laser light by electrical stimulation of arrays of laser diodes. An emission in the far infrared at 810 μm is most suited for otolaryngologic applications. It can be used both in near contact and contact mode (thermal effect).

Argon Laser

The argon laser produces a blue-green beam of light (454.6–528.7 nm) that is selectively absorbed by red pigment. This characteristic of argon laser light has been successfully applied in the treatment of vascular anomalies of the skin, particularly port-wine stains. Argon lasers spare epithelial tissue and photocoagulate subepithelial vascular tissue with little or no scarring.

Dye Laser

A dye laser is one that uses an organic dye as the lasing medium, usually in liquid form (compared with the gases or solids used as media in the other lasers above), so that it may emit a wide range of appropriate wavelengths.

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It may be used for photodynamic therapy (PDT) of small cancers and premalignant conditions; it activates a hematoporphyrin derivative that is selectively retained by malignant tissues, causing destruction of the malignant cells with no damage to surrounding normal tissues.

◆ Laser Safety Considerations

Special complications of laser surgery include direct or indirect ocular exposure to laser irradiation, fire, explosion, or electric shock, and transmission of infection via smoke plume or tissue spatter. Other complications include scarring or synechia surrounding tissue damage.

Whenever using a laser it is important to follow the proper safety precautions. According to the U.S. Food and Drug Administration (FDA), the most common safety-related incidents associated with lasers are eye injuries. In general, lasers between the wavelengths of 380 and 1400 nm (visible to near infrared) pass through the cornea and cause retinal injuries. Lasers of wavelengths longer than 1400 nm usually cause injury to the cornea. To avoid eye injuries, it is important to wear protective eyewear designed for the wavelength that is being used. Eyewear that protects for one wavelength may not provide protection for another wavelength. All laser eyewear should be clearly labeled as to the laser wavelengths for which they provide protection. It is important that everyone in the laser room, including the patient, the physician, and any observers, wear proper protective eyewear. Both direct and reflected laser beams can be hazardous. Many lasers, such as the CO_2 laser, do not emit in the visible light spectrum yet still may cause ocular injury.

Lasers also present fire and skin hazards, which require specific precautions. Depending upon the laser being used and the area being treated, these may include but are not limited to limiting potentially flammable materials or substances near the laser, wetting surgical drapes, using laser-approved endotracheal tubes, and having an appropriate fire extinguisher available. In terms of potential flammability of surgical scrubs, prep solutions, and topical ointments, it is best to review the products' informational inserts and if necessary, check with the manufacturer as to laser safety. Volatile agents such as alcohol and acetone should not be used to clean the skin for injections or skin prep prior to using a laser.

If a laser produces a significant plume, then it is important to protect against potential biocontaminants. Currently, protection against airborne biocontaminants includes both the use of a smoke evacuator and surgical masks. It is important to keep the smoke evacuator within 2 cm of the source of the laser plume. The masks should have antiviral 0.3-micron filters.

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1.8.7 Robotic-Assisted Head and Neck Surgery

◆ Key Features

- Robotic systems allow excellent visualization, stability, and increased range of motion for head and neck surgery.
- The technology enables minimally invasive techniques in the head and neck region.
- Current clinical applications in otolaryngology include transaxillary thyroidectomies and transoral resection of oral, oropharyngeal, and supraglottic tumors; further applications are expected to follow.

Robotic-assisted surgery is an exciting emerging technology. Its application to otolaryngology has been challenging due to the spatial and technical limitations of the head and neck region, but the value of robots for head and neck surgery has recently been demonstrated and is growing. The word "robot" is from the Czech word "robota" meaning forced labor, and was coined for the 1921 science-fiction play *R.U.R. (Rossum's Universal Robots)*. Real robots eventually followed, and the era of robots in surgery commenced in 1994 when the first AESOP (voice-controlled camera holder) prototype robot was used clinically.

◆ Da Vinci Surgical System

The robotic da Vinci Surgical System (dVSS; Intuitive Surgical, Inc., Sunnyvale, CA) is in use in fields such as general surgery, urology, gynecology, cardiothoracic surgery, and recently otolaryngology–head and neck surgery. The dVSS provides several advantages over conventional laparoscopy, such as three-dimensional (3D) vision, motion scaling, intuitive movements, visual immersion, and tremor filtration. The dVSS is composed of the following:

- **Four robotic hands:** Named EndoWrist instruments, they function like hands. They can grasp objects, twist, and turn. The robotic hands are relatively small and allow the surgeon to make very precise movements.

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- **3D camera:** This is a high-definition camera that gives the surgeon a 3D image of the surgical field. The camera includes significant magnification, enabling the surgeon to zoom in on the operative field as needed.
- **Console:** The surgeon sits at the console, where he or she controls the four robotic hands and sees images from the 3D camera.

The four robotic hands and the 3D camera are inserted through incision(s) or a natural orifice, depending on the specific procedure. The two access points most used to date for the dVSS are transoral access for tongue base, tonsil, supraglottic, parapharyngeal, and skull base tumors as well as obstructive sleep apnea surgery; and transaxillary access for scarless thyroidectomy. A third option, transcervical access for neck tumors, is currently under investigation.

◆ Transoral Robotic-Assisted Surgery

A surgical camera and instrumentation are passed through the patient's open mouth and controlled by the surgeon sitting at the adjacent console. The surgeon directs the graspers and cautery or laser to the tumor location and resects under direct visualization. Transoral robotic-assisted surgery (TORS) may preclude the need for mandibulotomy and tracheotomy and allow thorough resection of tumors under direct visualization.

McLeod reported a case using the dVSS to successfully marsupialize a vallecular cyst in a patient presenting with dysphagia. Weinstein et al documented feasibility studies using the dVSS on animal and human cadaver models prior to successfully applying robotic technology to the excision of many supraglottic, base of tongue, and tonsillar malignancies. Weinstein, O'Malley, and Genden have all published reports of its successful use in transoral robotic excision of oropharyngeal and laryngeal tumors. Current investigative work is being performed using the robot for transoral resection of parapharyngeal space tumors.

◆ Transaxillary Thyroidectomy

Transaxillary endoscopic thyroidectomy was described beginning in 2001. The goals of minimally invasive thyroid surgery are to maintain an acceptable level of safety while improving cosmesis. The patient is placed in the supine position under general anesthesia. The lesion-side arm is raised and fixed for the shortest distance from the axilla to the anterior neck. A skin incision is made in the anterior axillary fold. A subplatysmal plane is developed immediately superficial to the pectoralis major and clavicle. Once the subplatysmal plane is sufficiently large, retractors are placed and the dVSS robotic arms are docked. A second skin incision is made on the medial side of the anterior chest wall for insertion of the fourth robot arm. The operation then proceeds in similar fashion to an open thyroidectomy.

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◆ Other Robotic Applications in Otolaryngology–Head and Neck Surgery

Robotic-assisted surgery has been applied to neurotology, though only experimentally so far. The RX-130 robot (Staubli Unimation, Faverges, France) was successfully programmed to drill cochlear implant wells in human cadaveric temporal bone specimens. The use of robotic-assisted surgery in transnasal and otologic procedures is currently limited by the size of the instruments. However, with the development of smaller instruments and more flexible tools, its use can be further advanced in the fields of transnasal and otologic surgery.

◆ Advantages of Robotic-Assisted Surgery

- Robotic-assisted surgery allows for minimally invasive techniques with small incisions, less blood loss, and reduced recovery time.
- It allows access to areas typically inaccessible with conventional laparoscopic techniques.
- It increases the surgeon's dexterity while minimizing tremor.
- It provides excellent optics and improved visualization (3D view with depth perception).

◆ Disadvantages of Robotic-Assisted Surgery

- It is a new technology whose uses and efficacy are still being established.
- It has a high cost.
- The size of the system is awkward: robotic systems have relatively large footprints and cumbersome robotic arms.
- There is a lack of compatible instruments and equipment.
- The surgeon has no tactile feedback ("feel of tissue").
- No billing ICD-9 codes have been established for robotic-assisted surgery.

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1.8.8 Complementary and Alternative ENT Medicine

◆ Key Features

- Patients often use complementary and alternative medicine (CAM); it is important to inquire about use.
- There is evidence that some CAM treatments may be effective for allergies, sinusitis, and bronchitis in certain cases.
- Nutritional supplements and herbal remedies should be avoided 2 weeks prior to elective surgery.
- The two editors of this book do not endorse the use of CAM but provide this information as a resource because many patients and physicians do use these treatments.

The use of CAM therapeutic modalities by patients is common in the United States and elsewhere. As their use becomes more and more prevalent, more research on them is being performed. Otolaryngologic conditions for which patients may seek out CAM products include seasonal and perennial allergic rhinitis, acute and chronic rhinosinusitis, upper respiratory infections, tinnitus, and vertigo. Patients may be drawn to complements and alternatives to conventional medical therapies for various reasons, including (1) failure of the conventional medical therapy to treat a chronic medical condition; (2) perceived lack of potential side effects from a CAM modality; (3) patient appreciation of the philosophy of the alternative practitioner regarding their treatment paradigm. Possible uses of CAM therapies are discussed here, organized by the otolaryngologic condition for which they have been advocated. As more high-quality research is done, there may be further evidence-based recommendations for novel therapies based on CAM practices.

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◆ CAM for Allergic Rhinitis, Acute Sinusitis, and Asthma

The herb butterbur (*Petasites* spp.) contains the active ingredient petasin. This appears to act upon leukotrienes. The supplement Petadolex, standardized to 7.5 mg petasin, has been shown to be as effective as cetirizine hydrochloride for allergic rhinitis.

The use of probiotics (bacteria that are ingested in the form of a nutritional supplement to restore the normal gastrointestinal tract flora) has shown effectiveness in the prevention of seasonal allergic rhinitis symptoms.

A combination of herbs from traditional Chinese medicine (Reishi, shrubby Sophora, and Chinese licorice) has been advocated for asthma therapy in an effort to avoid systemic steroids. It is reported that both the herbal combination and prednisone improve FEV1 and decrease Th2 inflammatory cytokines; the herbal combination increases cortisol and interferon gamma levels whereas prednisone has the opposite effect.

The African herb *Pelargonium sidoides* has been positively reviewed by the Cochrane Database for use in acute sinusitis and acute bacterial bronchitis. The report comments on "...eight randomized clinical trials with acceptable methodologies. Two trials showed that *P. sidoides* was effective in relieving all symptoms, and in particular cough and sputum production in adults with acute bronchitis. . . . Similarly, *P. sidoides* was effective in resolving symptoms of acute bronchitis in two out of three pediatric studies. In acute sinusitis and the common cold *P. sidoides* was effective in resolving all symptoms including headaches and nasal discharge in adults when taken for an extended time period."

◆ CAM for Upper Respiratory Infections

In several double-blind placebo controlled trials, North American ginseng (*Panax quinquefolius*) has been found to be efficacious for influenza and common cold prevention.

Imu-Max (Ortho Molecular Products, Woodstock, IL), a proprietary blend of propolis, echinacea (*E. angustifolia* and *E. purpurea*), and vitamin C, is reported to be effective for cold prevention in children. Also, the use of milk fortified with probiotics was found to reduce the incidence of upper respiratory infections in children.

However, the Cochrane Database reviewed the use of echinacea and vitamin C for both cold prevention and treatment and found them both to be ineffective.

◆ CAM for Tinnitus and Vertigo

Coenzyme Q_{10} supplementation in individuals with coenzyme Q_{10} deficiency and tinnitus has been reported to result in an improvement in tinnitus. Ginkgo biloba and acupuncture are ineffective in all studies for tinnitus treatment. Biofeedback is helpful in some patients.

A homeopathic remedy, Vertigoheel (Heel, Inc., Albuquerque, NM), is suggested to be as effective as betahistine hydrochloride for moderate vertigo.

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◆ Acupuncture

As with the rest of traditional Chinese medicine, acupuncture has been practiced for thousands of years. Its efficacy has been established for multiple medical conditions. The National Institutes of Health position paper on acupuncture supports its efficacy and use for seasonal allergic rhinitis and asthma.

◆ Herbal and Nutritional Supplements and Surgery

Many herbal and nutritional supplements interact with general anesthetics and with the coagulation system; therefore, all supplements and herbs should be avoided 2 weeks preoperatively.

Homeopathic *Arnica montana* has not been found to be effective for hematoma prevention postoperatively.

◆ Dangerous CAM Practices

Ear candling is ineffective for cerumen removal and can cause severe burns. Ephedra (from the plant *Ephedra sinica*, known in traditional Chinese medicine as "ma huang") may cause arrhythmias and was removed from the market by the FDA in 2004. High doses of licorice may elevate blood pressure and cause potassium depletion (deglycyrrhizinated licorice, or DGL, does not cause these side effects).

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2 Otology

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2.0 Embryology and Anatomy of the Ear

◆ Embryology

Auricle

Week 6: Hillocks of His form from condensations of the first and second branchial arches. The first three hillocks are attributed to the first arch, and the second three hillocks are attributed to the second arch (**Table 2.1**).

Week 20: Adult configuration is achieved.

External Auditory Canal

Week 8: Ectoderm of the first pharyngeal groove (cleft) invaginates. Epithelial cells then grow as a solid core toward the middle ear. The cord dissolves at 20 to 21 weeks, and is complete by week 28. Medial epithelium forms the lateral surface of the tympanic cavity; lateral epithelium forms the skin of the bony external auditory canal (EAC). The EAC might not reach final adult size and shape until the early teenage years.

Middle Ear

Week 3: Endoderm from the first pharyngeal pouch forms the tubotympanic recess. This will become the middle ear and eustachian tube. Pneumatization begins at 10 weeks. The tympanic membrane (TM) forms after the canalization of the EAC. The outer layer is first groove endoderm, the fibrous layer is first arch mesoderm, and the innermost mucosal layer is first pouch endoderm.

Week 4: Mesenchyme from the first and second branchial arches fuses and begins to form the malleus and incus. The first arch is referred to as Meckel's cartilage, and the second arch is referred to as Reichert's cartilage. The cartilaginous ossicles form adult size and shape by week 16. They then

Table 2.1 Embryology of the Auricle

Hillock	Arch	Auricular Structure
1	1	Tragus
2	1	Crus of helix
3	1	Helix
4	2	Crus of antihelix
5	2	Antihelix
6	2	Antitragus and lobule

Note. There remains some controversy regarding the final contributions of hillocks 4, 5, and 6.

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Table 2.2

Cartilage	Arch	Ossicular Structure
Meckel	1	Head and neck of malleus, body and short process of incus
Reichert	2	Manubrium of malleus, long process of incus, stapes suprastructure

Note. The manubrium of malleus never completely ossifies.

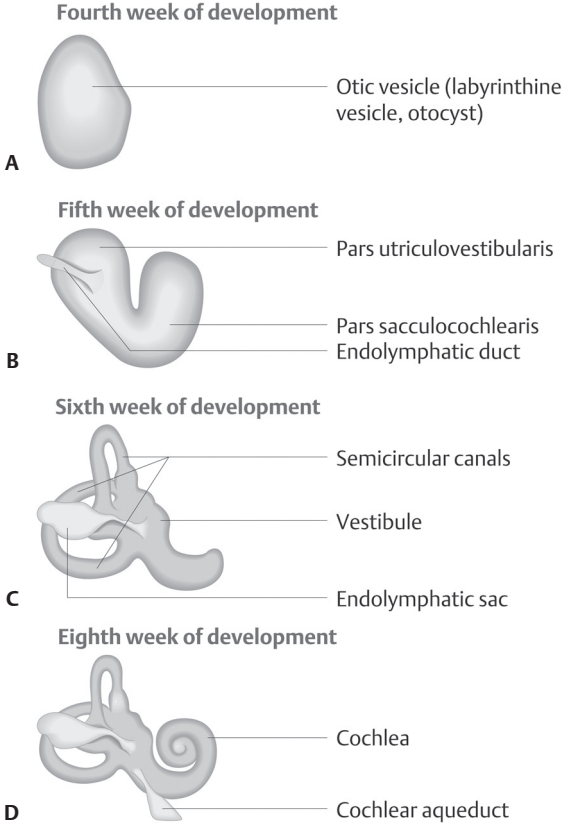

Fig. 2.1 Embryology of the inner ear. The otocyst forms from an epithelial thickening between the cutaneous ectoderm and neural groove in the third and fourth weeks of embryonic development. **(A)** This thickening invaginates and closes off to form a separate vesicle. **(B)** In the fifth week, the otocyst becomes infolded, forming the upper pars utriculovestibularis and the lower pars sacculocochlearis. **(C)** In the sixth week, the three semicircular canals form from the pars utriculovestibularis. **(D)** In the seventh to ninth weeks, the cochlear duct forms as a tubular extension of the pars sacculocochlearis and becomes coiled. (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:158.)

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ossify by endochondral bone formation (**Table 2.2**). Reichert's cartilage also goes on to form the stapes blastema between weeks 4 and 5, which gives rise to the stapes suprastructure. The footplate is of otic capsule origin.

Inner Ear

Week 3: The otic placode forms from ectoderm of the first pharyngeal groove. It invaginates and is completely encircled in mesoderm and termed the otocyst by week 4. The pars superior (semi canals and utricle) develops prior to the pars inferior (saccule and cochlea). The membranous labyrinth is complete by week 15 or 16. Ossification occurs between weeks 20 and 25 (**Fig. 2.1**).

◆ Anatomy

Auricle

The auricle, with the external auditory canal known as the external ear, is made of elastic cartilage with perichondrium and skin tightly adherent anteriorly and loosely adherent posteriorly (**Fig. 2.2**). It is attached to the head by the extension of cartilage into the ear canal and an anterior, superior, and posterior ligament, and an anterior, superior, and posterior muscle.

External Auditory Canal

The EAC is one-third cartilaginous laterally and two-thirds osseus medially. The cartilage is fibrocartilage, not elastic cartilage. The skin over the lateral

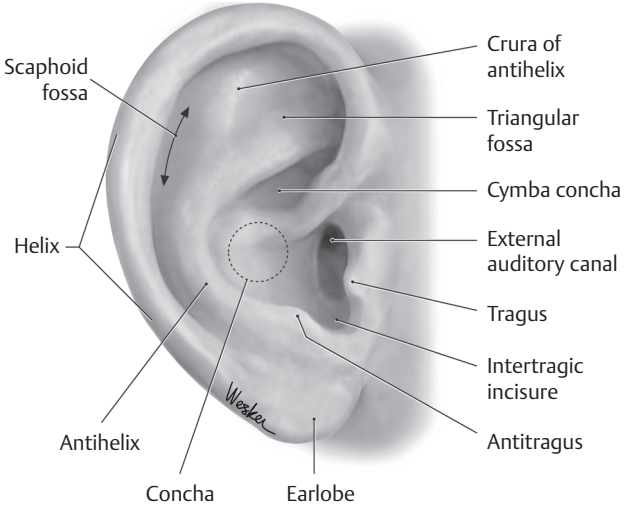

Fig. 2.2 Anatomy of the auricle. (From THIEME Atlas of Anatomy, Head and Neuroanatomy, © Thieme 2007, Illustration by Karl Wesker.)

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one-third is thick with hair follicles, sebaceous and ceruminous glands. The medial skin is thin without any adnexal structures or subcutaneous tissue. It is the only site in the body with skin directly onto periosteum, and is extremely pain sensitive. The canal is innervated by contributions by the trigeminal nerve (cranial nerve [CN] V, auriculotemporal branch), the cervical plexus (C3, great auricular nerve), the vagus nerve (CN X, Arnold's nerve) and the facial nerve (CN VII). C2 and C3 also form the lesser occipital nerve, which helps innervate the anterior and posterior inferior surface of the auricle, but not the canal.

Arnold's reflex is a cough during manipulation of the ear canal via Arnold's nerve, and the Hitselberger sign is numbness over the postportion of the external meatus secondary to a cerebellopontine angle tumor affecting the function of the facial nerve.

Middle Ear

The tympanic membrane is made up of three layers. From lateral to medial: squamous epithelium, a radiating and circular fibrous layer, an inner mucosal layer. It is ~10 mm in height and 9 mm in width. Circumferentially, the fibrous annulus sits within the bony annulus, but is discontinuous superiorly at the notch of Rivinus. Inferiorly to the notch of Rivinus, the TM has a well-organized fibrous middle layer, and is known as the pars tensa. At the level of the notch of Rivinus and above, the middle fibrous layer is less well organized, and is known as the pars flaccida. Prussak's space is at the level of the pars flaccida, bounded laterally by the TM and medially by the neck of the malleus. It is a frequent site of retraction and cholesteatoma formation.

The boundaries of the middle ear space are as follows:

- Lateral: tympanic membrane
- Superior: tegmen
- Inferior: jugular dome
- Anterior: carotid canal, eustachian tube
- Posterior: mastoid via facial recess or retro facial cells
- Medial: cochlear promontory and labyrinthine wall

Ossicles (**Fig. 2.3**)

The malleus consists of a manubrium, anterior and lateral processes, neck and head. It is incorporated into the TM from the lateral process to the tip of the manubrium (umbo). The tensor tendon runs from the cochleariform process of the middle ear to the medial surface of the neck and manubrium of the malleus.

The incus is the largest ossicle, and articulates with the malleus head in the epitympanum. It has a body and short, long, and lenticular processes. The short process is held in place by the posterior incudal ligament.

The stapes is the smallest ossicle, and articulates with the incus at the lenticular process. It has a capitulum, anterior and posterior crus, and a footplate.

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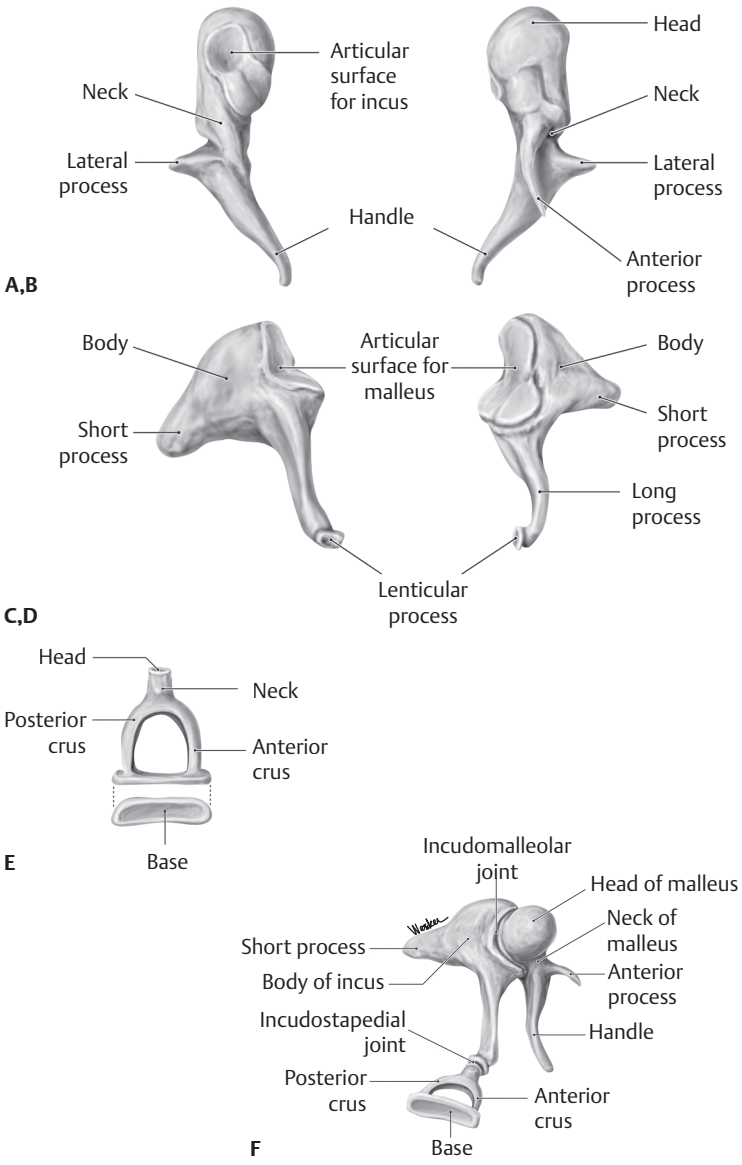

Fig. 2.3 Middle ear ossicles. Malleus, posterior **(A)** and anterior **(B)** views. Incus, medial **(C)** and anterolateral **(D)** views. **(E)** Stapes, superior and medial views. **(F)** Medial view of the ossicular chain. (From THIEME Atlas of Anatomy, Head and Neuroanatomy, © Thieme 2007, Illustration by Karl Wesker.)

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The stapedius tendon runs from the pyramidal eminence to the posterior surface of the capitulum, or the posterior crus. Both incudomalleolar and incudostapedial joints are synovial diarthrodial joints. The footplate is kept in position by the annular ligament.

Eustachian Tube

It measures ~18 mm at birth, and 35 mm in adulthood. It is one-third bony laterally at the anterior mesotympanum, and is lined with ciliated cuboidal epithelium. Two-thirds of the eustachian tube is cartilaginous as it travels medially to end at the torus tubarius of the nasopharynx, and this portion is lined with pseudostratified columnar epithelium. It functions to ventilate, clear secretions from, and protect the middle ear. At rest it is closed, and its primary dilator is the tensor veli palatini muscle (CN V).

Inner Ear

Bony Labyrinth

The vestibule is the central chamber. Its medial wall has two depressions. Posterosuperiorly is the elliptical recess for the macula of the utricle, and anteroinferiorly is the spherical recess for the macula of the saccule. The vestibular aqueduct contains the endolymphatic duct, and enters posteroinferiorly.

The cochlea lies anteriorly. It is a 32-mm canal that rotates 2½ turns around a bony modiolus. The cochlear aqueduct contains the periotic duct.

The semicircular canals lie posteriorly. They lie at a 90-degree angle to one another, and connect to the vestibule with an ampullated and a nonampullated end. They are the horizontal (or lateral), the superior (or anterior), and the posterior canal. All of the ampullated ends enter the vestibule separately, but the posterior and superior canals fuse at their nonampullated ends to join the vestibule together at the crus commune. Superior semicircular canal dehiscence syndrome is a well-recognized entity consisting of symptoms of disequilibrium with motion, exacerbated by loud sounds, and computed tomography (CT) evidence of a bony dehiscence.

Membranous Labyrinth

The membranous labyrinth is the fluid-filled chamber within the bony labyrinth; it is surrounded by perilymph, similar to extracellular fluid (high in sodium, low in potassium), and filled with endolymph, similar to intracellular fluid (high in potassium, low in sodium).

The vestibule is filled with perilymph and holds the saccule and the utricle. Both contain gelatinous membranes coated with otoconia. They sense linear acceleration and gravity.

The cochlear duct mimics the bony cochlea in size and shape (**Fig. 2.4**). It is also known as the scala media, and is filled with endolymph and contains the organ of Corti. Superiorly is the scala vestibuli, and inferiorly is the scala tympani, both filled with perilymph and joined at the helicotrema of the cochlear apex.

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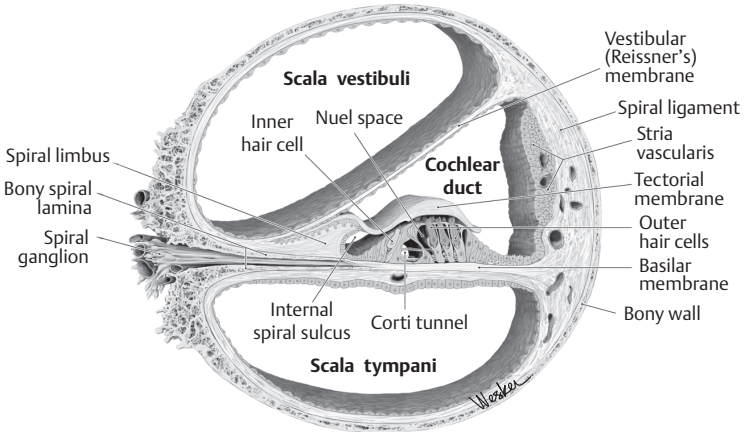

Fig. 2.4 The cochlear duct. (From THIEME Atlas of Anatomy, Head and Neuroanatomy, © Thieme 2007, Illustration by Karl Wesker.)

Semicircular Ducts

The semicircular ducts are within the semicircular canals. Each has an ampullated end with a crista that sends hair cells into a gelatinous cupula to sense angular acceleration. The horizontal canal lies 30 degrees from the true horizontal.

The endolymphatic duct lies medial to the crus commune and opens into the vestibule. It leads to the endolymphatic sac that lies within the dura of the posterior fossa adjacent to the temporal bone.

The cochlear aqueduct runs from the basal turn of the cochlea to the subarachnoid space.

Internal Auditory Canal

The internal auditory canal is the bony channel from the posterior fossa that houses the facial nerve, the cochlear nerve, and the vestibular nerves. It starts medially at the porous acusticus, and runs laterally to end at the fundus. At the fundus, it is split horizontally by the falciform or transverse crest. The superior portion of the canal above the falciform crest is then split again into an anterior and posterior compartment by the vertical crest or Bill's bar.

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2.1 Otologic Emergencies

2.1.1 Sudden Hearing Loss

◆ Key Features

- There is a rapid onset of hearing loss with no obvious explanation.
- Audiogram and tests to rule out identifiable causes are essential.
- Treatment (steroids) must be instituted as early as possible, preferably within 2 to 3 weeks.

Sudden sensorineural hearing loss (SNHL) represents a true otologic emergency. The probable underlying causes are thought to be viral infection or a microvascular occlusive, traumatic, autoimmune, or other event. The current standard of care is to institute high-dose systemic steroid therapy as soon as possible. Response rates to treatment appear to drop considerably if not administered within 2 to 3 weeks of onset.

◆ Epidemiology

Incidence is often estimated to be 1 in 15,000. This is likely an underestimate, as many patients do not seek medical care or are misdiagnosed by primary care providers. Men and women are equally affected; mean onset is middle age.

◆ Clinical

Signs and Symptoms

Patients may present with a complaint of severely decreased hearing of rapid onset in one, or more rarely both, ears. Occasionally, an illness or upper respiratory infection may precede onset. Some patients also experience dysequilibrium, aural fullness, or tinnitus.

Differential Diagnosis

Identifiable causes of hearing loss must be excluded to diagnose idiopathic sudden SNHL. These include vestibular schwannoma, autoimmune causes, and infectious causes such as syphilis. Occasionally, a patient with preexisting moderate SNHL will present with sudden more severe loss due to acute middle ear effusion, for example, following upper respiratory infection. Also, a history for recent exposure to ototoxic drugs (such as aminoglycosides or

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chemotherapeutic medications) must be sought; if present, dose schedules and possible serum levels should be obtained. A history of Ménière disease should be considered.

◆ Evaluation

Physical Exam

The examination should include a full head and neck exam, paying special attention to cranial nerves. A neurologic assessment to rule out signs of central or systemic disorder is important. On otoscopy, it is important to not overlook possible acute middle ear disease as an underlying cause for hearing loss. Also, a fistula test should be performed.

Imaging

Magnetic resonance imaging (MRI) of the brain with and without gadolinium enhancement is obtained to rule out a vestibular schwannoma or other intracranial process.

Labs

A rapid plasma reagin (RPR) test or a fluorescent treponemal antibody absorbed (FTA-ABS) test to rule out syphilis, erythrocyte sedimentation rate (ESR), rheumatoid factor, antinuclear antibody, and Lyme serology if in an endemic area or risk factors are present. The 68-kD antibody is recommended by some authors, although many have not found this to be useful. Others recommend antineutrophil cytoplasmic antibody (c-ANCA; to test for Wegener disease), complete blood count (CBC), chemistries, and thyroid function tests.

Other Tests

Audiologic assessment is mandatory. An accepted definition is at least a 30 dB sensorineural loss across three contiguous frequencies, commonly occurring within a 12-hour period. A poor word recognition score out of proportion to pure tone thresholds should raise suspicion for a retrocochlear lesion.

◆ Treatment Options

Approximately 85% of cases are considered idiopathic. The current standard of care for treatment of idiopathic sudden SNHL is high-dose systemic steroids. Most authors recommend prednisone (1–2 mg/kg orally tapering over 2 weeks). Treatment should begin without delay. At the time of writing, there is an ongoing multicenter clinical trial to evaluate the efficacy of intratympanic steroids. Although data are lacking, many authors recommend concurrent antiviral treatment, such as acyclovir (800 mg orally five times daily for 1 week).

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◆ Outcome and Follow-Up

Follow-up audiograms to assess for recovery are obtained, usually at 2 to 3 weeks following initiation of treatment. The natural history of idiopathic sudden SNHL is that two-thirds of patients should experience some recovery without treatment, often within 2 weeks. Less-severe initial loss, shorter duration of loss, and early treatment are associated with better recovery. The presence of vertigo is associated with poorer prognosis. Auditory rehabilitation in patients who fail to recover is important, especially in children.

◆ ICD-9 Code

388.2 Sudden hearing loss, unspecified

Further Reading

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2.1.2 Ear and Temporal Bone Trauma

◆ Key Features

- Soft tissue or bony trauma
- Trauma may involve hearing, balance, and facial nerve function.

A wide range of acute injuries may involve the ear and temporal bone. These range from injury to the auricle, the external ear canal, the tympanic membrane, the ossicles, the inner ear, and the facial nerve. Fractures involving the petrous bone are associated with severe head injuries.

◆ Epidemiology

Overall, between 14 and 22% of skull fractures involve the temporal bone. These are often associated with other serious injuries. Traumatic tympanic membrane perforations have been estimated to occur at an annual incidence of 1.4 per 100,000 persons. Lacerations, avulsions, hematomas, and thermal injuries to the auricle are common.

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◆ Clinical

Signs and Symptoms

Symptoms and signs depend on the type and extent of injury.

Auricular Injury

Blunt injury may result in local pain and swelling with auricular hematoma. Lacerations and avulsions will present with local findings. Burns to the auricle may appear mild or full thickness; underlying cartilage viability is the critical concern. The auricle is a common site of frostbite injury, which may present as a spectrum of symptoms over several weeks—from clear blistering, hemorrhagic blisters, a dry insensate wound, to blackened tissue demarcation.

Penetrating Trauma or Perforations

Foreign body, instrumentation, blunt injury, acoustic trauma, and barotrauma may all cause traumatic tympanic membrane rupture as well as middle or inner ear damage. Patients may present with pain, hearing loss, dizziness, or facial paresis.

Temporal Bone Fracture

This is associated with severe traumatic head injury and the patient will present as such, requiring resuscitation, stabilization and multiteam care. Victims may present with Battle's sign (mastoid bruise), raccoon eyes (periorbital bruising), otorrhea, hearing loss, nystagmus, and other cranial neuropathies. Fracture is classified by CT scan with respect to the long axis of the petrous pyramid: a transverse fracture crosses this axis (10–20% of fractures); a longitudinal fracture is oriented along this axis; fractures are most commonly mixed (**Fig. 2.5**).

Differential Diagnosis

The differential diagnosis may include soft tissue injury, an auricular hematoma, a cartilage injury, a tympanic membrane perforation, an ossicular disruption, a perilymph fistula, an inner ear injury, a temporal bone fracture, and a facial nerve injury.

◆ Evaluation

Physical Exam

The examination should include a full head and neck exam, paying special attention to cranial nerves. In a trauma patient with severe head injury or multisystem injury, standard trauma protocol and resuscitation are performed. Life-threatening injuries (cervical spine trauma, intracranial injury, etc.) must be stabilized prior to evaluation of a temporal bone injury. With severe injuries requiring intubation and other treatment,

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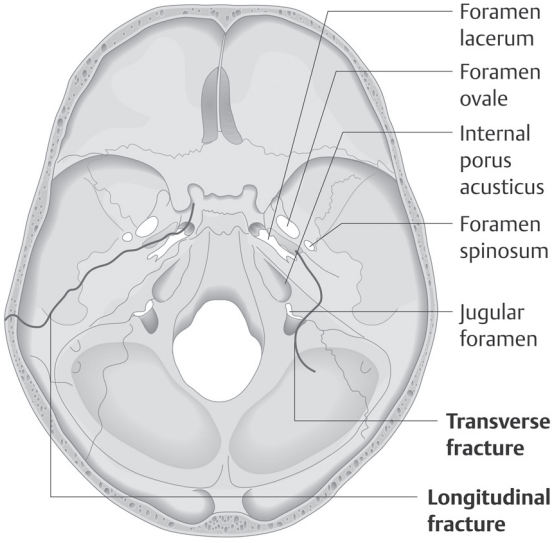

Fig. 2.5 Temporal bone fractures: a typical longitudinal temporal bone fracture (left) and transverse temporal bone fracture (right). (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:303.)

an initial assessment of facial nerve function is important. Cleaning and examination of the ear promotes the assessment of the injuries. Initially, hearing can be assessed with 512-Hz tuning fork. Presence and type of nystagmus should be noted. When stable, temporal bone CT scanning is performed.

In the patient with more limited injury, a more focused exam is feasible. Foreign body or penetrating injuries must be evaluated under the otomicroscope. In uncooperative children, there should be a low threshold for exam under anesthesia. Ear canal injuries can be cleaned and stented if circumferential. Traumatic perforations may require surgical exploration if located in the posterosuperior quadrant and associated with severe vertigo. However, most perforations are anteroinferior and can be observed. Avoid pneumatic otoscopy.

Soft tissue injuries isolated to the auricle are treated with focused exam and emergency care. Exposed cartilage and hematoma must be recognized and treated promptly.

Imaging

High-resolution CT scanning of the temporal bone is the most useful imaging study for temporal bone trauma. Angiography is indicated with gunshot wounds to rule out vascular injury.

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Other Tests

Perform an audiologic assessment as soon as feasible. Electrophysiologic testing of the facial nerve in the setting of traumatic paralysis may be helpful in terms of predicting recovery and guiding treatment decisions.

◆ Treatment Options

Soft Tissue Injuries

Auricular lacerations must be cleaned thoroughly. Cartilage may be reapproximated. When possible, soft tissue should be closed over exposed cartilage. If tissue is devitalized, wet-to-dry dressing coverage can be provided and surgical reconstruction planned in a delayed fashion. Antibiotics are prescribed, and *Pseudomonas* coverage is required.

Auricular hematomas must be drained to prevent a cauliflower deformity. To prevent reaccumulation, a pressure bolster is applied. Dental roles or rolled Xeroform (Kendall Company, Mansfield, MA) are placed on the anterior and posterior surface of the auricle and secured with 2–0 nylon in a through-and-through mattress stitch for 5 days. Antibiotics are prescribed.

Auricular burns are treated with mafenide topically. This may penetrate eschar and help to prevent cartilage loss. Infected cartilage is débrided. With a frostbite injury, avoidance of thawing and refreezing is critical. Injured areas are rapidly rewarmed and patients may benefit from ibuprofen. Ongoing local wound care is required as the injury demarcates.

Attempt at surgical reattachment of avulsions should be performed. There is a high failure rate, requiring delayed débridement and discussion of reconstructive options. Most animal bites are thoroughly irrigated, closed, and treated with oral antibiotics. Management of human bites remains controversial. Débridement, irrigation, intravenous (IV) antibiotics for 48 hours and delayed repair is advocated by some; others treat human auricular bite wounds with irrigation, immediate closure, and oral antibiotics.

Penetrating Trauma/Perforations

Ear canal lacerations should be suctioned and cleaned under the microscope. If circumferential, stenting with Oto-Wick (Medtronic Xomed, Inc., Jacksonville, FL) or Gelfoam (Pfizer Pharmaceuticals, New York, NY) for 10 days may be required to prevent stenosis. Antibiotic and steroid drops are prescribed.

Traumatic perforations in the posterosuperior quadrant with symptomatic vertigo should undergo exploratory tympanotomy due to possible stapes dislocation. At surgery, unstable bone fragments are removed and the oval window is grafted; prosthesis placement is controversial.

Traumatic perforation in other locations can be treated conservatively. Antibiotic steroid drops are prescribed, dry ear precautions observed, nose-blowing avoided, and follow-up exams planned. Vestibular symptoms are treated with antiemetics and rest. Approximately 90% of small perforations heal spontaneously. Large perforations or an accompanying infection may complicate healing and eventually require surgical repair.

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Temporal Bone Fracture

If there is no facial nerve injury, from an otologic standpoint the fracture may be handled conservatively. Local wound care and follow-up audiograms are necessary. However, patients often require neurosurgical intervention for other injuries. If there is complete facial nerve paralysis, this may require surgical exploration, depending on the nature of the injury. Gunshot wounds may involve widespread injury, carrying a high incidence of severe vascular injury and high mortality rate. Cerebrospinal fluid (CSF) otorrhea or rhinorrhea may require surgical repair via a neurosurgical or mastoid approach. Injuries with CSF otorrhea not requiring neurosurgical repair may be observed with conservative measures; a high percentage spontaneously stop. Antibiotic prophylaxis is controversial. Audiometry at 3 months should be performed to follow conductive hearing loss. Persistent conductive loss may require exploratory tympanotomy. Vestibular dysfunction is treated with rest and antiemetics; follow-up vestibular testing is performed.

◆ Outcome and Follow-Up

For temporal bone injuries involving hearing loss, follow-up audiograms are required, as discussed above. Most traumatic perforations heal spontaneously but should be reassessed at 3 months. Benign paroxysmal peripheral vertigo is common following temporal bone injuries, and is managed with canalith repositioning exercises. For patients without hearing recovery, auditory rehabilitation options should be offered, ranging from a conventional hearing aid, a bone-anchored hearing aid, to possible cochlear implantation.

◆ ICD-9 Code

801 Fracture of base of skull

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2.1.3 Acute Facial Paresis and Paralysis

◆ Key Features

- The onset of Bell palsy is rapid, within 24 to 48 hours.
- Treatment consists of high-dose prednisone plus an antiviral.
- Eye care is essential and must not be overlooked.
- Spontaneous recovery to normal or near-normal function is almost 85%.

Bell palsy, or acute idiopathic facial nerve palsy, accounts for 60 to 75% of all acute facial palsies. It is characterized by a rapid onset (24 to 48 hours), and may or may not progress to total paralysis. A facial weakness that progresses slowly over weeks to months is suspicious for a neoplasm. Treatment for Bell palsy consists of a prednisone taper (starting at 1 mg/kg) plus an antiviral for at least one week. Surgical therapy is controversial for patients meeting very specific criteria. Eye care is mandatory if eye closure is compromised (see also Chapter 7.2). If there is little to no recovery after 2 to 3 months, then imaging is recommended to rule out a neoplasm.

◆ Epidemiology

The incidence of Bell palsy is 20 to 30 cases per 100,000 people per year. It accounts for almost 75% of all unilateral facial palsy; 40,000 cases occur in the United States each year. Median age of onset is 40, but the incidence is highest over age 70. Men and women, and right and left sides, are affected equally. Pregnant women and patients with diabetes and hypertension are at increased risk.

◆ Clinical

Signs and Symptoms

Patients usually present with rapid-onset (24–48 hours) facial nerve weakness that may progress to complete paralysis. Patients often report pain and numbness around the ear, hyperacusis, and dysgeusia; 70% of patients will have a preceding viral illness.

Differential Diagnosis

Idiopathic facial nerve palsy is a diagnosis of exclusion (**Fig. 2.6**). Herpes zoster oticus (Ramsay-Hunt) is characterized by severe otalgia and vesicular lesions involving the ear, and accounts for 10 to 15% of acute facial palsies. Melkersson-Rosenthal syndrome consists of recurrent bouts of unilateral facial palsy in association with facial edema and a fissured tongue. Facial

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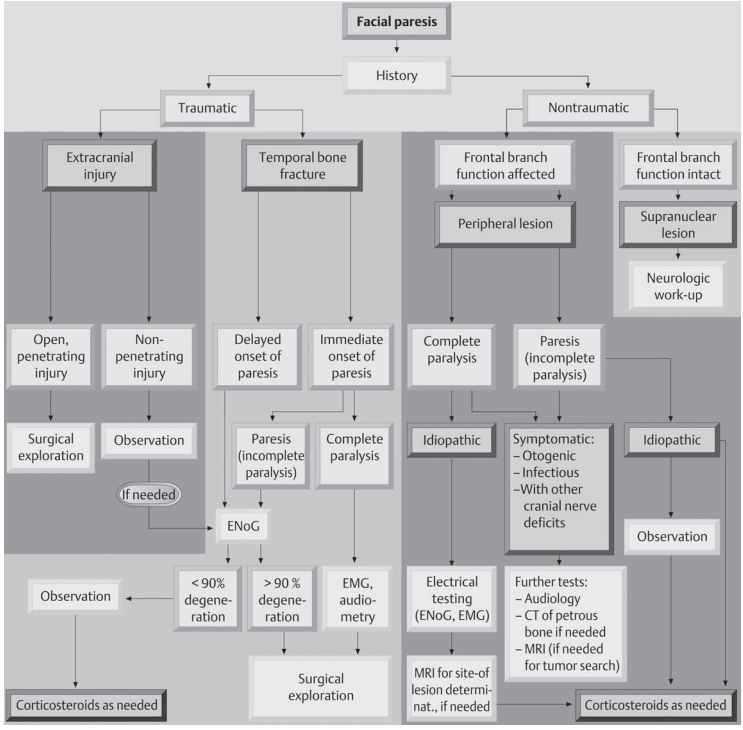

Fig. 2.6 Algorithm for the differential diagnosis of facial paresis. Many advocate surgical decompression only for cases of complete paralysis, and, although not specifically indicated on this algorithm, facial nerve decompression may be useful in cases of nontraumatic paralysis. (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:293.)

palsy occurs in 11% of patients with Lyme disease, and may be bilateral. Acute and chronic otitis media with or without cholesteatoma can also cause acute facial palsy, as can necrotizing or malignant otitis externa. Neoplasms need to be ruled out if there is no symptomatic improvement after 2 to 3 months or if the palsy is characterized by slow onset or relapse.

◆ Evaluation

History

The most important information is the history. Time of onset and history of previous episodes are paramount. Hearing loss and vertigo are also important to note. Patients with herpes zoster oticus are likely to experience involvement of the eighth cranial nerve (CN VIII).

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Table 2.3 The House–Brackmann Facial Nerve Grading Scale

Grade	Facial Nerve Findings
I. Normal	Normal
II. Mild dysfunction	Slight weakness on close inspection
III. Moderate dysfunction	Appears normal at rest; weakness with effort but still full eye closure
IV. Moderately severe dysfunction	Weakness visible at rest; incomplete eye closure
V. Severe dysfunction	Only barely perceptible motion
VI. Total paralysis	Complete paralysis

Physical Exam

A complete head and neck examination is required. Determination is made whether facial nerve function is weak or completely absent. The House–Brackmann scale is often used for charting and physician communication (**Table 2.3**). If the upper face is spared, then a central etiology is suspected. The eardrum is carefully inspected to rule out acute or chronic middle ear disease, cholesteatoma, or a temporal bone neoplasm, either benign or malignant. The skin is evaluated for signs of vesicular lesions. They may be periauricular, auricular, in the ear canal, or even on the palate, and are expected to be tender and in various stages of healing. Palpation of the neck and parotid gland is crucial in ruling out an extratemporal process. The remaining cranial nerves are also examined looking for evidence for polyneuropathy. An audiogram is recommended to evaluate for a skull base process involving the internal auditory canal (IAC).

Imaging

Imaging is not routinely obtained at presentation if the history is consistent with acute idiopathic palsy. If the history is suspicious for any reason, then a magnetic resonance imaging (MRI) scan of the head with gadolinium enhancement is obtained to visualize the entire course of the facial nerve. If there is any enhancement along the intratemporal course of the nerve, a noncontrast fine-cut temporal bone CT scan will help to delineate the fallopian canal looking for dilation or erosion of the canal. If the palsy shows no signs of improvement within 3 months, many clinicians will then order appropriate imaging as described. Some clinicians will not initially image patients with paresis, but then obtain imaging if the paresis progresses to complete paralysis.

Labs

Labs are not routinely ordered. If the clinical picture does not follow that of an idiopathic palsy, or there are risk factors for other disorders then labs are ordered as indicated. Possible systemic problems to look for include autoimmune syndromes, Wegener disease, sarcoid, Lyme disease, or syphilis.

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Electrical Testing

Electrical testing continues to be controversial. If a patient is evaluated within the first 2 weeks of the onset, an electroneuronography (ENoG) is obtained. It can only be done 3 days after total paralysis has occurred, and if the contralateral face is unaffected. A stimulating surface electrode is placed at the stylomastoid foramen, and a recording surface electrode is placed at the nasolabial fold. A compound muscle action potential is then recorded and compared with that recorded from the unaffected side. Changes in amplitude are calculated and interpreted as a percentage of nerve fiber dysfunction. If serial ENoGs are performed starting after day three, and the amplitude on the affected side indicates >90% loss of function, then surgical decompression may be an option. Electromyography (EMG) is also recommended in this circumstance, and surgical candidates are expected to demonstrate no voluntary action potentials within 2 weeks of paralysis.

Pathology

Similar to idiopathic SNHL, idiopathic facial nerve palsy as a symptom is likely secondary to multiple insults. Theories focus on virally mediated inflammation followed by ischemia, neural blockade, and degeneration. The inflammation leads to physical compression as the facial nerve enters the fundus of the temporal bone and forms the labyrinthine segment. This region is known as the meatal foramen and is the narrowest portion of the fallopian canal, forming a bottleneck area. Nerve conduction studies on patients undergoing decompression surgery for Bell palsy have consistently demonstrated decreased electrical responses distal to the meatal foramen. Histopathologic studies obtained from autopsy of patients who died shortly after onset of Bell palsy have been reported. The findings are similar though not uniform. Inflammatory neuritis consistent with a viral infection is most often reported, but intraneural vascular congestion and hemorrhage have also been identified.

Viral studies have shown evidence implicating herpes simplex virus (HSV) as the offending agent. Herpes virus has been harvested from nasal and oral secretions of patients experiencing an acute facial palsy. Animal models have also been used to demonstrate acute facial paralysis after intraoral and periauricular inoculation with HSV.

All of these studies are persuasive, but as of yet, not conclusive.

◆ Treatment Options

Medical

Management of Bell palsy consists of high-dose oral steroids (prednisone 1 mg/kg start dose) with a 2 to 3 week taper, and a concurrent week to 10 days of an oral antiviral (e.g., valacyclovir 500 mg orally three times daily). The natural history of the disorder suggests that almost 85% of patients will recover normal or near normal function without treatment, but there is no test to determine outcomes at the time of presentation. Therefore, all patients are treated with the above medical regimen.

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Patients must also be evaluated for the need for eye care. If paralysis is complete, and facial tone is poor, there will be corneal exposure throughout the day and night. Patients receive hourly doses of artificial tears to that eye, and use a nighttime lubricant to protect the cornea while they sleep. They also wear a clear moisture chamber over the eye, at least at night.

Surgical

Surgery for these patients remains controversial. Centers that see large volumes of these patients have developed algorithms that include serial ENoG testing in conjunction with EMG. If patients present within 2 weeks of onset of paralysis, and reach >90% facial nerve degeneration without evidence of EMG action potentials, they are offered middle fossa facial nerve decompression. The middle fossa route enables complete decompression of the labyrinthine portion of the facial nerve, including the meatal foramen, without sacrificing hearing. Small series have shown that patients choosing decompression have a 91% chance of good recovery (House-Brackmann I or II). Patients who refuse surgery have a 58% chance of poor recovery (House-Brackmann III of IV).

Patients who experience poor recovery often require facial enhancement or reanimation procedures. It is vitally important to continue to attend to the eye in these patients, and recommend surgical care to the eye, including tarsorrhaphy, spring, or gold weight, if corneal abrasions occur despite conservative medical care. Refer to Chapter 7.2 for more details. Botox has been used to help with bothersome blepharospasm due to synkinesis.

◆ Outcome and Follow-Up

Patients are followed longitudinally to assess final recovery. Final recovery from a facial nerve injury may not be seen until 18 to 24 months after insult. According to Peiterson's article charting the natural history of untreated Bell palsy, 71% of patients will recover completely, and an additional 13% will achieve near normal recovery (Hb I or II). Anyone with poor recovery or at risk for corneal abrasion should be considered for surgical reanimation or surgical management of the paralyzed eye. Patients with herpes zoster oticus are at a much higher risk (almost 50%) to experience poor recovery (Hb III or worse). Patients who recover, but then experience a recurrence of symptoms, should be evaluated for a neoplasm, Melkersson-Rosenthal syndrome, or inflammatory/autoimmune conditions.

◆ ICD-9 Code

351.0 Bell palsy

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2.1.4 Ear Foreign Bodies

◆ Key Features

- Ear foreign bodies are commonly found in young children.
- The injury may be acute or chronic, with associated infection.
- Foreign bodies often consist of insects, small batteries, food, beads, pencil erasers, and other inert objects.
- Injury to the tympanic membrane, ossicles, facial nerve, or inner ear must be considered.

Foreign bodies in the external auditory canal are a common problem, especially in pediatric patients. The otolaryngologist often becomes involved after unsuccessful attempts at removal by the patient, parent, emergency room providers, and/or pediatricians. With children, one should have a low threshold for removal under anesthesia in the operating room.

◆ Epidemiology

An ear foreign body is most commonly seen in children under 12 years of age.

◆ Clinical

Signs and Symptoms

An acute foreign body may cause otalgia due to pressure on thin skin of the medial ear canal. Insects are extremely bothersome if alive. Obstruction may cause conductive hearing loss. Chronic foreign bodies, especially organic material such as food, may present with foul otorrhea or purulence. Pronounced hearing loss, vertigo, or facial weakness suggests injury to the middle ear (see Chapter 2.1.2).

Differential Diagnosis

The differential diagnosis includes a foreign body, otitis externa, otitis media, middle ear trauma, or a tumor.

◆ Evaluation

History

In taking the patient's history, you should inquire about the time course and possible nature of foreign body, if unclear. The previous level of hearing is important, as is a history of known tympanic membrane perforation. Injury involving an only-hearing ear mandates careful, experienced management.

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Physical Exam

The examination should include a full head and neck exam. Rule out other foreign bodies. Document facial nerve function and perform a 512-Hz tuning fork exam, if patient is old enough and cooperative. The ear should be examined under the otomicroscope. Any purulence or drainage is suctioned with #3 or #5 Frazier tip suction to enable visualization of the foreign body, canal skin, and tympanic membrane, if possible.

Imaging

High-resolution CT scanning of the temporal bone is the most useful imaging study if there is concern for a middle or inner ear injury.

Other Tests

Perform an audiologic assessment if there is any concern of a middle ear injury.

◆ Treatment Options

Most foreign bodies do not constitute an emergency and can be removed within a reasonable time period (i.e., days). Exceptions requiring prompt treatment include batteries (which cause tissue necrosis due to alkaline or acid leakage), insects, or objects associated with additional trauma to the middle ear or temporal bone. In the adult or cooperative child, one may be able to easily remove a foreign body in the office. If a child is at all uncooperative, it is preferable to proceed with removal under anesthesia in the operating room when convenient, in the next day or so. Live insects are first treated by instillation of 1% lidocaine to the canal, which promptly drowns/paralyzes the insect and anesthetizes the canal skin. The most useful instrument is a blunt otologic right angle hook, which may be positioned medial to the foreign body enabling removal. This is especially helpful for smooth objects, such as beads. An object that can be grasped and is intact may be amenable to the use of alligator forceps. Longstanding decaying organic material associated with infection requires suctioning. Gravel or sand against an intact tympanic membrane is best removed with irrigation. Following removal of a foreign body, careful inspection of the canal skin and tympanic membrane is important. Topical antibiotic and steroid drops for a few days are often helpful in treating or preventing local infection. Oral antibiotics are added for cellulitis, chondritis, and otitis media, as well as for the diabetic or immunosuppressed patient.

The presence of canal skin edema may require the placement of an Oto-Wick. Necrotic tissue secondary to acid or alkaline battery leakage, or thermal burns from slag foreign bodies due to welding are managed with serial débridement and antibiotic steroid drops; wicks or stenting are required for a circumferential canal injury. Management of injuries to the tympanic membrane and the middle ear, as well as temporal bone penetrating trauma are discussed in Chapter 2.1.2.

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◆ Outcome and Follow-Up

Uncomplicated removal of a foreign body usually does not require follow-up. If there is any associated infection, follow-up within a few weeks to document resolution following topical therapy is routine.

◆ ICD-9 Code

931 Ear foreign body

Further Reading

McWhorter AJ, Limb CJ, Niparko JK. In Eisele DW, McQuone SJ, eds. Emergencies of the Head and Neck. St. Louis, MO: Mosby; 2000:367–369

2.2 Otitis Media

2.2.1 Acute Otitis Media

◆ Key Features

- The presence of middle ear fluid along with a bulging or an inflamed tympanic membrane and pain are features of acute otitis media.
- It may also be accompanied by hearing loss, nausea, vomiting, or otorrhea.
- It is one of the most common childhood illnesses, contributing greatly to healthcare costs.

Acute otitis media (AOM) is very common in childhood, affecting at least 60% of children by age one, and 80% of children by age 3. It is clinically defined as a painful inflamed or bulging eardrum with middle ear purulence and fever, often accompanied by one or more additional systemic symptoms. In this setting, it is primarily viewed as a bacterial infection, and needs to be differentiated from viral OM as well as otitis media with effusion (OME) and chronic otitis media. The mainstay of management remains antibiotics, but some children with recurrent AOM will require surgical management with tympanostomy tubes. Occasionally, children will also require surgical management of a complication of AOM.

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◆ Epidemiology

AOM is common in children: 60% of all children under the age of 1 will suffer at least one bout, and that percentage increases to 80% by age 3. It can account for up to 20% of all clinic visits for pediatric patients 10 years old and younger. Specific risk factors other than age include male sex, attendance in daycare, exposure to cigarette smoke, and history of previous infection. There also appears to be a seasonal fluctuation, with more cases occurring in autumn or winter.

◆ Clinical

Signs

Specific signs include an opaque, bulging, and erythematous tympanic membrane (TM) demonstrating poor mobility. If mastoiditis is suspected, the postauricular region may also be erythematous and edematous.

Symptoms

The Agency of Healthcare Research and Quality (Rockville, MD) defines acute otitis media as the presence of a middle ear effusion or the presence of fluid in the ear canal as a result of a ruptured TM with or without TM opacification, erythema, bulging, and hearing loss, and the rapid onset of one or more of the following: otalgia, otorrhea, irritability, and fever with or without anorexia, nausea, or vomiting.

Differential Diagnosis

The differential diagnosis of AOM is really a differential diagnosis for ear pain. Temporomandibular joint disease is probably the most common cause of ear pain that adult patients initially believe may be an ear infection. Another condition that can mimic AOM in adults is herpes zoster oticus. In that case the pain is from reactivation of the herpes virus, and vesicles will be seen in the canal and/or the periauricular region. The otolaryngologist should always consider the possibility of an occult malignant lesion of the upper aerodigestive tract, particularly the larynx, as a source of referred otalgia, especially in an adult patient with a normal ear exam and a history of tobacco abuse. Unilateral otitis media in the adult (more commonly chronic serous effusion) may arise secondary to a nasopharyngeal neoplasm causing obstruction of the eustachian tube orifice.

The most likely cause of ear pain in children is AOM, whether bacterial or viral. Other causes of ear pain are otitis externa, external canal trauma, and an external canal foreign body. Otitis externa can spread from the ear canal out to the auricle as a cellulitis. The auricle may then protrude from the skull with an increased auriculocephalic angle, and closely resemble AOM that has progressed to acute coalescent mastoiditis. AOM must also be distinguished from OME, which can be diagnosed as middle ear fluid with or without hearing loss, but without evidence of acute infection.

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◆ Evaluation

Physical Exam

The most important portion of the physical exam is inspecting the TM. The auricle and the external canal remain normal in appearance and are not tender to palpation. The appearance of the TM will change as the disease process follows its usual course. Initially, the TM is engorged and hyperemic. The hyperemia is most prominent along the manubrium of the malleus and the periphery of the drum. The TM is sluggish to pneumatic otoscopy, but all normal topographic landmarks are visible. As the infection progresses and the middle ear fills with pus, the TM thickens, bulges, and loses normal landmarks. There may be erythema, tenderness, and edema in the postauricular region, especially in small children.

If the infection progresses untreated, the TM perforates in the pars tensa, and the patient experiences a resolution of pain and fever. If purulence is seen in the external canal, then cultures should be obtained. If the perforation heals, and pus reaccumulates untreated, the infection may spread through the antrum into the mastoid, and the mastoid trabeculae may begin to decalcify, leading to coalescent mastoiditis along with other complications. At this stage, the auricle becomes more prominent from the skull as postauricular edema increases. It is important to differentiate this from a severe otitis externa with painful cellulitis and swelling of the auricle.

Imaging

Imaging is usually not indicated unless coalescent mastoiditis or another complication of otitis media is suspected. If such a complication is suspected, a fine-cut temporal bone CT with contrast is indicated. If the patient is an adult with a persistent unilateral otitis media, and the nasopharyngeal exam is equivocal, then an MRI of the head with attention to the nasopharynx may be considered to evaluate for a mass lesion obstructing the eustachian tube.

Labs

Labs are rarely needed to treat routine AOM. One would expect to see leukocytosis on CBC. Routine cultures can be obtained if the ear is draining, and is absolutely indicated in an infant less than 6 weeks of age. Tympanocentesis is rarely indicated to obtain cultures, unless suspicion is high for a resistant pathogen and empiric therapy might not be indicated. Tympanocentesis may be helpful in the immunocompromised patient, especially the patient with chemotherapy neutropenia or acquired immunodeficiency syndrome (AIDS).

Other Tests

An audiogram is not needed in the acute phase, but can be helpful in evaluating children with recurrent AOM, especially in light of other cognitive delays. In cases where the presence of an effusion is in question, tympanograms may be helpful. Occasionally, the combination of a good pneumatic

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exam and an accurate tympanogram are needed to determine the presence of an effusion, even for an experienced otologist.

Pathology

Most commonly, the bacteria that cause AOM will gain entry to the middle ear cleft by way of the eustachian tube. The eustachian tubes of infants are short and horizontally aligned. Infants are also likely to eat in a reclined position. These factors put them at high risk for penetration of nasopharyngeal secretions into the middle ears via the eustachian tubes.

Once they grow into early childhood, they continue to suffer from multiple upper respiratory infections each year, and each bout of nasopharyngeal mucosal congestion can close a eustachian tube, leading to negative pressure behind the eardrum, exudate formation, and infection once the exudate becomes contaminated. An allergy can also lead to AOM secondary to eustachian tube dysfunction.

AOM in adults, especially if unilateral, may be ominous. The nasopharynx must be fully evaluated to identify the cause of the eustachian tube blockage or dysfunction. A nasopharyngeal mass must be ruled out prior to ascribing the cause to either an upper respiratory infection or a recent airline flight. An allergy may also contribute to AOM in adults.

Historically, the most common bacterial pathogens causing AOM have been *Staphylococcus pneumoniae* (40–50%), nontypeable *Haemophilus influenzae* (20–30%), and *Moraxella catarrhalis* (10–15%). Over time, strains of *Pneumococcus* have become penicillin-resistant due to an alteration of the penicillin binding site, and strains of *H. influenzae* have become β-lactamase positive. Also, recently, children vaccinated with a 7-valent pneumococcal vaccine may have a decreased rate of *S. pneumoniae* and a subsequent increase in *H. influenzae*.

Histologically, middle ears display signs of inflammation and edema. The TM and middle ear mucosa are thickened and engorged with an inflammatory infiltrate. There is often frank pus in the middle ear space.

◆ Treatment Options

Medical

There has been a trend toward treating children with uncomplicated AOM symptomatically with pain control and no antibiotics for the first 24 to 48 hours, but this should only be done in children where close follow-up can be performed. Otherwise, uncomplicated AOM is a medical disease, and if persisting more than 24 to 48 hours requires antibiotic therapy. Traditional empiric therapy has been appropriately dosed amoxicillin as a first-line antibiotic because it is effective, well tolerated, and inexpensive. With emerging resistance, the dose of amoxicillin has been doubled to 80 to 90 mg/kg/day. If patients fail to improve at 48 to 72 hours, they can be switched to high-dose amoxicillin-clavulanate. If patients are penicillin allergic, second- or third-generation cephalosporins, macrolides, or clindamycin are all options.

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Surgical

AOM is not a surgical disease. In patients failing to respond to empiric therapy, diagnostic tympanocentesis may be done for culture. Removing the fluid from the middle ear may also relieve pain.

Patients evaluated for recurrent AOM may be candidates for tympanostomy tubes. Standard criteria for tube placement include more than three episodes of AOM in the preceding 6 months or more than four episodes of AOM in the preceding 12 months.

◆ Complications

See Chapter 2.2.3 for complications of AOM. Complications of tube placement include otorrhea, retained tubes, and postextrusion perforations. Otorrhea may occur in upwards of 10%, and is treated with appropriate ototopical drops, currently fluoroquinolones as they are not ototoxic. Tubes are watched yearly, and if they have not extruded after 3 years, many otolaryngologists plan removal under general anesthesia. Postoperative perforations are estimated to occur 3 to 5% of the time, and are followed conservatively. If they persist or cause considerable hearing loss, tympanoplasty may be recommended.

◆ Outcome and Follow-Up

Children with tympanostomy tubes require little postoperative care, and physician preference is usually the driving force determining postoperative recommendations. Many otolaryngologists advocate the use of 5 days of antibiotic topical drops twice daily [i.e., Ciprodex (Alcon Laboratories, Fort Worth, TX) or Floxin Otic (Daiichi Pharmaceutical Corporation, Montvale, NJ)] postoperatively only to children with mucoid or purulent effusions found at the time of tube placement. Many otolaryngologists also recommend ear plugs only for children swimming in fresh water, and not during bath or shower time.

The children are otherwise followed with a postoperative audiogram and interval visits at 6 to 12 months until the tubes extrude.

◆ ICD-9 Code

382.9 Acute otitis media

Further Reading

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2.2.2 Chronic Otitis Media

◆ Key Features

- Chronic or chronically recurring middle ear drainage through a perforated tympanic membrane (TM) is seen.
- Chronic otitis media is associated with a conductive or mixed hearing loss.
- It may occur with or without cholesteatoma.

Chronic otitis media (COM), also referred to as chronic suppurative otitis media with or without cholesteatoma, is characterized by continuous or intermittent otorrhea via a longstanding TM perforation. (This condition is to be distinguished from persistent serous effusion of the middle ear, sometimes referred to as chronic otitis media with effusion but better termed "chronic serous otitis.") If the drainage has stopped, but the perforation persists, the condition is considered inactive. An associated conductive hearing loss may be present, and polyps on the TM or in the middle ear raise the suspicion of concomitant cholesteatoma. Management is initially medical, but can become surgical for recalcitrant cases or those with significant hearing loss or cholesteatoma.

◆ Epidemiology

The epidemiology of COM is not well defined. This is due to its being part of the spectrum of otitis media. It shows no gender preference, but does show a preference for certain populations – Native Americans, American Eskimos, and Australian Aborigines.

◆ Clinical

Signs

Signs include a perforated eardrum with or without polypoid granulation within the middle ear or external auditory canal. Polypoid changes, retraction pockets, or desquamating debris may indicate cholesteatoma. Fevers, neck stiffness, and any change in mental status may be signs of an intracranial complication.

Symptoms

Symptoms usually consist of chronic or intermittent painless otorrhea. Pain may indicate a coincidental otitis externa, or an impending intratemporal or intracranial complication. Hearing loss is often present. An erosive cholesteatoma may breach the otic capsule resulting in SNHL, tinnitus, vertigo,

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and imbalance. Facial paresis or paralysis is rarely seen, but may be present with or without cholesteatoma.

Differential Diagnosis

Other than COM, painless otorrhea with a TM perforation may result from carcinoma of the TM or middle ear and can arise from ears with a history of COM. Any polypoid material removed needs to be sent for pathologic evaluation. Wegener granulomatosis may present in an adult as COM with fluctuating hearing loss and cranial nerve palsy without a previous history of OM. Tuberculous OM should be considered in a draining ear that does not improve despite maximal medical and surgical treatment. The classic description of tuberculous OM is painless otorrhea with multiple TM perforations.

◆ Evaluation

Physical Exam

The physical exam focuses on the draining ear, and specifically on adequate visualization of the eardrum. Often inflammation, especially of the skin of the ear canal, must be controlled first. This may require treatment with topical or oral antibiotics. Once the canal edema has resolved, the TM is meticulously cleaned under an operating microscope. All debris and granulation tissue are cleaned, especially those covering the posterosuperior quadrant of the TM, and any tissue samples are sent to the pathology laboratory. Frequent cleaning and home ototopical medication with an acidic irrigation (acetic acid) may be needed prior to successfully drying the ear.

Imaging

Obtaining CT scans prior to surgical management is controversial, and dependent upon surgeon preference and training. Some surgeons will not scan any patients, some will scan only those with cholesteatoma, and some obtain CT scans for all patients being evaluated with COM. Most surgeons would agree that anyone suffering from a suspected intratemporal or intracranial complication of COM should be imaged with a high-resolution CT scan of the temporal bones with and without contrast. Also, anyone undergoing revision surgery should be imaged. CT scans yield information regarding disease and local anatomy that improves the informed consent process, and makes patients and especially parents more comfortable regarding the decision to undergo surgery.

Labs

Drainage, especially if recalcitrant to empiric therapy, should be cultured. Aerobic and fungal cultures are routinely sent. Additional blood work is rarely indicated. If the onset of symptoms is bilateral, recent, and accompanied by fluctuating hearing loss, or cranial nerve palsy, then c-ANCA is sent to rule out Wegener granulomatosis.

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Other Tests

An audiogram is obtained once the ear is dry and prior to any surgical intervention. A conductive hearing loss is expected, and a mixed loss is not uncommon. Some authors feel that COM can lead to SNHL over time. Any granulation retrieved from the ear is sent to pathology to rule out carcinoma.

Pathology

The most common pathogens leading to COM are *Pseudomonas aeruginosa* and *Staphylococcus aureus*. They gain access to the middle ear through the perforated TM. They then spread from the middle ear to mastoid. These same pathogens can also colonize the avascular debris collecting within a cholesteatoma. A recent problem is the emergence of methicillin-resistant *S. aureus* (MRSA) COM.

The middle ear mucosa becomes thick, fibrotic, and infiltrated with inflammatory cells. Mucosal edema leads to polyp formation and granulation. Bony vascular channels embolize secondary to chronic inflammation, leading to bone erosion, particularly involving the ossicular chain. Cholesteatoma may erode bone by additional local inflammatory response and osteolytic enzymes.

◆ Treatment Options

The goals of treatment are elimination of infection and restoration of function.

Medical

In many cases, medical therapy is employed to dry disease preoperatively, permit better office assessment, or manage the patient with comorbidities in whom surgery is contraindicated. COM may be initially treated with empiric ototopical antibiotic drops. Awareness of ototoxicity has made fluoroquinolone drops the preferred method of treatment. Ototopical drops reach the middle ear in such high concentrations, that resistance is rarely an issue. See **Table 2.4** for topical treatment options. If the drainage does not respond, then cultures are indicated to rule out a rare resistant strain such as MRSA or a fungal infection. Vinegar washes or 2% acetic acid drops may be effective. There are several topical powders that also may periodically be applied if drops do not work. One such mixture includes ciprofloxacin, boric acid, dexamethasone, and fluconazole. Appropriate long-term IV antibiotics may also be indicated if an osteitis is suspected. Another effective topical powder preparation to help dry the chronically draining ear that is unresponsive to drops is chloramphenicol 50 mg, *p*-aminobenzenesulfonamide 50 mg and amphotericin 5 mg, with or without hydrocortisone 1 mg; this is mixed and delivered 1 or 2 puffs via a powder insufflator (e.g., OTOMED, Lake Havasu City, AZ) twice daily. Another option for office management is aqueous gentian violet, which has antifungal properties, and may be "painted" over inflamed areas under the otomicroscope.

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Table 2.4 Topical Therapy for Chronic Otitis Media

Topical Agent	Typical Dose Regimen
2% acetic acid	5 drops 2–3 times daily
2% acetic acid/1% hydrocortisone solution	5 drops 2–3 times daily
Floxin otic	10 drops twice daily
Ciprodex or Cipro HC otic	5 drops twice daily
Cortisporin otic suspension	5 drops 3 times daily
CSF powder (chloramphenicol 50 mg, p-aminobenzenesulfonamide 50 mg and amphotericin 5 mg, with or without hydrocortisone 1 mg)	2 puffs twice daily
Gentian violet (aqueous)	Physician applies topically under microscope in office as needed

Surgical

There are multiple reasons to operate for COM. The infection and drainage can be eradicated, the TM can be repaired, the hearing can be improved, and in the case of cholesteatoma, the disease can be removed and the ear made safe. It is better to dry the ear prior to surgical intervention, but sometimes it is not possible. In cases of drainage at the time of surgery continue with culture-directed antibiotics in the preoperative and postoperative period.

Surgical algorithms vary by preference, training, and experience. The following is a general list of options (nonexhaustive) with definitions.

1. Tympanoplasty without mastoidectomy: eradicating disease limited to the middle ear, whether TM grafting is or is not required. Otorrhea can be controlled, the TM repaired, middle ear cholesteatoma removed, and hearing improved or stabilized. TM grafting material includes loose areolar tissue, temporalis fascia, perichondrium, vein, or cartilage. The graft can be placed either medial or lateral to the TM.
2. Atticotomy: during tympanoplasty, but without performing a mastoidectomy, removing the posterior bony annulus and scutum can improve visualization into the posterior mesotympanum, hypotympanum, and attic. The entire ossicular chain can be visualized and cleaned, as can the tympanic portion of the facial nerve. This can facilitate removal of granulation, retractions, and small cholesteatomas, especially for disease lateral to the ossicles. This is ideal for pars flaccida cholesteatoma that is lateral to the ossicular chain. Cartilage is used to reconstruct the missing bone to prevent repeat retractions, and a mastoidectomy can be avoided in certain cases.
3. Tympanomastoidectomy with an intact canal wall: opening the mastoid in conjunction with debriding and reconstructing the middle ear can improve outcomes in selective patients. Patients with previously failed

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tympanoplasty or longstanding otorrhea can be harboring mastoid granulations and infection. Cholesteatoma extending into the antrum will require this approach.

Microsurgical techniques are required to exenterate all involved air cell tracts. With appropriate thinning of the posterior ear canal and the tegmen, the epitympanum can often be completely exposed. When patient anatomy makes this impossible, partial transcanal atticotomy can be performed concurrently. The facial recess may be opened to improve middle ear access and aeration. Removal of the malleus head is often required for disease extending medial to it or into the supratubal recess. A second-look operation is often planned in cases of cholesteatoma, to assess for recurrent disease and/or perform ossicular reconstruction.

4. Tympanomastoidectomy with canal wall down: this technique is employed in managing COM with extensive cholesteatoma, and rarely in uncomplicated COM without cholesteatoma. This technique is indicated in otherwise unresectable disease, noncompliant patients, only-hearing or already anacusic ears, and patients with small or contracted/sclerotic mastoids prior to surgery. Removing the posterior canal wall increases visualization of the anterior epitympanum, the posterior mesotympanum, and provides access to remove disease more effectively from the sinus tympani. Mastoid cavities may fail, harbor cholesteatoma, or chronically drain if a high facial ridge is left, a dependent mastoid tip results, the TM graft fails, or the meatoplasty is inadequate. Surgeons must carefully attend to these fine details to ensure success.

◆ Complications

Complications of COM are divided into intratemporal and intracranial, and are covered in Chapter 2.2.3. Complications from surgery for COM are covered here. Postoperative complications are rare if surgery is performed meticulously. The best care for otologic postoperative complications is prevention. Postoperative pain is usually mild, and all patients calling with severe pain need to be evaluated. Wound infections are treated with culture-directed antibiotics. *Pseudomonas* is the most likely offending organism, and fluoroquinolones are likely to be the most effective agent used. Postoperative hematomas at the site of graft harvesting are rare, but need to be drained when encountered.

Facial nerve injuries are rare. The use of facial nerve monitoring during even routine ear surgery is becoming widely accepted. Intraoperative injury needs to be addressed immediately, with appropriate nerve exploration, decompression, and grafting if indicated. Getting the advice of a partner or a more experienced otologic surgeon can be helpful in these cases. Facial function that is normal initially, and then worsens over time is usually the result of edema, and is managed expectantly with antibiotics and oral steroids. When a patient awakens from a mastoid procedure with an unexpected facial weakness, it is prudent to observe the patient for a few hours to allow for the reversal of the affects of any local anesthetic used during the procedure prior to reexploration.

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CSF can be encountered intraoperatively, and also needs to be addressed immediately with appropriate use of fascia, cartilage, or bone cement. Rarely, a neurosurgical consultation with lumbar drain placement is required.

Mild vertigo is common following any middle ear procedure, but intraoperative labyrinthine injury results in severe postoperative vertigo (and possible anacusis). Addressing the defect immediately with bone wax or fascia is helpful, but patients will likely still suffer severe vertigo requiring a few days of hospitalization. In this setting, postoperative oral or parenteral vestibular suppressants are administered, and vestibular exercises will hasten recovery.

Granulations and synechiae in the ear canal or canal wall down cavity are removed when encountered, and occasionally meatoplasties require revision.

◆ Outcome and Follow-Up

Perioperative antibiotic use varies widely among surgeons. Many surgeons advocate the use of preoperative and postoperative systemic antibiotics only if the ear cannot be dried prior to the surgery. Surgery is primarily outpatient, with patients returning for an office visit at either 1 or 2 weeks. At that visit most of the reachable packing is removed, and the patient is placed on ototopical drops nightly. Canal wall down cavities are filled with an antibiotic/steroid ointment, and the patient is started on ototopical drops twice a day. Patients are then seen at 4 to 6 week intervals to inspect the graft and clean any granulations. Water is kept out of the ears until complete epithelialization occurs. Routine audiometry is generally performed at 8 to 12 weeks.

The literature cites success rates of surgical management for COM anywhere from 78% to over 90%. Surgical technique and disease severity both factor into outcomes. Cholesteatoma recurrence rates cited in the literature also range anywhere from 3 to 50%. With these percentages in mind, patients are usually followed for several years.

◆ ICD-9 Codes

381.3, 381.4, 382.1, 382.2, 382.3 Chronic otitis media

385.30 Cholesteatoma

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2.2.3 Complications of Acute and Chronic Otitis Media

◆ Key Features

- Antibiotics have produced an overall decline in the frequency of complications of otitis media.
- Complications of acute and chronic otitis media may be both intracranial and extracranial.
- The most common extracranial complication is postauricular abscess; the most common intracranial complication is meningitis.

Acute otitis media is one of the most common diagnoses in patients presenting to physicians. Approximately 85% of all children will have at least one episode. A fraction of patients go on to suffer complications due to progression of infectious and inflammatory processes. More than 60% of patients who suffer complications do so within the first two decades of life. Complications are subdivided by site (extratemporal, intratemporal, and intracranial).

◆ Extratemporal Complications

Subperiosteal Abscess

Mastoiditis either directly erodes the bone of the lateral wall of the mastoid or traverses mastoid emissary veins into the subperiosteal space adjacent to the mastoid.

Signs and Symptoms

Fever, malaise, and pain are associated with a subperiosteal abscess.

Physical Exam

On the physical exam the patient may present with otalgia, otorrhea, an anteriorly and laterally displaced auricle, and fluctuant, erythematous, postauricular fluid collection.

Imaging

CT is the preferred imaging mode.

Treatment Options

The recommended treatment typically includes myringotomy with or without tube, incision and drainage of the abscess, and IV antibiotics. Cortical mastoidectomy is frequently recommended, particularly in the presence of cholesteatoma.

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Bezold Abscess

Mastoiditis leads to erosion of the mastoid tip bone deep to the digastric ridge. Purulent material is directed into the neck, deep to the sternocleidomastoid muscle (SCM).

Signs and Symptoms

Fever, malaise, and neck pain are associated with a Bezold abscess.

Physical Exam

The patient may present with otalgia, otorrhea, and a tender upper cervical mass.

Imaging

Contrast-enhanced CT will confirm the presence of a rim-enhancing cervical abscess in combination with mastoiditis.

Treatment Options

Recommended treatment includes IV antibiotics, myringotomy with or without tube, cortical mastoidectomy, and incision and drainage of the neck abscess.

◆ Intratemporal Complications

Labyrinthine Fistula

A labyrinthine fistula is caused by an erosion of otic capsule bone and exposure of the membranous labyrinth. The incidence is 7% of patients with cholesteatoma. 90% of fistulas occur in the horizontal semicircular canal.

Signs and Symptoms

A significant number of patients will be asymptomatic, and fistulas will only be discovered within the course of mastoidectomy. 62% of patients complain of periodic vertigo or imbalance. Patients may experience the Tullio phenomenon (vertigo induced by loud sounds). SNHL may also be present, but is neither sensitive nor specific.

Physical Exam

Fistula testing (nystagmus with pneumatoscopy) is positive in ~50% of patients.

Imaging

Thin-cut temporal bone CT is preferred; however, small fistulas may still be missed.

Treatment Options

Treatment is controversial, as violation of the labyrinth may result in a dead ear. Many authors recommend canal wall down mastoidectomy, leaving

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cholesteatoma matrix overlying the fistula to form the lining of the exteriorized mastoid cavity. In cases of small fistulas that have not violated the membranous labyrinth, some authors advocate complete matrix removal and semicircular canal resurfacing with bone pate, fascia, or a similar sealant. Others recommend leaving the matrix in place, leaving the canal wall up, and coming back to remove the matrix and resurface the labyrinth at a second stage.

Coalescent Mastoiditis

Suppurative progression of untreated or incompletely treated acute OM results in erosion of the mastoid cavity trabecular bone. Time course of progression is typically 2 to 4 weeks.

Signs and Symptoms

Symptoms include fever, malaise, and otalgia.

Physical Exam

On the physical examination, purulent otorrhea or a bulging TM, postauricular erythema, tenderness, and edema displacing the auricle anteriorly and inferiorly are found.

Imaging

A CT scan confirms mastoid and middle ear opacification with erosion of trabecular mastoid cavity bone. Frequently, erosion occurs primarily adjacent to the sigmoid sinus.

Treatment Options

Recommended treatment includes myringotomy with or without tube, IV antibiotics, and cortical mastoidectomy. Others advocate myringotomy alone and IV antibiotics for 3 to 6 weeks with serial CT confirmation of resolution of mastoid and middle ear opacification.

Petrous Apicitis

Petrous apicitis is a rare complication resulting from the spread of infection into air cells within a pneumatized petrous apex (the prevalence of pneumatization is 30%).

Signs and Symptoms

The classic triad of deep retroorbital pain, purulent otorrhea, and ipsilateral abducens palsy (Gradenigo syndrome) is seen. CN VII and VIII dysfunction also may occur.

Imaging

A CT scan will show opacification of a pneumatized petrous apex.

Other Tests

MRI and lumbar puncture (LP) are helpful to assess concurrent intracranial processes.

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Treatment Options

First-line therapy is long-term IV antibiotics secondary to the difficulty of surgical access to the petrous apex. Abscess, necrotic bone, or failed medical therapy requires surgical drainage. Hearing status determines the choice of approach. Hearing preservation may be attempted by infracochlear, infralabyrinthine, retrolabyrinthine, subarcuate, and middle fossa approaches. Translabyrinthine or transcochlear approaches may be used for nonhearing ears.

Facial Paralysis

Facial paralysis results from inflammation of dehiscent segments of the facial nerve secondary to infection.

Imaging

CT scanning for AOM-associated incomplete paralysis may not be necessary, but may help in operative planning in the setting of cholesteatoma or complete paralysis.

Pathology

In the setting of children with AOM, a congenitally dehiscent tympanic segment of the facial nerve is suspected. The paralysis is usually incomplete and resolves within 3 weeks. In the face of COM and cholesteatoma, dehiscence is typically due to fallopian canal erosion secondary to disease. Onset may be slow and progressive. The prognosis is poorer and recovery slower.

Treatment Options

Paralysis due to AOM is treated with IV antibiotics and myringotomy. Cholesteatoma-associated paralysis requires mastoidectomy, nerve decompression proximal and distal to the diseased segment, and débridement of inflammatory tissue.

Acute Suppurative Labyrinthitis

Acute suppurative labyrinthitis results from direct bacterial invasion of the labyrinth, resulting in total auditory and vestibular loss. Acute suppurative labyrinthitis may lead to meningitis and vice versa.

Signs and Symptoms

Acute-onset total sensorineural deafness and severe vertigo are signs of acute suppurative labyrinthitis.

Pathology

Predisposing factors include congenital inner ear malformations and otic capsule erosion secondary to cholesteatoma.

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Treatment Options

Labyrinthine functional loss is unavoidable, but patients should be treated with antibiotics to prevent the progression to meningitis. Vertigo is treated symptomatically and typically improves as patients compensate.

◆ Intracranial Complications

Meningitis

Meningitis is the most common intracranial complication of OM.

Signs and Symptoms

Fever, headache, nausea, vomiting, photophobia, and nuchal rigidity are symptoms of meningitis. Seizures, ataxia, and other focal neurologic signs are ominous findings.

Imaging

CT or MRI is essential to rule out other intracranial processes and establish the safety of LP.

Other Testing

LP is diagnostic (suggestive CSF findings: increased pressure, increased protein, decreased glucose, inflammatory cells or bacteria present).

Pathology

The potential routes of bacterial spread from the ear to the CSF include hematogenous seeding, bony erosion with direct spread, and bony channels (suppurative labyrinthitis, Hyrtl's fissures, congenital defects, and traumatic defects).

Treatment Options

Because meningitis is the most common complication seen with OM, detailed antibiotic guidelines are listed in **Table 2.5**. First-line therapy is IV antibiotics, myringotomy, and steroids (decreases hearing and neurologic sequelae). In the presence of cholesteatoma, coalescent mastoiditis, or failure of medical therapy, mastoidectomy is indicated.

Brain Abscess

The most frequently lethal intracranial complication of OM is a brain abscess.

Signs and Symptoms

The clinical course is multistage, starting with fever, malaise, nausea, vomiting, headache, mental status changes, and seizures. This may be followed by a quiescent phase with moderate clinical improvement. The third stage, thought to represent abscess growth and ultimate rupture, is a rapid and fulminant return of symptoms with sudden clinical decline.

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Table 2.5 Intravenous Antibiotic Treatment for Meningitis*†

Neonate (<1 month) • Ampicillin + gentamicin; or ampicillin + third generation cephalosporin
Newborn (1–3 months) • First choice: Ampicillin + (cefotaxime or ceftriaxone) + dexamethasone (0.15 mg/kg every 6 h × 4 d) • Alternative: Chloramphenicol + gentamicin
Infant or Child (>3 months) • First choice: (Cefotaxime or ceftriaxone) + dexamethasone • Alternative: Ampicillin
Older Child or Adult • (Cefotaxime 2 g IV every 6 h or ceftriaxone 2 g IV every 12 h) + ampicillin 2 g IV every 4 h + dexamethasone 0.4 mg/kg every 12 h × 2 d + vancomycin (child 15 to 22.5 mg/kg IV every 6 h; adult 1 g IV every 12 h) • If *Pseudomonas* suspected: Cefepime 2 g IV every 8 h instead of cefotaxime or ceftriaxone • For a penicillin-allergic patient in whom cephalosporin cannot be used: Vancomycin 15–22.5 mg/kg IV every 12 h + trimethoprim/sulfamethoxazole 15 to 20 mg/kg/day IV divided every 6 h *or* • Vancomycin 15 to 22.5 mg/kg IV every 12 h + chloramphenicol 1 g IV every 6 h ± rifampin 600 mg once daily

*Other regimens may be indicated for certain situations, such as an alcoholic patient, a patient with a cerebrospinal fluid leak or a debilitating condition such as acquired immunodeficiency syndrome, or a postneurosurgical patient.

†Length of treatment is generally at least 14 days.

Data from Johns Hopkins Online Antibiotic Guide, http://hopkins-abxguide.org; and Greenberg MS. Handbook of Neurosurgery. 7th ed. Stuttgart/New York: Thieme, 2010.

Imaging

CT and/or MRI may aid surgical planning.

Pathology

Infection spreads secondary to thrombophlebitis of venous channels leading from the mastoid to brain parenchyma. The temporal lobe and cerebellum are the most common sites of abscess.

Treatment Options

Treatment is with IV antibiotics and urgent surgical drainage when the patient is medically stable. IV steroids may reduce brain edema. Anticonvulsants are given if needed. Surgery consists of craniotomy with drainage of the abscess. Mastoidectomy is typically recommended at the same time but may be delayed if the patient is medically unstable.

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Lateral Sinus Thrombosis

Bone overlying the sigmoid is eroded by infection, and perisinus inflammation leads to vessel wall necrosis and mural thrombus formation. Thrombus may propagate proximally to the confluence of sinuses and superior sagittal sinus, resulting in life-threatening hydrocephalus. Clot may also propagate distally into the internal jugular vein, leading to possible pulmonary embolus. Additionally, the infected thrombus may shower leading to septicemia or deeper intracranial infections.

Signs

Tobey-Ayer or Queckenstedt's test (lack of CSF pressure elevation with occlusion of the internal jugular vein ipsilateral to the thrombosis) is suggestive of the diagnosis.

Symptoms

Headache, malaise, and "picket-fence" pattern of diurnal spiking fevers. Neck pain may imply distal clot propagation, while obtundation and papilledema may imply resulting hydrocephalus.

Imaging

Contrasted CT may show "delta sign" resulting from sinus wall enhancement with a central filling defect. MRI with magnetic resonance venography (MRV) is very sensitive.

Treatment Options

IV antibiotics and mastoidectomy are the treatment options. Treatment of the dural sinus is controversial. Most authors advocate full exposure of the sigmoid with removal of granulation tissue. Some advocate opening the sinus and removing infected clot. IJ ligation may be performed in the face of significant distal propagation and resulting cervical infection. Anticoagulation is typically reserved for involvement of the cavernous sinus or distal clot propagation.

Epidural Abscess

Granulation tissue and abscess form between the bone of the skull base and overlying dura.

Signs and Symptoms

There are no specific symptoms. Most patients experience deep mastoid pain. Epidural abscess may be encountered incidentally at the time of mastoidectomy.

Imaging

MRI or CT is typically diagnostic.

Treatment Options

IV antibiotics and mastoidectomy with removal of granulation tissue and wide débridement of overlying bone until normal dura is found.

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Subdural Empyema

A subdural empyema is a rapidly progressive, fulminant bacterial infection between the dura and arachnoid layers. Infection spreads rapidly, resulting in significant brain edema, herniation, and death.

Epidemiology

A subdural empyema most often results from sources other than OM/mastoiditis.

Signs and Symptoms

Early symptoms are similar to meningitis, with progression to decreased mental status, seizures, and focal neurologic signs.

Imaging

CT or MRI is diagnostic.

Other Tests

LP is contraindicated, as edema puts patients at high risk of herniation.

Treatment Options

Treatment is with an IV antibiotic and urgent neurosurgical drainage. If the patient is stable, mastoidectomy may be performed following craniotomy.

Otitic Hydrocephalus

Otitic hydrocephalus is a condition of increased intracranial pressure associated with temporal bone infection.

Signs and Symptoms

The symptoms include headache, nausea, vomiting, visual changes, diplopia, and lethargy.

Physical Exam

Papilledema or abducens palsy may be observed.

Imaging

MRI or MRV will detect dural thrombosis, rule out other causes, and confirm the safety of LP. Unlike typical hydrocephalus, the ventricles are not dilated.

Pathology

The condition is thought secondary to lateral sinus thrombosis with proximal clot propagation, resulting in blockage of CSF flow through the arachnoid villi of the superior sagittal sinus.

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Treatment Options

Treatment of coincident lateral sinus thrombosis is as above. The otitic hydrocephalus is treated medically with acetazolamide, fluid restriction, steroids. If medical management fails, lumbar drainage or long-term ventriculoperitoneal shunting may be considered.

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2.2.4 Cholesteatoma

◆ Key Features

- A cholesteatoma is a slow-growing destructive lesion of the middle ear and mastoid.
- It consists of trapped and frequently infected desquamated keratin.
- A cholesteatoma requires definitive surgical management to avoid complications.

A cholesteatoma is a slowly enlarging and destructive cystic lesion composed of desquamating keratin. It is accurately referred to as a keratoma. There are two types: acquired and congenital. The acquired primarily arise superiorly or posteriorly in the middle ear and extends over time into the mastoid, but it also can be found anywhere intratemporally (i.e., petrous apex). A congenital cholesteatoma primarily arises in the anterior superior portion of the middle ear. Cholesteatomas slowly enlarge and lead to complications if not properly managed. There is no effective medical therapy, and surgery is indicated for all patients healthy enough to undergo the procedure.

In an active cholesteatoma, significant squamous proliferation is occurring, likely in the face of local infection and inflammation. This inflammation causes fibroblasts and leukocytes to release enzymes that erode bone directly. Inflammatory cytokines also act as osteoclastic-activating factors, and the osteoclasts then erode bone. Persistent pressure by an expanding cholesteatoma may also erode bone.

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◆ Epidemiology

The epidemiology of a cholesteatoma is unclear. It is likely a result of poor eustachian tube function, and has been reported in 1 to 3% of children with chronically draining ears. Data regarding the prevalence or incidence of acquired cholesteatoma in the general population is unclear. The incidence of congenital cholesteatoma is 0.12 per 100,000.

◆ Clinical

Signs

Signs of a congenital cholesteatoma include conductive hearing loss and a white pearl in the anterior superior quadrant of the middle ear behind an intact TM. Signs of an acquired cholesteatoma include conductive hearing loss, a posterior superior retraction pocket with a keratinaceous crust or desquamating debris, or an aural polyp. There may also be visible erosion of the posterior wall of the external auditory canal.

Symptoms

The symptoms of cholesteatoma are similar to that of COM. The most common presenting symptoms are painless chronic otorrhea and hearing loss. The otorrhea may be bloody. Patients may also present with complications of cholesteatoma, including dizziness secondary to a labyrinthine fistula, facial nerve paralysis, or fever, malaise, and altered mental status suggesting an intracranial process.

Differential Diagnosis

Cholesteatoma frequently presents along with COM, but not all patients with COM harbor cholesteatoma. The symptoms and the complications of both diseases are essentially the same, and the final differentiation between the two sometimes can be done only in the OR.

◆ Evaluation

Physical Exam

A general head and neck exam is performed, noting cranial nerve function. Attention is focused on meticulous evaluation of the TM under the operating microscope. Frequently a large attic or posterior TM retraction with keratin debris is encountered. The retraction may be covered by a crust, which must be removed to accomplish the exam. Occasionally, a polyp may be encountered covering the retraction and debris. The polyp should be manipulated with a suction to see around it or debulk it. Polyps should not be aggressively removed with cup forceps, as ossicular injury can result. A congenital cholesteatoma will slowly grow undetected, but may be seen as a white pearl anterosuperiorly behind an intact TM. Pneumatic otoscopy may be helpful in detecting a lateral canal fistula.

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Imaging

CT is the best way to image cholesteatoma. Some surgeons only image patients with either SNHL or dizziness, indicating a complication of cholesteatoma has arisen. However, it is useful to routinely image all cholesteatomas to inspect extent of disease and local anatomy. A thin section 1-mm CT with true axial and true coronal cuts is best. The cholesteatoma appears as a homogeneous soft tissue density that is frequently difficult to differentiate from soft tissue edema or fluid. Therefore, it is imperative to use the physical exam and the CT in conjunction, and not just rely on a CT to diagnose cholesteatoma.

Things to note on coronal images:

1. Scutal erosion
2. Ossicular erosion
3. Tegmen breach
4. Lateral semicanal fistula
5. Status of the facial nerve

Things to note on axial images:

1. Ossicular erosion
2. Status of antrum and other air cells
3. Lateral semicanal fistula
4. Status of the facial nerve

Other Tests

An audiogram is essential. The majority of patients will demonstrate some degree of conductive hearing loss indicating TM or ossicular involvement. They need to be counseled that the conductive hearing loss may remain despite surgical removal of disease, especially if a staged approach is followed (with an ossicular reconstruction at a later date).

SNHL may indicate cochlear invasion, and patients need to be counseled that hearing may be the same or worse after surgery.

Obvious polyp or debris can be removed in the office and sent to pathology to rule out a carcinoma.

Pathology

The pathophysiology of both congenital and acquired cholesteatoma remains controversial. The following are the current theories.

Pathology of Congenital Cholesteatoma

Congenital cholesteatoma is initially defined as a pearly mass behind an intact TM in the absence of OM. The absence of OM has been deleted

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from the definition owing to the high prevalence of OM in children. Two competing theories of formation:

1. Epithelial rest: there is a localized epithelial rest that has been identified in fetal temporal bones at the lateral wall of the eustachian tube in the anterosuperior quadrant of the middle ear that disappears at 33 weeks gestation. Persistence of this rest into childhood can account for congenital cholesteatoma.
2. Acquired inclusion: the speculation is that a microinjury and retraction occurs to the pediatric TM allowing small foci of epithelial tissue to invade the middle ear and proliferate just anterior to the malleus handle.

Pathology of Acquired Cholesteatoma

Four theories:

1. TM retraction (primary acquired): there is retraction of the TM secondary to negative pressure from eustachian tube dysfunction. This most commonly involves the pars flaccida retracting up into the attic, collecting keratinaceous debris, and forming a cholesteatoma.
2. Epithelial migration (secondary acquired): migration of canal and TM epithelium through a TM perforation. This occurs most commonly with a marginal perforation. The epithelium then proliferates within the middle ear.
3. Implantation (secondary acquired): similar to migration except that epithelium is actively implanted into the middle ear secondary to either trauma or surgery.
4. Metaplasia (historical theory): chronic OM is believed to trigger metaplastic change of middle ear mucosa from cuboidal to keratinizing squamous cells.

◆ Treatment Options

Medical

There is no medical therapy for cholesteatoma; however, presurgically, patients should have the ear carefully cleaned and treated with an appropriate antibiotic/steroid ototopical preparation. This will dry the ear, and decrease bacteria and granulation prior to surgery.

Surgical

The goals of surgery are to remove the disease, to make a safe ear, and to restore hearing. Surgeons will differ on their methods of achieving the above goals. Some surgeons prefer a staged method with ossicular chain reconstruction at a later stage; others prefer to do everything at a single

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stage, and only reexplore ears with signs of persistent or recurrent disease. Either way, there are three primary surgical options:

1. Tympanoplasty alone or with atticotomy: indicated for small cholesteatomas primarily of the middle ear with no evidence of mastoid involvement. This is applicable to most congenital cholesteatomas. It is also applicable to small retraction cholesteatomas that have not extended posteriorly deep into the antrum.
2. Tympanomastoidectomy with canal wall up (CWU): indicated for cholesteatomas that extend through the antrum into the mastoid air cells. This allows for total removal of disease with complete inspection of all vital areas while preserving the configuration of the ear canal. It may be performed with a facial recess approach to improve middle ear exposure and aeration. Diseased ossicles are removed during the surgery and reconstructed at the same time, or at a staged procedure. The patient requires follow-up to monitor for recurrence of disease, but does not require long-term mastoid cavity maintenance.
3. Tympanomastoidectomy with canal wall down (CWD): indicated for large cholesteatomas, cholesteatomas with significant preoperative complications (i.e., large semicanal or tegmen fistulas), patients with small mastoids, disease eroding a significant portion of the external auditory canal, cholesteatoma matrix adherent to a dehiscent facial nerve, or patients with disease in an only-hearing ear. Surgeon preference, degree of disease, and patient factors all play a role in when to perform CWD surgery. Once the facial nerve is identified, the posterior wall of the ear canal is removed down to the level of the facial ridge. All air cells are removed, all edges are saucerized, the mastoid tip, if dependent, can be amputated, the eardrum is grafted, ossicular reconstruction can be performed, and a meatoplasty is required. Some of the mastoid cavity dead space can be obliterated with soft tissue from the postauricular region. Cholesteatoma recurrence rates are lower for CWD procedures, but patients require lifelong mastoid cavity care. There is controversy regarding the improved access to middle ear spaces that the CWD technique provides. Historically, surgeons performed a radical mastoidectomy, which consisted of removing the TM and all of the middle ear contents, taking down the posterior ear canal wall, and packing the eustachian tube to render it nonfunctional.

◆ Complications

Complications from cholesteatoma and COM are addressed in a separate chapter. The best way to deal with intraoperative complications in otologic surgery is careful technique with avoidance of any operative misadventures. See Chapter 2.2.3 for a complete discussion of the management of intraoperative complications.

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◆ Outcome and Follow-Up

Perioperative antibiotic use varies widely among surgeons. Perioperative antibiotics are prescribed only if the ear cannot be dried prior to the surgery. Surgery is primarily outpatient; however, patients with suspected labyrinthine or cochlear fistulas remain overnight to monitor them for dizziness. Patients return for an office visit at either 1 or 2 weeks. At that visit, most of the reachable packing is removed, and the patient is placed on an ototopical drop nightly. CWD cavities are filled with an antibiotic/steroid ointment, and started on ototopical drops twice daily.

Patients are then seen at 4- to 6-week intervals to inspect the graft and clean any granulations. Water is kept out of the ears until complete epithelialization occurs. CWU ears can return to full preoperative activities once healed. CWD ears can engage in water-related activities with caution, knowing that they are at risk for infection secondary to retained moisture, and dizziness from exposure to cold water.

For surgeons who prefer a staged CWU technique, patients undergo a second-look operation within 6 to 12 months to rule out and manage recurrent or residual disease, and reconstruct the ossicular chain if indicated. If moderate disease is encountered, then a third look may be indicated, and if extensive disease is encountered, a CWD procedure may be performed.

Cholesteatoma patients are followed indefinitely to monitor for recurrence. This involves yearly or twice yearly visits for inspection and mastoid cavity cleaning if needed.

◆ ICD-9 Codes

381.3, 381.4, 382.1, 382.2, 382.3 Chronic otitis media

385.30 Cholesteatoma

Further Reading

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2.3 Otitis Externa

2.3.1 Uncomplicated Otitis Externa

◆ Key Features

- Otitis externa (OE) is an acute or chronic infectious process of the external auditory canal.
- The most common pathogens are *Pseudomonas aeruginosa* and *Staphylococcus aureus.*
- Otomycosis is less common and caused by *Aspergillus* and *Candida.*
- Treatment involves meticulous cleaning and topical preparations.
- Systemic therapy is required if the infection spreads out of the confines of the canal, or the patient is immunocompromised or a poorly controlled diabetic.

Otitis externa is a localized infection of the skin of the external auditory canal (EAC). The EAC contains varying amounts of cerumen and desquamated skin. Acute OE, called "swimmer's ear," is most common after water exposure, but may also follow EAC trauma. Retained moisture will alkalize the canal, making it prone to bacterial infection. As long as the infection is confined to the ear canal, local aural toilet and topical drops will be curative. If the infection extends outside the confines of the canal to become a periauricular cellulitis or the patient has a complicating factor that may impede the effectiveness of topical antibiotics, then oral and occasionally IV antibiotics are required.

◆ Epidemiology

Acute otitis externa affects from 1:100 to 1:250 for the general population. A lifetime incidence may be as high as 10%. The disorder is more common in warm environments with high humidity and increased water exposure.

◆ Clinical

Signs

Discharge, conductive hearing loss, and EAC swelling are all common. Pressing on the tragus or pulling the auricle may lead to significant pain. Signs of otomycosis include itching and visible hyphae on inspection. If the infection has spread to a cellulitis, then the ear may be prominent with an increased auriculocephalic angle, similar to that seen with acute mastoiditis.

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Symptoms

Usually present with a 48- to 72-hour history of progressive pain, itching, discharge, and aural fullness. Patients may also complain of jaw pain. As the skin of the EAC swells, the periosteum is irritated and becomes very painful. If the ear canal fills with debris or swells completely, then hearing loss will also occur. The auricle and periauricular tissues may also become edematous and tender if the condition becomes a periauricular cellulitis.

Differential Diagnosis

Foreign body of the EAC, otitis media, malignant or necrotizing OE in a diabetic, coalescent mastoiditis, malignancy, chronic OE, or other inflammatory lesion (e.g., eosinophilic granuloma). Dermatological conditions (eczema, contact dermatitis) need to be excluded; along with allergic reaction to ear drops; and herpes zoster oticus, which can present with painful EAC vesicles. A localized furuncle may also mimic OE as can COM or AOM with discharge. The presence of inflammation of the tympanic membrane with bullae formation and severe pain indicates bullous myringitis. This is rare, may be associated with influenza, and may be superinfected bacterially. Myringitis is often treated with an oral macrolide antibiotic.

◆ Evaluation

Physical Exam

Inspection may reveal eczema, discharge, swollen canal skin, erythema, moist cerumen, debris, or hyphae. One classic finding is pain with palpation or manipulation of the auricle or the tragus. If visible, the TM may appear inflamed. It should be mobile, however, differentiating it from AOM. Frequently, the skin is so swollen that the TM cannot be seen. The skin of the auricle and periauricular region may also be involved.

Imaging

Imaging is rarely needed. A situation in which imaging can be useful is when the auricle protrudes in a young child with signs of a severe infection. CT can differentiate coalescent mastoiditis versus severe OE with postauricular cellulitis.

Labs

Labs are rarely indicated. The major exception is checking the blood glucose level in the diabetic patient. In diabetics, significantly elevated blood glucose levels and a high sedimentation rate can be helpful in diagnosing necrotizing OE. In patients with signs of spreading cellulitis, or systemic illness, a white blood cell count (WBC) can be helpful.

Other Tests

Culture of the debris is helpful, especially in patients failing empiric therapy. Debris should be sent for routine culture and sensitivity as well as fungus.

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Any abnormal appearing tissue or polyp should be biopsied for histopathology to exclude neoplasm.

Pathology

The infection usually begins with moisture buildup in the EAC. The acidic and hydrophobic qualities of cerumen make it bacteriostatic. A warm, moist EAC with decreased cerumen favors bacterial overgrowth. The bacteria will readily invade the skin. Although >90% are bacterial, a moist alkaline environment also favors fungal growth. *Pseudomonas aeruginosa* and *Staphylococcus aureus* are most common bacteria, and *Aspergillus* and *Candida* are the most common fungi.

◆ Treatment Options

Medical

The ear must be meticulously cleaned under an operating microscope with complete removal of debris. This may need to be repeated in a few days. Once the ear is cleaned, otic drops should be placed. There are currently many preparations available, including nonantibiotic, antibiotic alone, antibiotic plus steroid, and they all show similar efficacy (**Table 2.6**). They may be used two or three times per day for 7 to 14 days.

Placement of a wick (i.e., a fine Merocel sponge; Medtronic XOMED, Inc., Jacksonville, FL) to carry drops medially may be required, especially in canals so swollen that the TM is not easily visible. Keep in mind that placing a wick can be a very uncomfortable experience for the patient. Analgesics, even those with narcotic, may be required to control the pain. As edema regresses, the wick will usually fall out or may be removed. OE due to a foreign body will not resolve without removal of the foreign body.

Table 2.6 Topical Preparations Useful for Management of Otitis Externa

Topical Agent	Typical Dose Regimen
Antibacterial agents	
2% acetic acid	5 drops 2–3 times daily
2% acetic acid/1% hydrocortisone solution	5 drops 2–3 times daily
Floxin Otic	10 drops twice daily
Ciprodex or Cipro HC otic	5 drops twice daily
Cortisporin otic suspension (neomycin/polymyxin/hydrocortisone)	3–5 drops 3 times daily
Antifungal agents	
Lotrimin cream	Apply twice daily
Gentian violet (aqueous)	Physician applies topically under microscope in office as needed

Note: Use in combination with serial débridement; may require Oto-Wick placement if canal is severely swollen.

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Relevant Pharmacology

Topical preparations are superior to systemic medications in cost and a lower rate of side effects. Drug delivery is important, and patients must be educated, aural toilet preformed, and wicks used if indicated. The local medication concentration is far superior with topicals than with systemics.

Ototopical antibiotic drops: Cortisporin (neomycin/polymyxin/hydrocortisone), ofloxacin, or ciprofloxacin. Acidifying solutions such as Domeboro (2% acetic acid; Bayer Consumer Health, Morristown, NJ) or VoSoL (2% acetic acid and propylene glycol; Wallace Labs, Cranbury, NJ) will treat both *Pseudomonas* and otomycosis. There is a 10 to 15% sensitization rate with neomycin containing products. Currently, U.S. Food and Drug Administration (FDA)-approved preparations for possible middle ear exposure are ofloxacin and ciprofloxacin/dexamethasone.

Surgical

There is no specific role for surgery in AOE. Patients may occasionally require a general anesthetic to carry out the canal cleaning and wick placement if the ear is too tender to instrument in the office. If a foreign body is suspected, especially in a child, then a general anesthetic may be needed to remove it.

Chronic OE results after fibrosis thickens the skin of the EAC, sometimes to the point of completely obliterating the canal. In some of these cases a canaloplasty with meatoplasty and skin grafting may be necessary.

◆ Outcome and Follow-Up

Most cases resolve within 7 to 10 days of treatment. Causative factors such as eczema, swimming, or cotton swab (Q-tip) use need to be addressed to promote resolution and prevent recurrence. Patients may need to be seen frequently in the office for repeated cleanings until the infection resolves. Patients should keep the ears dry for 7 to 10 days. Swimmers can return to the water with plugs in 3 days, and hearing aids can be replaced after pain and discharge resolve.

◆ ICD-9 Code

380.1 Otitis externa

Further Reading

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2.3.2 Malignant Otitis Externa

◆ Key Features

- Malignant otitis externa (MOE) is most frequently caused by *Pseudomonas aeruginosa*.
- Patients are immunocompromised, most commonly with diabetes.
- It is characterized by severe deep aural pain, especially at night.
- The facial nerve is the most common cranial nerve involved.

MOE is a severe, serious form of OE caused by *Pseudomonas* that if not diagnosed and treated early, progresses to skull base osteomyelitis, cranial nerve palsies, and potentially death. Surgical management is limited to local ear canal débridement, and treatment is medical with long-term antipseudomonal antibiotics. Diagnosis is made clinically and corroborated with imaging and nuclear medicine scans.

◆ Epidemiology

90% of patients are diabetic, and most are over 60 years old. Other predisposing factors include hearing aid use, history of OM, leukemia, alcoholism, and swimming. Unsterile tap water cerumen irrigations may also be a risk factor.

◆ Clinical

Signs

On physical examination, patients will have external canal and periauricular tissue edema and tenderness. The TM may be normal or inflamed with a middle ear effusion. There is granulation tissue along the floor of the canal at the osseocartilaginous junction. Palsy of CNs VII, IX, X, and XII may be present.

Symptoms

Aural pain and discharge are not responsive to the usual measures. Pain is often severe at night and may awaken patients from sleep. Also, headache and temporomandibular joint (TMJ) pain are common. Symptoms typically persist for more than a week. As the disease progresses, patients may complain of lethargy, nausea, blurred vision, and mental confusion.

Differential Diagnosis

MOE must be differentiated from routine OE or AOM with mastoiditis. Fungal MOE must also be considered. Granulation tissue should be biopsied to rule out a malignancy.

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◆ Evaluation

History

The history should focus on elucidation of predisposing factors, such as diabetes, immunosuppression, leukemia, alcoholism, and swimming. If the patient is diabetic, information regarding recent glucose levels and hemoglobin A1c (HbA1c) is useful.

Physical Exam

Include a microscopic ear exam with thorough cleaning of the ear canal and placement of a myringotomy tube if necessary. This may require general anesthetic if pain is severe. A complete head and neck exam with attention to cranial nerves is also required. Parotid palpation and TMJ mobility is noted. See **Table 2.7** for a consideration of diagnostic tests.

Imaging

CT of the temporal bone is often the initial study. Fluid is often seen in the mastoid and middle ear. More importantly, soft tissue edema can be seen in the ear canal and periauricular tissues, as well as the parapharyngeal, infratemporal, and subtemporal spaces. Bone erosion may or may not be detectable depending on its extent. CT is not especially helpful in determining the endpoint of treatment.

MRI of the skull base is helpful in detecting the progression of disease. Gadolinium-enhanced studies with fat suppression will demonstrate involvement of the dura and other skull-base soft tissues.

Technetium-99m (^{99m}Tc) nuclear scanning highlights areas of osteoblastic activity seen in osteomyelitis. Increased uptake indicative of skull-base

Table 2.7 A Consideration of Key Diagnostic Tests for Malignant Otitis Externa

Labs
CBC with differential (follow ANC level if neutropenic), blood glucose level (serially), HbA1c, ESR (serially)
Pathology
Histopathologic exam of granulation tissue to exclude neoplasm
Microbiology
Bacterial and fungal culture and sensitivities of ear canal debris
Imaging
Temporal bone CT to assess for extent of disease, bone erosion
MRI with gadolinium may be helpful to assess dura, soft tissue involvement.
Nuclear scanning: Technetium-99m scan to assess for skull base osteomyelitis; gallium-67 citrate nuclear scanning preferred for follow-up and assessment of disease resolution

Abbreviations: HbA1c, hemoglobin A1c; ANC, absolute neutrophil count; CBC, complete blood count; CT, computed tomography; ESR, erythrocyte sedimentation rate; MRI, magnetic resonance imaging.

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osteomyelitis is seen between 4 and 24 hours after ^{99m}Tc administration. This modality may be positive prior to bony changes seen on CT, but cannot be used to determine the endpoint of treatment, as it may remain positive for months to years as a sign of bony repair. It is very helpful in confirming an early diagnosis.

Gallium-67 (^{67}Ga) citrate nuclear scanning highlights areas of inflammation by binding to leukocytes. It also reverses back to normal once the inflammatory response has resolved, so it is helpful in determining the treatment endpoint. It may be repeated several times throughout the treatment course to monitor response.

Labs

Routine laboratory testing will display an increased WBC count, an increased ESR, and elevated blood glucose. Serial ESR levels can be used to monitor treatment response.

Other Tests

The ear canal should be swabbed for culture and sensitivity. Granulation tissue needs to be sent for permanent pathology sectioning. An audiogram is good as a baseline measure.

Pathology

Most often *Pseudomonas aeruginosa* is the causative bacteria. This should be confirmed with a culture. Infection begins at the skin of the EAC and spreads via venous channels and fascial planes. Infection can also spread to the skull base via fissures of Santorini and tympanomastoid suture line. Cranial nerves become involved in the soft tissue inflammatory response at the stylomastoid foramen and the jugular foramen.

Patients are immunocompromised, most commonly secondary to diabetes mellitus. Their cerumen is neutral pH, not acidic, and many have had their ear canals irrigated with nonsterile tap water 2 to 3 weeks prior to symptom onset. Patients with human immunodeficiency virus (HIV) or hematologic malignancies are prone to fungal OE, mainly secondary to *Aspergillus*.

◆ Treatment Options

Medical

Medical therapy is the mainstay once the ear canal is cleaned and all granulation débrided. A myringotomy tube may be required if middle ear fluid is present. Once cultures and sensitivities return, long-term culture-directed antibiotics can be used (**Table 2.8**). Traditional therapy has been an IV antipseudomonal antibiotic and an aminoglycoside. Nephrotoxicity in diabetics given aminoglycosides is a concern, so ceftazidime has been given as monotherapy. Drug resistance, however, is a concern with monotherapy.

Fluoroquinolones, primarily ciprofloxacin, can be given orally with excellent cure rates, but again, concerns of drug resistance have arisen. As long as resistance does not emerge, oral fluoroquinolones can be used, and average

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Table 2.8 Antibiotic Therapy for Malignant Otitis Externa

Intravenous therapy
Fluoroquinolone or 3rd/4th generation cephalosporin + aminoglycoside Ceftazidime monotherapy is considered if aminoglycoside is contraindicated due to nephrotoxicity/diabetic nephropathy.
Oral therapy
Possible fluoroquinolone (i.e., ciprofloxacin) use, only if no drug-resistant *Pseudomonas* is confirmed

Note: Therapy should be culture-directed whenever possible. Duration of 6 or more weeks is typical, following erythrocyte sedimentation rate.

treatment durations are 4 to 6 weeks. A peripherally inserted central catheter (PICC) line or central venous catheter is typically required. If resistance emerges, then long-term IV management is required. The endpoint of therapy is individualized with gallium scanning, and treatment courses have been documented to run several months.

Surgical

Surgical management is limited to initial canal débridement and myringotomy tube placement if needed.

◆ Complications

Progressive MOE becomes osteomyelitis of the skull base. With spreading infection, CNs VII, IX, X, and XI may become involved. Consequences include aspiration with difficulties in speech and swallowing. CNs III, V, and VI may also be involved. Meningitis, lateral sinus thrombosis, cerebral abscess, and death may all occur.

◆ Outcome and Follow-Up

With proper diagnosis and treatment, cure rates are over 80%. Patient follow-up is dictated by symptoms. Treatment is complete when the TM and ear canal return to a normal appearance, pain resolves, ESR returns to normal, and the gallium scan returns to normal.

◆ ICD-9 Code

380.14 Malignant otitis externa

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2.4 Audiology

2.4.1 Basic Audiologic Assessments

◆ Key Features

- Pure tone testing – air and bone conduction thresholds
- Speech testing – speech reception thresholds (SRTs) and word recognition ability
- Immittance battery – tympanometry, acoustic reflex thresholds, acoustic reflex decay, eustachian tube function assessment

Air and bone conduction pure tone testing determines the type, degree, and configuration of hearing loss. Thresholds represent the softest sound audible at least 50% of the time measured in decibels. SRT serves to verify pure tone thresholds. Word recognition ability represents an individual's ability to discriminate speech. Immittance testing assesses the status of the middle ear and acoustic reflex pathways.

◆ Epidemiology

Approximately 28 million Americans have hearing loss. Approximately 314 in 1000 people over age 65 have hearing loss, as do 40 to 50% of people over age 75.

◆ Clinical

Signs and Symptoms

Persons with hearing loss frequently report difficulty hearing in background noise. They mistakenly feel others around them are mumbling. They often need the television volume louder than is comfortable for others and ask for things to be repeated. Many experience tinnitus.

Differential Diagnosis

Results of audiometric testing are typically plotted on an audiogram (**Fig. 2.7**). An audiogram is a graph with frequency (in Hz) plotted on the *x* axis and intensity (in decibels) plotted on the *y* axis. It is necessary to employ masking (a static type of noise) in certain situations to prevent the nontest ear from detecting the test signal.

Results of the audiogram and tympanogram (**Fig. 2.8**) may suggest various forms of otologic disease. Red flags should prompt referral to

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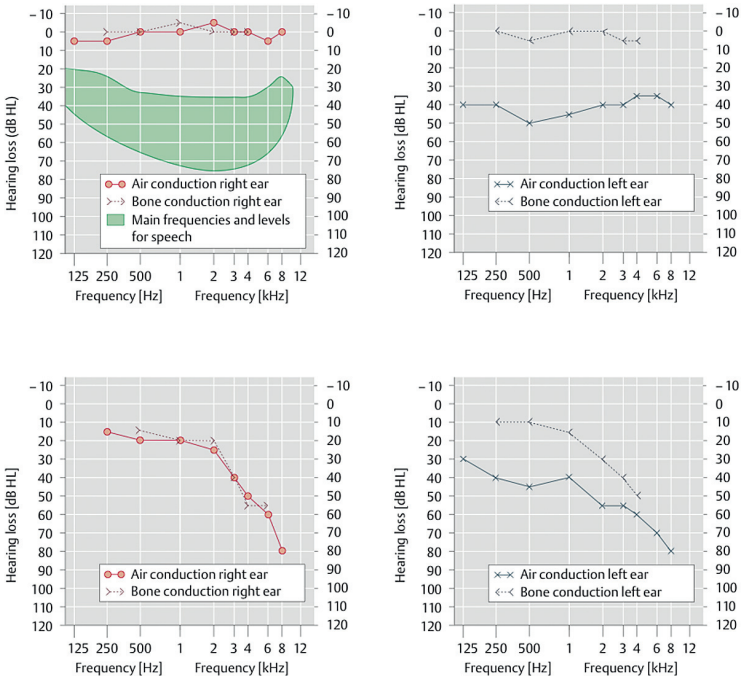

Fig. 2.7 Pure-tone threshold audiometry. **(A)** Normal pure-tone audiogram. **(B)** Audiogram showing conductive hearing loss. **(C)** Audiogram showing sensorineural hearing loss. **(D)** Audiogram showing mixed hearing loss. (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:179.)

an otolaryngologist (discussed below). Otologic disease may be categorized as conductive hearing loss, sensorineural loss, or mixed hearing loss. In a conductive loss, there is an air–bone gap on the audiogram, with better bone-conduction thresholds, with appropriate masking. The loss may be due to dysfunction involving the external canal, tympanic membrane, or ossicular chain, or middle ear fluid. In a sensorineural loss, bone conduction thresholds typically equal air conduction, and the loss may be due to dysfunction at the level of the cochlea, CN VIII, or central pathways. A poor word-recognition score, especially out of proportion to pure tone thresholds, suggests possible retrocochlear pathology, such as an acoustic tumor.

Patterns of sensorineural loss may be suggestive of certain etiologies, taken together with clinical history and exam. For example, acoustic trauma (noise-induced loss) often results in a loss centered around 4 kHz with recovery at higher frequency, i.e., a 4-kHz notch; presbycusis often involves

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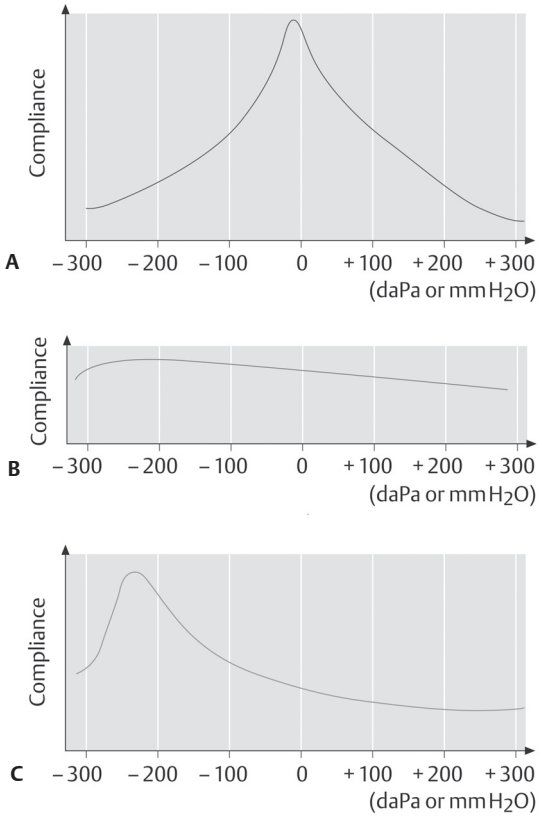

Fig. 2.8 Normal and abnormal tympanogram patterns. **(A)** The normal tympanogram has a prominent, sharp peak between +100 and −100 daPa. **(B)** The type B tympanogram is flat or has a very low, rounded peak. This indicates immobility of the tympanic membrane, which may be due to fluid in the middle ear or tympanic atelectasis. A type B tympanogram with large volume is seen with the perforation. **(C)** The type C tympanogram has a peak in the negative pressure region below −100 daPa, consistent with impaired middle ear ventilation. (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:185.)

symmetric downsloping loss worse at higher frequency; a fluctuating low-frequency loss is often seen with Ménière disease.

The Jerger system is commonly used to classify tympanometric results:

- Type A – Normal
  - Pressure peak +100 to −100 daPa
  - Immittance 0.3 to 1.7 mL

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- Type As – Shallow
  - Pressure peak +100 to −100 daPa
  - Immittance <0.3 mL
- Type Ad – Deep
  - Pressure peak +100 to −100 daPa
  - Immittance >1.7 mL
- Type C – Negative pressure
  - Pressure peak ≤100 daPa
  - Immitance normal or shallow
- Type B – Flat
  - No pressure peak or immittance
  - Small ear canal volume suggests ear canal occlusion.
  - Normal volume suggests otitis media.
  - Large ear canal volume suggests patent pressure equalization tube or perforated eardrum.

◆ Evaluation

Tuning Fork Tests

The goal of these tests, including the Weber test (**Fig. 2.9**), is to differentiate between conductive and SNHL. The tuning forks vibrate at between 256 to 1024 Hz. To test bone conduction, the fork is placed firmly on the skull bone. Typically, a 512-Hz tuning fork is used.

Weber Test

The Weber test will lateralize to the ear with conductive loss, if sensory hearing is equal (see **Fig. 2.9**).

Rinne Test

The Rinne test (**Fig. 2.10**) compares the level of air conduction and bone conduction in the same ear. In a normal ear, air conduction is greater. It usually takes about a 30-dB conductive loss to flip the tuning fork response, to bone conduction greater than air.

Physical Exam

Test results are best obtained in a sound-treated booth. Insert earphones placed in the ear canal are the transducer of choice in assessing air-conduction thresholds due to infection control, increased interaural attenuation, and prevention of collapsed ear canals. Supra and circumaural headphones are options as well in certain situations. Bone conduction is assessed by placing an oscillator on a mastoid bone. Forehead placement is another option in special situations.

Conventional behavioral audiometry is conducted by having a person respond to pure tones (e.g., raising a hand, pushing a button). SRTs are obtained

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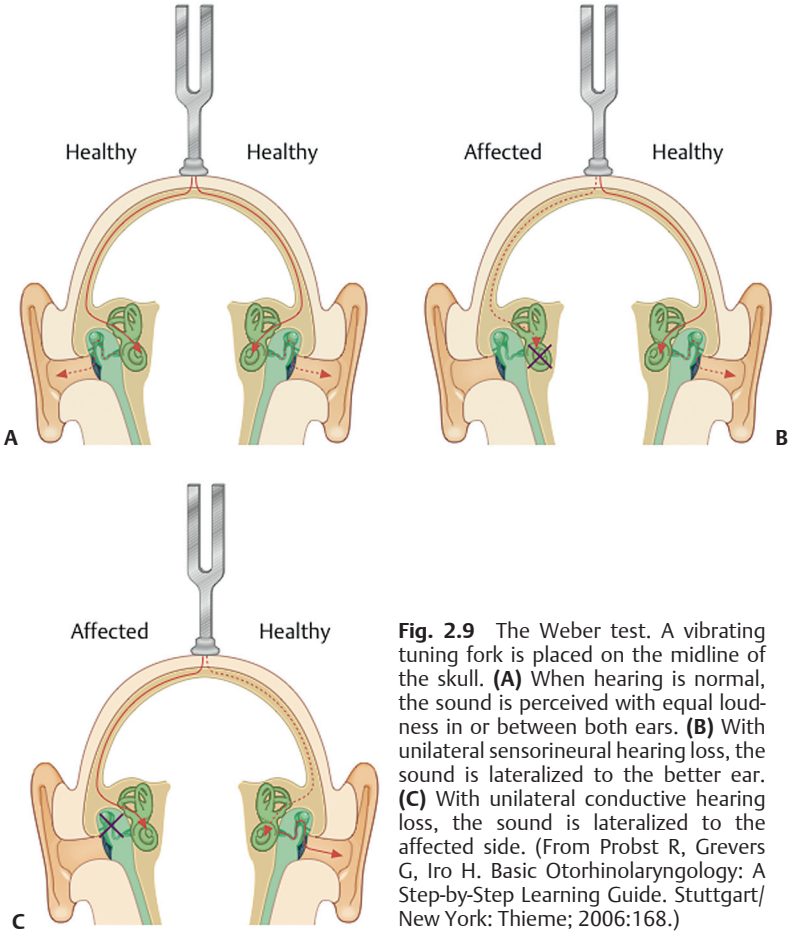

Fig. 2.9 The Weber test. A vibrating tuning fork is placed on the midline of the skull. **(A)** When hearing is normal, the sound is perceived with equal loudness in or between both ears. **(B)** With unilateral sensorineural hearing loss, the sound is lateralized to the better ear. **(C)** With unilateral conductive hearing loss, the sound is lateralized to the affected side. (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:168.)

by repeating spondees (two-syllable words—e.g., hotdog, baseball). Word recognition is assessed by repeating phonetically balanced lists of monosyllabic words at suprathreshold intensity. In tympanometry, the maximum compliance peak is determined by presenting a probe tone (typically 1000 Hz in infants up through 6 months of age, 226 Hz in all others) in the sealed ear canal while varying air pressure (+200 to −400 daPa). Maximum compliance occurs when the middle and external ear air pressures are equal.

Other Tests

Acoustic reflex thresholds and acoustic reflex decay can be measured both ipsilaterally and contralaterally and across different frequencies. A probe tone is presented in either ear while a change in immittance is monitored.

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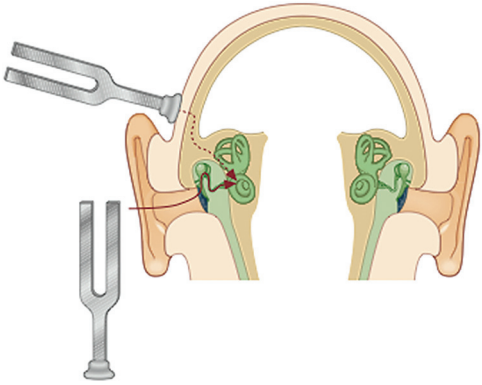

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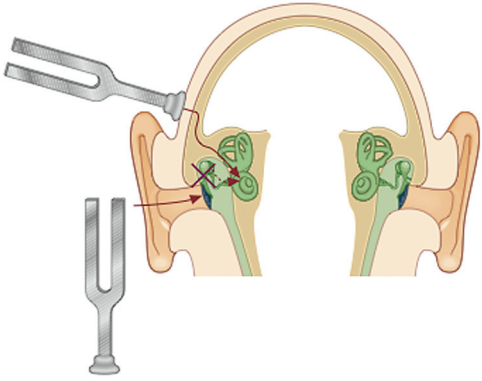

B

Fig. 2.10 The Rinne test. Air and bone conduction are compared in the same ear to determine the auditory threshold for the tuning fork and/or its loudness. **(A)** In the absence of conductive hearing loss, air conduction is perceived as being louder and/or of longer duration than bone conduction. **(B)** When conductive hearing loss is present, bone conduction is perceived as being louder and/or more prolonged than air conduction. (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:69.)

This assesses the acoustic pathways by measuring the contraction of the stapedius muscle in response to acoustic stimulation. Eustachian tube function testing reveals how well middle ear pressure is equalized. Testing occurs in the same manner as basic tympanometric testing, but with a person swallowing in between artificial changes in ear canal air pressure.

Pathology

Hearing loss may be congenital, due exogenous sources such as perinatal infection or due to a genetic cause (hereditary). Hereditary loss may be syndromic or nonsyndromic. Hearing loss may also, of course, be acquired. Acquired hearing loss is frequently related to the aging of the auditory system (presbycusis). It

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can also occur from trauma, disease, excessive noise exposure, and ototoxic medications. Hearing loss may remain stable over time or be progressive.

◆ Treatment Options

Patients with unilateral or conductive hearing losses should be referred for medical evaluation. Other red flags should prompt otolaryngology referral, such as evidence of a sudden hearing loss, rapidly progressing or fluctuating sensorineural loss, word recognition score poorer than expected based on pure tone thresholds, word recognition score asymmetry greater than 10%, evidence of middle ear dysfunction, as well as evidence of otorrhea, or any hearing loss in a child.

◆ Outcome and Follow-Up

Following medical clearance, a variety of amplification devices are available and appropriate for most persons with hearing loss.

Further Reading

Audiology Awareness Campaign. Hearing Information and Resources. Available at: http://www.audiologyawareness.com/hearinfo.asp

WebMD. Otolaryngology and facial plastic surgery articles. Available at: http://www.emedicine.com/ent/index.shtml

National Institute on Deafness and Other Communication Disorders. Statistics about hearing disorders, ear infections, and deafness. Available at: http://www.nidcd.nih.gov/health/statistics/hearing.asp

2.4.2 Pediatric Audiologic Assessments

◆ Key Features

- The goal of universal newborn hearing screening is to have all babies screened by 1 month, diagnostic testing when needed by 3 months, and rehabilitation by 6 months.
- There are more stringent cutoffs for normal hearing thresholds in pediatric patients: 15- to 20-dB hearing loss, as opposed 25-dB hearing loss in adults.
- Testing protocols need to be modified to obtain the same audiologic information readily acquired from cooperative adults.

Prior to the implementation of newborn hearing screenings, children with mild hearing loss were often not identified until speech/language difficulties were already occurring or upon entering school. Children with chronic/recurrent otitis media may also experience difficulties due to fluctuating hearing loss.

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◆ Epidemiology

About 2 to 3 out of every 1000 children in the United States are born with hearing loss. Most of these babies are otherwise healthy and have no family history of hearing loss.

◆ Clinical

Signs and Symptoms

Even mild, untreated hearing loss in infants and young children can significantly impact their speech, language, and learning development. Children do not have the experience to fill in the acoustic cues they are missing. Any concern expressed by caregivers should be formally evaluated. Informal assessments are not adequate in evaluating a child's hearing.

◆ Evaluation

Physical Exam

Depending on the developmental age of the child, behavioral observation audiometry, visual reinforcement audiometry, and play audiometry are utilized to obtain pure tone thresholds. With any of these tests, results are most reliable when testing is conducted with two people (i.e., one person performing the test protocol and one person in the booth to redirect the child—it is typically not advantageous to use a child's parent for this role). Testing with insert earphones is preferred so that ear-specific information can be obtained (headphones may also be used in some instances, but caution is needed to avoid collapsing young ear canals). Testing can also be conducted in the sound field; however, ear-specific information will not be known. Warbled tones and narrowband noise are frequently used in testing children in addition to pure tones.

In behavioral observation audiometry, examiners look for physical changes in a child in response to sound (i.e., blinking or other eye movement, cessation of sucking). In visual reinforcement audiometry, children are rewarded (i.e., a lighted box with an animated character) by turning their head in response to sound. In play audiometry, children are conditioned to perform a task (i.e., drop blocks in a bucket, put pegs on a board) in response to pure tones. Speech awareness thresholds may be obtained in place of speech reception thresholds. Picture boards can also help in obtaining speech information. High-frequency probe tone (1000 Hz) testing is needed in tympanometry to appropriately assess the middle ear function in children 6 months of age or younger. In some children, behavioral information should be confirmed through objective/electrophysiologic testing. If the child has severe cognitive impairment or is otherwise unable or too young to cooperate for behavioral testing, objective testing should be obtained, such as auditory brainstem response (ABR) testing.

◆ Treatment Options

The priority in children is to obtain appropriate auditory rehabilitation as soon as the problem is identified. Because plasticity within the audi-

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tory cortex is finite and diminishes dramatically after the first few years, a failure to obtain auditory input within this critical period will result in cortical deafness, i.e., an inability of the brain to adequately process auditory input later in life if aided or implanted. Thus, if hearing loss cannot be corrected by treating obvious causes such as middle ear fluid, amplification should be obtained. If there is no benefit from appropriate amplification, cochlear implant (CI) evaluation should be sought as soon as possible. There are many modifications available to assist in the fitting of hearing aids to even very young infants and children. To accommodate ear growth, children are fit with behind-the-ear hearing aids and their earmolds need to be replaced frequently. In addition to hearing aids, children may benefit from use of FM systems in classrooms to improve the signal-to-noise ratio. FM systems provide a handheld microphone for teachers to talk into, which transmits the sound of the teacher's voice directly to child's hearing aid.

◆ Outcome and Follow-Up

Children diagnosed with SNHL should receive an otologic consult, an ophthalmologic consult, possibly genetic counseling, and routine monitoring of their hearing. Depending on the origin of the hearing loss, siblings of diagnosed children should also have their hearing assessed. Children with high risk factors for hearing loss should receive periodic reevaluations as well.

Further Reading

American Academy of Pediatrics, Joint Committee on Infant Hearing. Year 2007 position statement: principles and guidelines for early hearing detection and intervention programs. Pediatrics 2007;120(4):898–921

Madell JR, Flexer C, eds. Pediatric Audiology: Diagnosis, Technology, and Management. Stuttgart/New York: Thieme; 2008

2.4.3 Objective/Electrophysiologic Audiologic Assessments

◆ Key Features

- Otoacoustic emissions (OAEs)
- Auditory brainstem responses (ABRs; aka BAER, BSER)
- Auditory steady-state responses (ASSRs)
- Electrocochleography (ECochG)
- Electroneuronography (ENoG)

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Otoacoustic emissions are a reflection of the status of the outer hair cells in the cochlea. ABR, ASSR, and ECochG measurements are reflective of auditory nerve and brainstem functions. ENoG is reflective of distal facial nerve functioning.

◆ Epidemiology

Transient evoked and distortion product OAEs are present in >99% of persons with normal hearing. ABR norms vary with age in children under 2. ECochG sensitivity is only 60–65%. ENoG testing should not be conducted before 48 to 72 hours after injury onset but has little value beyond 21 days after injury.

◆ Clinical

OAEs can be influenced by middle ear status and hearing loss. Distortion product and transient evoked OAEs are the most frequently employed. Spontaneous and sustained frequency OAEs do not have clinical significance.

ABR, ASSR, and ECochG measurements can be influenced by hearing loss, test parameters (stimulus rate and intensity), patient factors (movement, body temperature), and environmental factors (ambient noise, electrical interference).

ENoG measures can be influenced by medications that induce chemical paralysis of muscles.

◆ Evaluation

OAEs are assessed by presenting a stimulus in the ear canal through a probe tip. The response of the ear is then measured in the same probe tip.

ABRs and ASSRs are assessed and ECochG performed by presenting a stimulus to the ear canal. Electrical potentials are then recorded through electrodes on the mastoids, earlobes, forehead, and/or tympanic membranes.

ENoG is performed by presenting electrical stimulus near the ear and cheek bone. Electrical potentials are then recorded through electrodes at the base of the nose and at the corner of the mouth.

◆ Uses

Otoacoustic emissions are valuable in newborn hearing screenings, difficult to test patients, assessing auditory neuropathy, and in chemotherapy monitoring.

ABRs and ASSRs are used in hearing threshold estimation. ABRs are also used in newborn hearing screenings, neurodiagnostics (i.e., vestibular schwannomas, auditory neuropathy/dys-synchrony) and intraoperative monitoring. The five waves seen on ABR can be recalled using the mnemonic "ECOLI": eighth nerve, cochlea, inferior olive, lateral lemniscus, inferior colliculus. Prolongation of wave five latency is suggestive of an acoustic tumor.

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ECochG is used in assessing endolymphatic hydrops and in intraoperative monitoring. A demonstration of elevated SP/AP ratio (cochlear summating potential/ auditory nerve action potential) may help confirm the diagnosis of Ménière disease in the appropriate clinical setting.

ENoG is used in assessing residual facial nerve function or possible recovery. The presence of only fibrillation potentials suggests denervation.

Further Reading

Hall JW III. Handbook of Auditory Evoked Responses. Needham Heights, MA: Allyn & Bacon; 1992
Hall JW III. Handbook of Otoacoustic Emissions. San Diego, CA: Singular Publishing Group; 2000
Robinette MS, Glattke TJ. Otoacoustic Emissions: Clinical Applications. 3rd ed. Stuttgart/ NewYork: Thieme; 2007

2.5 Hearing Loss

2.5.1 Conductive Hearing Loss

◆ Key Features

- Conductive hearing loss (CHL) occurs when sound is not adequately transferred from the outside world to a functioning cochlea.
- CHL can occur due to dysfunction of the external auditory canal (EAC), the tympanic membrane, or the ossicles.
- Most cases of CHL can be surgically corrected.

The causes of conductive hearing loss are extremely varied. However, causes may be categorized by location:

- External ear: Blockage of the EAC from impacted cerumen or foreign bodies may cause up to 40 dB CHL. Congenital canal atresia, inflammatory stenosis with acquired atresia, or bony exostoses can also diminish sound conduction to the middle ear.
- Tympanic membrane: Any mechanism preventing proper vibration of the ear drum will lead to CHL. Perforations of any size may interfere with the sound collection. Thickening of the drum from scarring, tympanosclerosis, negative middle ear pressure, atelectasis, and middle ear effusion may also prevent adequate vibration of the drum. Controversy

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exists regarding how the size and position of a perforation contributes to the degree of conductive loss.

- Ossicles: Interruption of the continuity or mobility of the ossicular chain leads to CHL. Common causes of decreased ossicular mobility are malleus fixation, middle ear adhesions, tympanosclerosis, and otosclerosis. Incus erosion or traumatic ossicular dislocation with an intact TM may lead to a maximal CHL of 60 dB.

◆ Evaluation

Physical Exam

Binocular microscopic evaluation of the EAC and drum is extremely helpful in identifying pathology of CHL, which may otherwise be missed with handheld otoscopy. Pneumatic otoscopy can assist in identifying tympanic membrane mobility issues and should always be documented. Complete removal of cerumen aids in visualization.

Imaging

Imaging studies are typically not needed for cases of CHL. Selective use of high-resolution CT of the temporal bones may detect middle ear masses, otosclerotic foci in the otic capsule, but usually is not of adequate resolution to detect subtle ossicular abnormalities.

Other Tests

Comprehensive audiometry with both air conduction and bone conduction testing should be performed to determine air–bone gap, which represents the amount of hearing loss attributed to the conductive mechanism. Tympanometry can be performed to determine the mobility of the drum, detecting perforations and middle ear effusions. Acoustic reflexes should be absent. If acoustic reflexes are present in a patient with a conductive hearing loss, superior semicircular canal dehiscence should be suspected. In this case, cervical vestibular evoked myogenic potential (cVEMP) testing can be helpful, as superior semicircular canal dehiscence syndrome patients typically demonstrate larger amplitudes and lower thresholds on cVEMP testing at 500 Hz. Tuning fork testing at 512 Hz, specifically Rinne testing to compare air conduction and bone conduction, is often a helpful adjunct, especially when determining surgical candidacy.

◆ Treatment Options

Medical

Amplification and observation may always be initially offered to patients, unless their CHL is caused by a treatable, progressive disease or a neoplasm. Surgical intervention will most often result in hearing improvement in patients with significant air–bone gaps.

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Surgical

Outer Ear

- Cerumen removal
- Foreign body removal
- Canaloplasty with or without skin graft (for exostosis or stenosis)
- Meatoplasty

Tympanic Membrane

- Myringotomy with or without placement of a tympanostomy tube for middle ear effusion
- Paper patch or fat myringoplasty for small perforation (often office procedures)
- Tympanoplasty

Middle Ear

- Ossicular chain reconstruction using remodeled incus or synthetic prosthesis
- Stapedectomy/stapedotomy
- Exploratory tympanotomy with possible
  - Lysis of middle ear adhesions (tympanosclerosis)
  - Repair of malleus fixation
  - Removal of cholesteatoma or neoplasm

◆ Typical Clinical Pictures

Otosclerosis

Otosclerosis is a process in which the stapes loses mobility by excessive bony growth at the oval window. It typically causes a CHL with a distinctive dip in sensorineural levels at 2 kHz, known as Carhart's notch. Patients may have tinnitus, progressive hearing loss in one or both ears. Otosclerosis has a bimodal age epidemiology: it usually presents in the early twenties or later in the fifties. Early otosclerosis can be managed with fluoride supplementation, but often the CHL will progress and patients will need to consider amplification or stapes surgery. Stapedectomy or stapedotomy involves removing the superstructure of the stapes, and entering into the inner ear, either by making a fenestration in the footplate (stapedotomy), or partially removing it (stapedectomy). A synthetic prosthesis is then placed into the oval window and attached to the incus to reestablish ossicular transmission of sound waves.

Ossicular Disease

Cholesteatoma, chronic ear infections, and trauma can all cause erosion or disruption of the ossicular chain. After appropriately treating the aforementioned

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causes, one may evaluate the remaining middle ear structures to determine the appropriate surgical repair. Once the current ossicular status (anatomic and functional integrity of each ossicle) is determined, the appropriate prosthesis can be selected. Prostheses exist for nearly every situation of ossicular disease. Common prostheses include a partial ossicular reconstruction prostheses (replaces incus and malleus), incus struts (replace incus only), and a total ossicular reconstruction prostheses (replaces all ossicles).

◆ Complications

Revision surgery may be necessary after ossicular chain reconstruction if the prosthesis shifts and no longer conducts sound effectively. Six months is generally considered the earliest time point at which revision surgery should be considered. Progressive hearing loss 10 to 14 days after stapes surgery may indicate reparative granuloma, and middle ear exploration should be considered.

◆ Outcome and Follow-Up

Water precautions should be observed in the postoperative period. If the TM is grafted or a stapedotomy is performed, caution regarding airline flight should be considered in the immediate postoperative period to avoid TM movement. Smoking cessation should be encouraged.

A repeat audiogram to determine level of hearing restoration should be performed 6 to 8 weeks after procedure (adequate time for Gelfoam packing in the middle ear to absorb).

◆ ICD-9 Codes

382, 384.21, 389.03, 389.05, 389.06, 385.02–385.33 Conductive hearing loss

Further Reading

De Souza C, Glasscock ME. Otosclerosis and Stapedectomy. Stuttgart/New York: Thieme; 2004

Tos M. Surgical Solutions for Conductive Hearing Loss. Stuttgart/New York: Thieme; 2000

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2.5.2 Sensorineural Hearing Loss

◆ Key Features

- Hearing loss is caused by the dysfunction of the sensory (cochlea) or neural components of the auditory system.
- Asymmetric hearing loss requires MRI or ABR to rule out retrocochlear pathology.
- Amplification is the best option for presbycusis or familial hearing loss.

Hearing loss is one of the most common medical problems and it is often left undiagnosed and undertreated. Hearing loss can usually be grouped into symmetric and asymmetric losses. Asymmetry is often described as a 10-dB difference in three consecutive pure tone frequencies, a 15-dB difference in two consecutive frequencies, and/or a ≥12% point difference in speech recognition.

◆ Clinical

Signs and Symptoms

Patients typically notice difficulty in crowds or with background noise. High-pitch voices may also be more difficult to understand. Many patients will not realize they have hearing loss until it has progressed significantly, as the hearing loss is usually very gradual. Often, tinnitus is associated with hearing loss and may be the presenting symptom.

Differential Diagnosis for Symmetric Hearing Loss

Presbycusis

Slowly progressive bilateral hearing loss in the older population (presbycusis) is widely prevalent and is the most common form of SNHL. It is often a familial condition. However, a detailed otologic history may find other factors that may contribute to SNHL.

Noise-Induced Hearing Loss

Exposure to loud noise can lead to permanent hearing threshold shifts. This may happen immediately with extreme exposure (nearby explosion or gunfire), but more commonly occurs slowly over time with repeated exposure to industrial or environmental noise. Patients will often have a typical 4-kHz notch in their audiograms. Patients should be counseled to prevent further noise damage by wearing appropriate hearing protection.

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Table 2.9 Common Ototoxic Drugs

Aminoglycoside antibiotics
Platinum-based chemotherapy (especially cisplatin)
Loop diuretics
Vinca alkaloids (especially vincristine)
Quinine
Salicylates

Ototoxicity

Exposure to a variety of medications may induce permanent hearing loss (**Table 2.9**). This hearing loss is typically first noted in the highest frequencies, and then progresses to lower pitches. Common agents include aminoglycoside antibiotics, vinca alkaloids, and platinum-based chemotherapeutic agents. Careful monitoring of audiograms during therapy allow for early identification of hearing loss. Prolonged use of high dose loop diuretics (e.g., furosemide) may also lead to hearing loss. Of note, many ototoxic drugs are also nephrotoxic, therefore renal function studies should be obtained as well.

Hearing Loss Present at Birth

See **Fig. 2.11** for a classification overview of hearing loss at birth. About 50% of cases are nonhereditary, i.e., due to prematurity, sepsis, or TORCHES infections (toxoplasmosis, rubella, cytomegalovirus, herpes simplex encephalitis, and otosyphillis). Of the 50% that are congenital hereditary cases, these may be syndromic (one third of cases) or nonsyndromic (two thirds

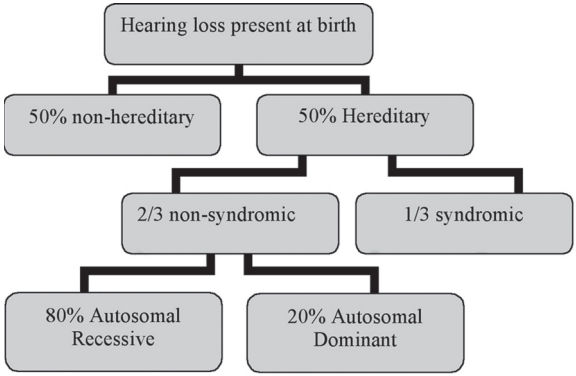

Fig. 2.11 Hearing loss present at birth: a classification overview.

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Table 2.10 Hereditary Syndromic Hearing Loss

Autosomal Dominant	Autosomal Recessive	Sex-Linked
Treacher-Collins	Usher	Alport
Goldenhar	Pendred	
Waardenberg	Jervell-Lange-Nielson	
Branchio-oto-renal		
Stickler		

of cases); see **Table 2.10**. Of the nonsyndromic cases, ~80% are autosomal recessive.

Hearing loss may be present at birth due to congenital defects in either the structure or the physiology of the inner ear. Cochlear malformations (e.g., Mondini malformation) may be associated with various syndromes. Many cases of nonsyndromic congenital hearing loss have been attributed to chromosomal defects in the hair cell protein connexin 26 (Cx 26). Most congenital cases are now discovered early due to universal newborn screening programs. However, certain types, e.g., enlarged vestibular aqueduct syndrome, may not manifest until later in life.

Metabolic

Symmetric bilateral rapidly progressive hearing loss may be caused by a variety of systemic diseases, including autoimmune disease.

Other

Cochlear otosclerosis, primary autoimmune inner ear disease, barotrauma, perilymph fistula, migraine, vertebrobasilar occlusion, and a variety of autoimmune disorders may all be associated with SNHL.

Differential Diagnosis for Asymmetric Hearing Loss

Tumors

Neoplasms of the CPA and/or internal auditory canal, such as vestibular schwannomas and meningiomas may cause unilateral hearing loss, and are the primary pathologies the must be ruled out when encountering significant asymmetric hearing loss. Metastatic disease and endolymphatic sac tumors must also be considered. Diseases of the temporal bone such as fibrous dysplasia and Paget disease can cause hearing loss through destruction of the otic capsule.

Ménière Disease

Active Ménière disease typically causes a fluctuating low-frequency hearing loss. As the disease progresses, higher frequencies are effected and can progress to severe levels.

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Infectious

Meningitis may lead to the spread of bacteria to the inner ear through the cochlear aqueduct, which can lead to cochlear fibrosis and ossification. TORCHES infections are congenital and early-onset causes of SNHL. Infectious causes may lead to symmetric or asymmetric losses.

Traumatic

Fractures of the temporal bone involving the otic capsule usually lead to profound hearing loss. Leakage of perilymph from the oval or round windows can cause progressive hearing loss and dizziness. Strong Valsalva during heavy lifting, head trauma, or barotrauma may initiate these perilymphatic fistulas.

Neurologic Disease

Multiple sclerosis is well known to cause a myriad of neurologic symptoms including hearing loss. ABR can be helpful in diagnosis, but MRI is more definitive. Cerebrovascular disease leading to brainstem stroke can also cause hearing loss, but usually multiple other neurologic symptoms will also be present.

◆ Evaluation

History

Pertinent history includes timing of onset, and whether abrupt or gradual, or fluctuating. Perceived severity and possible laterality are important. Associated symptoms and signs may be important, especially vertigo, visual disturbance, tinnitus, aural fullness, otalgia, or otorrhea. Past otologic history such as infection or surgery, a family history of hearing loss, and past exposures to noise or ototoxic agents is pertinent. A review of systems for associated neurologic or rheumatologic disease is sought.

Physical Exam

A thorough head and neck examination should be performed. The ear should be examined for any potential cause of CHL, such as cerumen, tympanic perforation, or middle ear effusions. Tuning fork tests may be done to document laterality and nature of hearing loss.

Imaging

Temporal bone CT is the single most useful test in children; specifically assess cochlea as well as vestibular aqueduct (enlarged vestibular aqueduct syndrome is defined as >1.5 mm anteroposterior [AP] diameter). To assess for retrocochlear pathology, MRI scanning of the head with fine cuts through the internal auditory canals should be performed with and without contrast media. Vestibular schwannomas and multiple sclerosis are typical diagnoses for which imaging studies should be obtained.

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Labs

There is controversy regarding laboratory testing. For pediatric hearing loss, one may consider urinalysis (for Alport and branchio-oto-renal syndrome), ECG (for Jervell-Lange-Nielson), RPR or FTA-ABS, toxoplasmosis, cytomegalovirus-related tests, thyroid function testing, ESR, antinuclear antibody testing, rheumatoid factor, liver function tests, CBC, referral for ophthalmology exam if retinitis pigementosa is suspected, Cx 26 gene testing. (Temporal bone CT is the single most useful test in children.)

In adult progressive or fluctuating hearing loss, consider 68-kD antibody testing, ESR, Lyme serology, and RPR.

Other Tests

Pure tone audiometry is the standard for documentation of hearing loss. Speech testing should also be performed. Patients may also be screened with otoacoustic emission testing and ABR testing. Hearing loss can be classified by its severity:

- Mild (15–30 dB)
- Moderate (30–50 dB)
- Moderate to severe (50–70 dB)
- Severe (70–90 dB)
- Profound (>90 dB)

◆ Treatment Options

Medical

Common forms of hearing loss (presbycusis, noise-induced hearing loss) can be treated with appropriate amplification. New hearing aid technologies include directional microphones, the capacity to filter background sounds, and many other programmable features. For symmetric hearing loss, bilateral aids are best. A CROS aid (contralateral routing of sound) may be used in severe unilateral loss to decrease the head-shadow effect.

Hearing loss associated with Ménière disease may respond to medical therapy with diuretics [Dyazide (GlaxoSmithKline, Brentford, Middlesex, UK) or Diamox (Lederle Laboratories, Philadelphia, PA)] or steroids (oral or possibly intratympanic).

Otoprotective treatment strategies are under study for concomitant use during known ototoxic drug treatments.

Surgical

Patients with profound bilateral loss that is not helped by hearing aids may be candidates for cochlear implantation (see Chapter 2.5.4). Patients with profound unilateral loss can be candidates for osteointegrated bone-anchored hearing devices (BAHAs; see Chapter 2.5.5).

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◆ Outcome and Follow-Up

Frequent adjustments and cleaning of hearing aids will ensure better patient compliance and satisfaction. Yearly audiograms should be performed on patients with identified SNHL to monitor for progression.

◆ ICD-9 Codes

389.10 Sensorineural hearing loss

Further Reading

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2.5.3 Hearing Aids

◆ Key Features

- A person with any degree of hearing loss has the potential to benefit from amplification.
- At this time all hearing aids should be digital and programmable.
- A variety of options are available for many different hearing losses.

Hearing aid technology has changed dramatically in the last few years. Although no hearing aid will restore hearing to normal abilities, most patients with hearing impairment can benefit from an appropriate aid.

◆ Epidemiology

About 5 million people in the United States wear hearing aids. However, only about one in five persons who could benefit from a hearing aid actually wears one. Seven years is the average time it takes someone with hearing loss to seek help.

◆ Clinical

Signs and Symptoms

If left untreated, hearing loss can have numerous negative effects, including:

- Anger, stress, depression, and anxiety
- Decreased interpersonal contacts and increased communication breakdowns

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- Social isolation and professional misunderstandings
- Misinterpretation of dementia
- Significant speech, language, and learning delays in children

◆ Evaluation

Physical Exam

Audiologic testing is done to determine the type, degree, and configuration of hearing loss to select the most appropriate hearing aids for an individual. As discussed in Chapter 2.4.1, certain red flags (**Table 2.11**) should prompt referral to the otolaryngologist. Indeed, most audiologists have a close relationship with the otolaryngologist and as a matter of routine obtain "medical clearance" prior to fitting hearing aids. Red flags for referral include conductive loss, asymmetric or unilateral loss, evidence of a sudden hearing loss, rapidly progressing or fluctuating sensorineural loss, word recognition score poorer than expected based on pure tone thresholds, word recognition score asymmetry greater than 10%, evidence of middle ear dysfunction, as well as evidence of otorrhea, or any hearing loss in a child. In children and other special populations assessment may need to include electrophysiologic testing in addition to behavioral testing. Other factors such as cost, lifestyle, listening needs, dexterity, cognition, and physical ear structure may need to be taken into consideration as well.

Other Tests

Hearing aid parameters are set based on prescriptive algorithms using the audiologic test information. However, these may need to be adjusted based on subjective patient preferences. Hearing aid function is assessed utilizing electroacoustic analysis in a 2-cc coupler. Real ear measurements are also valuable in determining individual differences in ears, such as with children's growing ear canal volumes.

Table 2.11 Indications for Otolaryngology Exam Prior to Hearing Aid Fitting*

Any hearing loss in a child
Conductive loss
Otorrhea
Evidence of middle ear dysfunction
Asymmetric or unilateral loss
Evidence of a sudden hearing loss
Word recognition score asymmetry >10%
Word recognition score poorer than expected based on pure tone thresholds
Rapidly progressing or fluctuating sensorineural loss

*One must avoid simply aiding an ear with significant underlying medical condition.

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◆ Treatment Options

Medical

The cost of pediatric hearing aids is covered by insurance. However, this is not true for most adults. Hearing aids can be divided into several sizes or styles, such as in-the-ear or behind-the-ear.

In general, the greater the degree of hearing loss, the larger the aid needs to be to provide adequate amplification. Depending on the technology in the hearing aid and the individual manufacturer, a wide variety of features may be available. This can include, but is not limited to, directional microphones, multiple programs, telecoils, feedback suppression, autoswitching, and noise management.

There are also specialized aids available. A CROS or BiCROS (contralateral routing of signal) hearing aid allows for sound to be sent from the side of an unusable ear to the other better-hearing ear. Transpositional hearing aids shift sound from higher frequencies where the hearing is no longer usable to lower frequencies with residual hearing.

FM systems used in conjunction with hearing aids can be valuable in adverse listening situations by improving the signal-to-noise ratio.

Surgical

For persons who no longer receive benefit from hearing aids due to the severity of their thresholds and/or reduced discrimination ability, cochlear implants may be an option. For persons with conductive hearing loss that have difficulty in utilizing traditional hearing aids, or in single-sided sensorineural deafness, a BAHA may be an option.

◆ Outcome and Follow-Up

Hearing aids do require routine maintenance to keep them dry and clean to continue functioning optimally. In some styles of aids, wax filters and ear mold tubing may need to be changed periodically. Battery life is only anticipated to be from a few days up to 2 weeks depending on the size of the battery, the style of the aid, and the amount of hearing loss. The average lifespan of a hearing aid is 5 to 7 years depending on the quality of care it receives. Hearing aids should fit comfortably for all-day wear and should not feedback unless covered in some way. Hearing aids are not likely to improve significantly poor speech discrimination; however, they may still be beneficial for sound awareness and in assisting with lip reading. Hearing aids need to be worn routinely for the wearer to receive the most benefit. Research has shown that persons with bilateral hearing loss perform best when aided binaurally. It takes time for the ears and brain to relearn how to use the sounds they have been missing. There are numerous other assistive devices available for persons who need help with their hearing loss beyond hearing aids (e.g., vibrating alarm clocks, light-connected doorbells).

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◆ ICD-9 Codes

389.00 Conductive hearing loss
389.10 Sensorineural hearing loss

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2.5.4 Cochlear Implants

◆ Key Features

- Cochlear implantation can restore the perception of sound to patients with profound hearing loss.
- Implants can be performed in patients as young as 12 months.
- Careful preoperative evaluation is vital for appropriate patient selection.
- Implants are more successful in younger patients and the postlingually deaf.

CIs have two main components: an internal, cochlear electrode array with receiver and external speech processor and transmitter. It allows for electrical stimulation of the cochlear nerve in patients with profound hearing loss. The external processor receives sound and converts it into electrical impulses. These impulses are sent transcutaneously to the receiver where they are routed to multiple electrodes within the cochlea. Electrical signals that reach the cochlea then depolarize cochlear nerve fibers, initiating the perception of sound. CIs should be approached as a team effort among surgeons, audiologists, and speech pathologists to ensure comprehensive care and maximize benefit to the patient.

◆ Clinical

Pediatric CI candidacy criteria include:

- Physiologic age ≥12 months
- Severe to profound bilateral SNHL

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- Expected benefit greater than that from an appropriate hearing aid
- Medical clearance to undergo general anesthetic
- Family support, proper motivation, and realistic expectations
- Rehabilitation/educational support for speech and language

Patients must be medically stable for surgery and have undergone audiologic testing. Counseling for appropriate expectations should always be done. There is some variation among surgeons regarding candidacy. FDA 2005 recommendations for adults include an SRT >70 dB with a word discrimination <50% with HINT (hearing in noise testing) as well as <40% discrimination in best-aided audiogram. Others summarize that audiologically adult patients must have severe to profound hearing loss, with poor performance using appropriate hearing aids. Pediatric patients can be tested with threshold ABRs and behavioral questionnaires filled out by parents. Patients have better performance with CIs when deafened postlingually.

◆ Evaluation

History

It is important to determine if the patient developed deafness pre- or postlingually, in terms of anticipated outcome. One must inquire about a prior history of meningitis, as this can lead to ossification of the cochlea, which may make electrode insertion impossible.

Physical Exam

Examine the tympanic membrane for perforation, retraction, or other signs of OM. Determine if there has been prior CWD mastoidectomy. It is more difficult to perform CIs on patients with chronic ear disease, but it is not a contraindication. However, active middle ear disease must be eradicated. If needed, a mastoid and middle ear obliteration with ear canal closure procedure can be performed initially to eradicate middle ear disease; the implant is then placed at a later date.

Imaging

High-resolution CT scan of the temporal bone should be obtained prior to all CIs. Anatomy to be assessed by CT: inner ear morphology, cochlear patency, the position of the facial nerve, the size of the facial recess, the height of the jugular bulb, and the thickness of parietal bone (especially in young children).

For full electrode array insertion, normal shape and turns should be seen in the cochlea. Congenital cochlear malformations should be carefully identified on imaging. Mondini malformation may not allow for a full insertion of electrodes, but this is not an absolute contraindication for implantation. Prior history of meningitis can indicate possible cochlear fibrosis and ossification, making electrode insertion difficult to impossible. MRI can play a role in evaluating candidates, especially children, for cochlear patency following meningitis, or for cochlear nerve aplasia. Imaging is also utilized to evaluate for a normal IAC diameter.

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Other Tests

Audiograms should be done with and without current and best-fitted hearing aids. Complete cochlear implant evaluation should be performed by a qualified audiologist.

◆ Treatment Options

Treatment of profound deafness with cochlear implantation is an excellent option for patients with appropriate expectations. However, lip-reading training and sign language are options for patients who are not candidates for medical or social reasons.

Typically, implantation is performed near a transmastoid approach. A simple mastoidectomy is performed and the vertical facial nerve is identified. The facial recess is then opened, allowing good visualization of the round window niche. A cochleostomy is performed near the round window, and the electrode array is then inserted via this opening. The receiver stimulator is typically secured to a shallow bony well developed posterior to the auricle, and the wound is closed. After a few weeks of healing, the external magnet can be placed, activated, and adjusted. Approximately 40 hours of therapy is to be expected.

◆ Complications

- The incision and flap complication rate is ~2–3% and is the most common problem.
- The risk of postoperative facial paralysis is 0.4%. Electrodes are delicate and can be damaged intraoperatively; the incidence of damaged or misplaced electrodes is 1.2%.
- In case of implant extrusion, local flaps to cover the implant can be employed. Explantation with replacement may be necessary if cochlear electrodes migrate out of the cochleostomy.
- In case of implant failure, explant and perform a reimplantation.
- Tinnitus and dizziness typically subside slowly after implantation.
- Scalp flaps may have to be thinned to ensure good magnet contact in obese patients.
- If meningitis develops, prompt treatment with IV antibiotics is required. If recurrent meningitis, consider hardware removal. (An older device associated with increased meningitis risk—Advanced Bionics Clarion with Positioner—has been removed from the market.)

◆ Outcome and Follow-Up

An AP skull x-ray can be obtained postoperatively to ensure appropriate placement of electrodes within the cochlea, although this is not typically necessary. Mastoid dressing should remain in place for 24 hours. Inspection of the wound should be performed to rule out hematoma formation. Facial nerve function should be documented. Once implanted, patients may not

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have monopolar electrocautery used during any subsequent surgeries. Caution should be used when considering MRI scans, as the receivers may need to have their internal magnets removed.

Vaccination for *Pneumococcus aureus* and *Haemophilus influenzae* is required, to help prevent possibility of meningitis. Postoperatively, aggressive antibiotic treatment for OM and other infections will help reduce risk of meningitis.

Mapping of the electrodes and programming can begin as early as 2 weeks postoperatively, but more commonly, patients begin programming 4 to 5 weeks postoperatively to ensure adequate wound healing. The external processor can be updated when the technology becomes available. Younger patients may develop excellent speech discrimination; however, prelingually deafened adults may only gain sound awareness and limited speech ability. Currently, data are accruing regarding the results of bilateral implantation, both simultaneous and sequential. Outcomes are very promising.

◆ ICD-9 Codes

389.10 Sensorineural hearing loss

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2.5.5 Other Implantable Hearing Devices

◆ Key Features

- Auditory brainstem implant (ABI)
- Bone-anchored hearing aid (BAHA)
- Middle ear implant (MEI)

For those patients with hearing loss who cannot benefit from CIs, there are other implantable devices available including auditory brainstem implants, bone-anchored hearing aids, and middle ear implants.

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◆ Epidemiology

Several hundred people worldwide have ABIs. More than 30,000 people worldwide use the BAHA system. A few MEI devices received FDA approval in the United States around 2000 to 2002; however, their use has not become widespread.

◆ Clinical

ABIs are for persons who cannot benefit from a CI because there has been damage to both auditory nerves (i.e., neurofibromatosis type II [NF2]). BAHAs are used in cases of conductive or mixed hearing loss where fitting of traditional hearing aids is not feasible (i.e., atresia, chronic drainage) and in single-sided deafness.

◆ Treatment Options

ABIs place electrodes on the cochlear nucleus at the base of the brain. This delivers input to the auditory pathway beyond the cochlea and the damaged auditory nerve.

The BAHA device is attached to the mastoid bone by an osseointegrated titanium screw. This allows the device to transmit sound directly to the cochlea through the skull, bypassing the damaged outer and/or middle ears. In the case of single-sided deafness, bone conduction transmits sound to the contralateral cochlea to achieve sound awareness from the deaf side.

MEIs are attached to the bones of the middle ear and move them directly as opposed to sending sound waves down the ear canal to the ear drum.

All of these implants require the use of external equipment to relay the sound information to the implanted device. The Envoy Esteem implant was approved by the FDA in March 2010 and does not require an external device.

◆ Complications

For ABIs, there are some MRI limitations once the device is implanted. With head trauma it is possible that any of these devices may be damaged. Although rare, it is also possible for there to be an internal device failure, usually due to the loss of a hermetic seal on the internal components.

◆ Outcome and Follow-Up

For ABIs a healing time of 4 to 6 weeks is generally required before the external equipment is used and the device is activated. ABIs provide increased sound awareness. Although this provides an improvement in the quality of their life, few recipients can understand speech without lip reading.

In adults, osseointegration for BAHAs takes ~3 months and in children 6 months before the external equipment is used. Routine cleaning of the surgery site is necessary to prevent infection. BAHA recipients typically report more comfort than other traditional forms of amplification. It also provides for sound awareness in single-sided deafness.

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2.6 Vertigo

2.6.1 Balance Assessment

◆ Key Features

- Electronystagmography (ENG)
- Rotational chair
- Dynamic posturography
- Vestibular evoked myogenic potentials

Balance involves three components: sensory input from the visual, vestibular, and proprioceptive systems; central nervous system integration of these sensory signals with the subsequent generation of appropriate motor commands; and adequate musculoskeletal abilities to perform the motor tasks. Proper assessment of balance/vertigo issues involves looking at all three of these components.

◆ Epidemiology

Dizziness is a common symptom in ~30% of people over the age of 65. Approximately 615,000 persons in the United States have been diagnosed with Ménière disease. Between 10 and 64 persons per 100,000 are affected by benign paroxysmal positional vertigo (BPPV) each year. Hundreds of thousands of hospital days are incurred every year in the United States due to vertiginous symptoms.

◆ Clinical

Signs and Symptoms

Persons with vestibular disorders report a variety of symptoms. They can include, but are not limited to, vertigo (i.e., spinning sensation), dis-

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equilibrium (i.e., subjective sense of falling), imbalance (i.e., observable unsteadiness), lightheadedness (i.e., feeling of faintness), oscillopsia (i.e., vision instability with head movement), nausea and vomiting, hearing loss, tinnitus, and sound sensitivity.

◆ Evaluation

Physical Exam

A full head and neck exam is performed. Include rapid head thrust testing in the horizontal plane to assess for catch-up saccades, Dix-Hallpike testing, cerebellar testing, gait assessment, Romberg testing, and full cranial nerve assessment.

Basic Balance Testing

ENG is reflective of the central and peripheral vestibular systems; specifically, the horizontal semicircular canal and the superior portion of the vestibular nerve. ENG is conducted by attaching electrodes above and below one eye, at the outside corner of each eye and on the forehead. Electrical potentials reflecting nystagmus are recorded while the person performs a series of activities including oculomotor activities (i.e., smooth pursuit tracking, saccade analysis, gaze fixation, spontaneous nystagmus, and optokinetic stimulation), positioning nystagmus (i.e., Dix-Hallpike maneuver), positional nystagmus (i.e., supine, head left and right, body left and right and 30-degree incline), and caloric irrigations (i.e., warm and cold water or air). In videonystagmography (VNG), video analysis of eye motion is conducted. VNG measures the movements of the eyes directly through infrared cameras, and may be more accurate, more consistent, and more comfortable for the patient than traditional ENG.

Rotational chair testing expands the evaluation of the peripheral vestibular system beyond the frequency and intensity limitations of the ENG evaluation. The phase, gain, and symmetry of the vestibular ocular reflex are assessed through recordings collected from electrodes placed as in ENG testing. The patient is then subjected to various rotations through sinusoidal harmonic acceleration tests and step tests with the head restrained to a chair with a computer-controlled motor in a darkened enclosure. Off-axis rotation provides ear-specific information and tests the utricle and superior vestibular nerve function. Standard rotational chair tests the horizontal semicircular canal function and stimulates the otoliths. Utricular function can specifically be assessed through subjective visual vertical or horizontal testing. The patient attempts to set an illuminated light to true vertical or horizontal in the absence of a visual reference and ambient light.

Dynamic posturography assesses the functional relative use of vision, vestibular, and somatosensory cues. Sensory organization is assessed by measuring a patient's ability to maintain balance while disrupting somatosensory and/or visual input. The patient is exposed to six conditions using a combination of normal, eyes closed, and the tilting of the support surface and/or the visual surround.

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Vestibular Evoked Myogenic Potentials

VEMPs are recorded by electrodes placed on the sternocleidomastoid muscle in response to click stimuli presented to the ipsilateral ear. This assesses saccular function and the integrity of the inferior division of the vestibular portion of CN VIII. Alterations in VEMPs responses may help confirm the diagnosis of superior canal dehiscence syndrome.

Other Tests

An audiologic evaluation should be conducted when the source of the balance disorder is suspected to be vestibular in origin.

Pathology

Balance disorders can be caused by problems of the peripheral vestibular system, the central vestibular system, systemic issues with parts of the body other than the head and brain, and vascular disorders. Peripheral vestibular system issues can be caused by Ménière disease, labyrinthitis, BPPV, perilymphatic fistula, vestibular neuritis (VN), ototoxicity, vestibular schwannomas, superior semicircular canal dehiscence (SCDS), autoimmune disease, and other disorders. Central vestibular system issues can be caused by brain tumors, multiple sclerosis, cerebrovascular disease including transient ischemic attack or stroke, stress, tension, fatigue, vision disturbances, and other disorders. Systemic issues can include peripheral neuropathies, hyperventilation, and dehydration. Vascular disorders can include orthostatic hypotension, arteriosclerosis, and vasovagal syndrome.

◆ Treatment Options

In cases of BPPV, canalith repositioning is highly effective. Vestibular and rehabilitation therapy may be useful for some balance problems. Ablation of the vestibular organ through vestibulotoxic injections or surgery may be appropriate in certain situations to allow for symptom reduction through compensation. In cases of SCDS or a perilymphatic fistula, surgery to repair the defect may alleviate the patient's symptoms.

◆ Complications

Vision problems and limitations in physical mobility can influence the ability to accurately conduct some components of ENG/VNG, rotational chair, and posturography testing. Results can also be influenced by a variety of medications and alcohol. VEMPs are not influenced by hearing loss. However, they can be impacted by the degree of muscle contraction.

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2.6.2 Benign Paroxysmal Positional Vertigo

◆ Key Features

- Transient, episodic vertigo is induced by head position changes.
- The Dix-Hallpike maneuver evokes geotropic rotatory nystagmus that fatigues the patient.
- Benign paroxysmal positional vertigo (BPPV) is most commonly caused by otoliths displaced into the posterior semicircular canal.
- BPPV is treated with the Epley repositioning maneuver, or other types of vestibular physical therapy.

BPPV is the most common form of vertigo reported by multidisciplinary balance centers. Displaced otoliths settle in the lowest part of the inner ear, the posterior semicircular canal. Head motion moves these crystalline masses, stimulating the neuroepithelium of the posterior semicircular canal. This causes patients to experience short-lived vertigo. Vertigo with geotropic rotary nystagmus elicited by the Dix-Hallpike maneuver is diagnostic and most cases are treated successfully with the Epley repositioning maneuver.

◆ Epidemiology

BPPV is the most common form of dizziness. Typically, it occurs spontaneously in elderly patients; in younger patients, BPPV often presents after head trauma or viral labyrinthitis. Symptoms can resolve without treatment.

◆ Clinical

Signs

Geotropic rotatory nystagmus is noted when a patient is placed in the Dix-Hallpike position. There may be a short latency (delay) in the onset of nystagmus, and repeated maneuvers lead to fewer symptoms and less nystagmus (fatiguing).

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Symptoms

The patient experiences a spinning sensation that lasts less than a minute, which is associated with head movement, most often when bending forward, looking upward, or lying down and rolling from one side to another. Patients may also have mild, generalized imbalance.

Differential Diagnosis

Most diagnoses that cause nonrecurrent or persistent vertigo can be eliminated based on history alone. Processes causing sudden and recurrent vertigo can be broadly differentiated by duration of symptoms: minutes versus hours. Conditions associated with a long duration of symptoms include Ménière disease, autoimmune inner ear disease, and vestibular neuritis (VN). Conditions associated with a short duration of symptoms include BPPV, perilymph fistula, superior canal dehiscence syndrome, labyrinthine fistula, and migrainous vestibulopathy.

◆ Evaluation

Physical Exam

A full neurotologic exam should be performed. A fistula test and the Dix-Hallpike maneuver should be completed. Geotropic rotatory nystagmus is elicited with the Dix-Hallpike maneuver and is fatiguing: this implicates the downward facing ear as the affected side.

Imaging

Patients with a positive Dix-Hallpike test do not typically require imaging.

Labs

Laboratory evaluation is not useful in the diagnosis of BPPV.

Other Tests

Atypical cases of vertigo may be evaluated with ENG/VNG and MRI with gadolinium to rule out other vestibular pathology. Audiologic testing should also be obtained if a condition other than BPPV is thought possible.

Pathology

Cadaveric temporal bone dissections and labyrinthine operations performed in patients with symptoms of BPPV have demonstrated the presence of small calcium crystals (thought to be displaced otoliths from the saccule or utricle) within the posterior semicircular canal. Changing head position causes movement of otoliths within the canal inducing motion of the cupula. There has also been speculation that cupulolithiasis (otolithic crystals trapped on the cupula) plays a role in some cases.

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◆ Treatment Options

Medical

Conservative measures include avoiding fast movements of the head and other aggravating motions, i.e., looking up while turning, lying flat. The Epley repositioning maneuver is the mainstay of treatment with success rates exceeding 90% (**Fig. 2.12**). This maneuver entails performing a Dix-Hallpike maneuver to elicit vertigo. Following cessation of vertigo, the patient's head

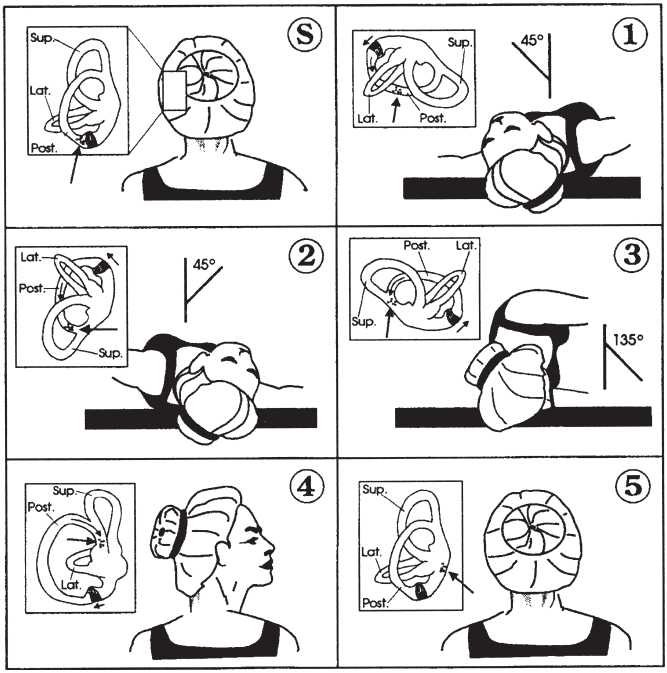

Fig. 2.12 The Epley repositioning maneuver. Positioning sequence for left posterior semicircular canal, as viewed by operator (behind patient). The inset boxes show exposed views of labyrinth, with migration of particles (large arrows). S, Start, patient seated. 1. Place head over end of table, 45 degrees to the left. 2. Keeping head tilted downward, rotate 45 degrees to the right. 3. Rotate head and body until facing downward 135 degrees from supine. 4. Keeping head turned right, bring patient to a sitting position. 5. Turn head forward, chin down 20 degrees. Pause at each position until induced nystagmus approaches termination, or for T (latency + duration) seconds if no nystagmus. Keep repeating entire series (1 through 5) until there is no nystagmus in any position. (From Epley JM. Particle repositioning for benign paroxysmal positional vertigo. Otolaryngol Clin North Am 1996;29:327. Reprinted with permission.)

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is rotated 90 degrees to the unaffected side. After 45 to 60 seconds, the head is then turned a further 90 degrees by having the patient lie on his or her side, such that the face is toward the floor. The patient is then brought back to the upright position with his or her chin tucked to the chest. Each of these positions can be kept for approximately one minute. The canaliths are ultimately repositioned in the vestibule away from sensory epithelium. This procedure may be repeated as needed for relapses or refractory cases. Following the Epley, patients are instructed to not lie flat or assume any provoking position for the next week. Vestibular rehabilitation may help for longer-term imbalance. Brandt-Daroff exercises provide a home alternative for relapsing cases, or for patients who cannot tolerate an Epley maneuver in the office.

Surgical

Two surgical procedures can be performed for refractory cases of BPPV: singular neurectomy and posterior canal occlusion. Singular neurectomy entails sectioning the singular nerve as it courses from the ampulla of the posterior canal to join the utricular nerve to form the inferior vestibular nerve. This procedure is technically difficult, carries a high risk of postoperative SNHL, and is not often performed. Posterior canal occlusion involves identifying the posterior canal via a transmastoid approach and packing the canal to create a barrier preventing the flow of endolymph and stimulation of neuroepithelium.

◆ Outcome and Follow-Up

Following the Epley maneuver, patients are instructed to not lie flat or assume any provoking position for the next week, to allow canaliths to settle. Patients with persistent symptoms may have the Epley maneuver repeated, or may benefit from Brandt-Daroff exercises or treatment with a trained vestibular physical therapist.

◆ ICD-9 Code

386.11 Benign paroxysmal positional vertigo

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2.6.3 Ménière Disease

◆ Key Features

- Recurrent episodic vertigo and hearing loss may last minutes to hours.
- Ménière disease is caused by excess endolymph within the labyrinth.
- Conservative treatment with diuretics and sodium restriction are often effective.
- Multiple treatment options are available for disabling vertigo.

Ménière disease, or endolymphatic hydrops, is a common cause of recurrent vertigo. Symptoms classically consist of unilateral pressure and tinnitus in the affected ear with associated vertigo and hearing loss in the low frequencies. If both ears are affected simultaneously, autoimmune inner ear disease should be suspected. Diagnosis is based on history and confirmed with an audiogram showing low frequency hearing loss and ECochG demonstrating an elevated SP/AP ratio. Treatment is tailored to the severity of disease and the level of hearing loss, the spectrum ranging from conservative dietary modifications to destructive surgical procedures.

◆ Epidemiology

Ménière disease has a slight female predominance. Age of onset usually is around 40 years.

◆ Clinical

Signs and Symptoms

The classic triad of symptoms of Ménière disease includes unilateral tinnitus, fluctuating hearing loss, and vertigo. Many patients also note a sensation of fullness in the affected ear, which may get worse immediately prior to an attack. Vertigo attacks can be variable in their frequency and typically will last several hours. Often they are associated with nausea and vomiting, and may be followed by severe fatigue.

Differential Diagnosis

The differential diagnosis should include all processes causing recurrent episodic vertigo: BPPV, superior semicircular canal dehiscence (SCDS) syndrome, perilymph fistula, autoimmune inner ear disease, vestibular schwannoma, endolymphatic sac neoplasm, other intracranial neoplasm, migraine-related vestibulopathy, and transient ischemic attacks.

The American Academy of Otolaryngology–Head and Neck Surgery has issued diagnostic guidelines, summarized in **Table 2.12**.

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Table 2.12 American Academy of Otolaryngology–Head and Neck Foundation Diagnostic Guidelines for Ménière Disease

Possible Ménière disease
Episodic vertigo of the Ménière type without documented hearing loss, or sensorineural hearing loss, fluctuating or fixed, with dysequilibrium but without definitive episodes
Other causes excluded
Probable Ménière disease
One definitive episode of vertigo
Audiometrically documented hearing loss on at least one occasion
Tinnitus or aural fullness in the treated ear
Other causes excluded
Definite Ménière disease
Two or more definitive spontaneous episodes of vertigo 20 minutes or longer
Audiometrically documented hearing loss on at least one occasion
Tinnitus or aural fullness in the treated ear
Other causes excluded
Certain Ménière disease
Definite Ménière disease, plus histopathologic confirmation

Source: American Academy of Otolaryngology–Head and Neck Foundation, Inc. Guidelines for the diagnosis and evaluation of therapy in Meniere's disease. Otolaryngol Head Neck Surg 1995;113(3):181–185. Reprinted with permission.

◆ Evaluation

Physical Exam

A complete neurotologic examination should be performed. A physical examination may demonstrate the presence of low-frequency hearing loss via tuning fork exam. Nystagmus may be present during an acute attack. A Dix-Hallpike maneuver and fistula test should be performed to rule out BPPV, SCCD, or a labyrinthine fistula. Most patients with Ménière disease will have a normal exam at the time of an office visit.

Imaging

Patients with unilateral hearing loss and vertigo should have an MRI with contrast of the brain including internal auditory canals to rule out acoustic neuroma or other retrocochlear pathology.

Labs

Autoimmune serology may be considered for patients exhibiting symptoms of bilateral Ménière disease.

Other Tests

Audiograms are routinely used in the diagnosis and monitoring of Ménière disease. Audiograms classically reveal hearing loss that is more severe in

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the low frequencies. Fluctuation may occur rapidly during acute episodes. ECoG may be used to confirm the diagnosis where a summating potential to action potential greater than 0.5 is arguably pathognomonic. A VNG may help by demonstrating a significant vestibular weakness in the same ear with the SNHL. This is essential prior to ablative management.

Pathology

Ménière disease is hypothesized to be caused by excess endolymph, which causes dilation of the stria vascularis and resultant ischemia. Ischemia then results in weakness in the walls of the scala media causing rupture and mixing of endolymph with perilymph. Cadaveric temporal bones studies on patients with Ménière disease demonstrate these findings.

◆ Treatment Options

Medical

Conservative approaches are initially trialed in the treatment of Ménière disease and include instituting a low-salt diet (<1500 mg of sodium daily) and potassium-sparing diuretics (Dyazide 25 mg daily). Systemic steroids may be used short term to control acute attacks (Medrol Dose Pack; Pfizer Pharmaceuticals, New York, NY).

Intratympanic gentamicin injection to medically ablate the labyrinth (40 mg/cc of buffered gentamicin solution) is a highly effective alternative to surgical ablation in patients with Ménière disease who have debilitating vertigo. Gentamicin is toxic to dark cells as well as hair cells within the inner ear, but preferentially affects vestibular cells. Hearing loss may occur in as many as 15% of patients treated in this way. Vestibular training exercises may be helpful to improve balance following medical labyrinthectomy.

Intratympanic perfusion of steroids has also been employed in the management of Ménière disease. As with most nonablative Ménière treatments, efficacy data has been difficult to establish. Although vertigo control may not be as good as it is with gentamicin, patients may report a general improvement in aural symptoms, and they are at no risk for additional hearing loss. In fact, patients may demonstrate improved hearing thresholds following intratympanic steroid treatment. There is little risk to an intratympanic administration of 0.5 mL dexamethasone 4 mg/mL, and some have reported giving this via a tympanostomy tube to facilitate ease of retreatment. A series of treatments every 3 to 4 weeks, up to five treatments, has been advocated, if needed, based on response.

Surgical

Refractory Ménière disease may require more invasive treatments predicated on degree of residual hearing. Surgical procedures may be broadly grouped into hearing conservation (endolymphatic sac decompression, vestibular nerve sectioning) or hearing destruction (labyrinthectomy). Endolymphatic sac surgery is performed via a transmastoid approach where the sac is identified and subsequently either decompressed or shunted into

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the mastoid cavity or arachnoid space. Vestibular nerve sectioning may be performed via a middle cranial fossa, retrosigmoid or retrolabyrinthine approach. Labyrinthectomy may be performed via a transmastoid approach, but results in hearing destruction. Of note, vestibular nerve section and/or labyrinthectomy (medical or surgical) cannot be done bilaterally, as the patient will experience debilitating profound vestibular hypofunction.

◆ Outcome and Follow-Up

Efficacy data are generally limited for most treatments. The natural history of the disease appears to involve a gradual resolution of active symptoms in most patients over 5 years. Patients are generally followed clinically as needed. Serial audiometry to monitor stability of hearing is helpful, especially in patients with substantial fluctuation, and in patients who have been treated with aminoglycosides.

◆ ICD-9 Codes

386.01 Ménière disease, cochleovestibular
386.03 Ménière disease, vestibular
386.9 Unspecified vertigo

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2.6.4 Vestibular Neuritis

◆ Key Features

- Severe vertigo lasts days to weeks.
- VN must be distinguished from acute intracranial pathology.
- Recovery may take weeks.

VN is a fairly common cause of acute onset nonrecurrent vertigo. The vertiginous episode is often preceded by viral upper respiratory tract infection. Inflammation of the vestibular nerve results in hypofunction of the affected side and causes vertigo. VN typically has no associated hearing loss.

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◆ Epidemiology

Vestibular neuritis primarily affects middle age to the elderly, but can occur at any age. No gender predominance has been noted.

◆ Clinical

Signs and Symptoms

Some cases of VN may be preceded by upper respiratory infection symptoms. Severe nausea and vomiting with dehydration may warrant hospital admission. Spontaneous nystagmus is usually present and can be more easily noted by using Frenzel glasses. Episodes usually last several days to weeks. Full compensation from a severe vestibular loss can sometimes take months. Interestingly, it is not unusual for some patients to develop BPPV several months after their VN symptoms resolve.

Differential Diagnosis

The differential diagnosis includes pathologies of both central and peripheral etiology. Cerebellar or brainstem infarction can cause sudden-onset vertigo. Peripheral causes of sudden-onset vertigo include perilymph fistula, Ménière disease, BPPV, or lesions of the cerebellopontine angle. Patients who in addition have fever and hearing loss must be suspected of having bacterial labyrinthitis.

◆ Evaluation

Physical Exam

A complete neurotologic exam should be performed. Nystagmus is generally present, directed away from the affected ear and is suppressible. Due to a hypofunctioning labyrinth, patients will fall to the affected side on Romberg testing. Patients will often present their heads tilted to one side, which often lessens their symptoms. A head thrust test will often be abnormal and indicates loss of the vestibuloocular reflex.

Imaging

Imaging is warranted to rule out intracranial infarction or hemorrhage or other retrocochlear pathology. MRI is the preferred study to interpret infarction, but CT can be used initially to rule out hemorrhage.

Labs

Lyme disease titers may be of value depending on geographic location.

Other Tests

VNG may be performed to confirm the unilateral vestibular weakness on the affected side. An audiogram should also be performed to determine baseline hearing and assess for SNHL.

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Pathology

No conclusive evidence of the exact etiology of VN exists. Cadaveric temporal bones have demonstrated inflammation and degeneration of primarily the superior vestibular nerve and Scarpa's ganglion. Herpes virus has been demonstrated as a cause in some cases.

◆ Treatment Options

See **Table 2.13** for management options. As a rule, VN is self-resolving. The vertigo induced by the hypofunctioning vestibular nerve is gradually compensated for both centrally and by the normally functioning contralateral nerve. Admission for severe vertigo and dehydration from persistent vomiting is sometimes required for rehydration and IV medication administration. Corticosteroids can be administered in the fashion of a Medrol Dose Pack or an IV taper (Decadron 10 mg; Merck & Co., Inc., Whitehouse Station, NJ), and may improve long-term recovery. Acyclovir or valacyclovir (Valtrex; GlaxoSmithKline, Brentford, Middlesex, UK) may be utilized due to the association with herpes virus, but their efficacy is yet to be determined. Nausea may be symptomatically treated most effectively with vestibular suppressants: promethazine (25 mg parenterally—intramuscular or rectal suppository preferred; an IV route has been discouraged due to severe reactions from inadvertent intraarterial infusion), meclizine (25 mg orally [PO]), or benzodiazepines such as diazepam (5 mg PO). Patients should be cautioned about using vestibular suppressants for long periods, as they can slow down central compensation. Patients should be encouraged to perform home vestibular exercises. Vestibular rehabilitation can be helpful for patients with slow compensation.

Table 2.13 Management Options for Vestibular Neuritis

Vestibular suppressants
Promethazine (Phenergan) 25 mg IM, PR, PO every 6 hours as needed
Diazepam (Valium) 2–10 mg IM, IV, PO two to three times daily as needed
Clonazepam (Klonopin) 0.5–1 mg PO three times daily as needed
IV hydration
Lactated Ringer's solution bolus or maintenance infusion
Steroids
Consider taper, i.e., methylprednisolone (Medrol) dose pack 4-mg tabs PO, start 24 mg/day and taper by 4 mg daily over 6 days
Consider antivirals
Acyclovir 800 mg PO 5x daily × 1 week
Valacyclovir (Valtrex) 1000 mg PO twice daily × 1 week
Vestibular physical therapy

Abbreviations: IM, intramuscularly; IV, intravenously; PR, per rectum; PO, orally.

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◆ Outcomes and Follow-Up

Most patients never have recurrence of symptoms. Unsteadiness and dizziness may be experienced for months after an event. Some patients may go on to develop BPPV months later.

◆ ICD-9 Code

386.12 Vestibular neuritis

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2.6.5 Migraine-Associated Vertigo

◆ Key Features

- Over a quarter of patients with migraine will report vestibular symptoms.
- Pathophysiology is poorly understood.
- Many patients do not experience headache in conjunction with attacks.
- Management focuses on treatment and prevention of migrainous attacks.

Migraine is among the most common central disorders that produce vestibular symptoms ranging from head motion intolerance to spontaneous vertigo. Though the association between migraine and vertigo has been well documented, a causal relationship has not been elucidated. Diagnosis is based largely upon the migraine criteria established by the International Headache Society (IHS) and recently developed criteria specifically for migraine-associated vertigo. Treatment is aimed at controlling the frequency and severity of migrainous attacks.

◆ Epidemiology

Migraine is a common disorder that affects 18 to 29% of women and 6 to 14% of men. Symptoms usually begin in the third to fourth decade and tend

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to recur. Most patients with migraine have a family history. More than a quarter of patients with migraine have attacks of vertigo, compared with only 7% of patients with tension headaches.

◆ Clinical

Signs and Symptoms

The typical migraine headache is unilateral, throbbing, and moderate to severe, exacerbated by physical activity and accompanied by sensitivity to light and noise. Migraines are classified as either with or without aura. Several vestibular symptoms can accompany migraine including dizziness, head motion intolerance, and spontaneous vertigo. The attacks of vertigo can last minutes to hours. Most patients are symptom-free between attacks, which can occur several times a month. Some patients will also experience other neurotologic symptoms such as aural fullness. For approximately half of all patients with migraine-associated vertigo, the attacks of vertigo are not temporally associated with headache symptoms. In some women, there is an association with menstruation, i.e., perimenstrual migraine with vertigo.

Differential Diagnosis

Other causes of vertigo must be considered in patients in whom vestibular symptoms are thought to be associated with migraine. These include peripheral conditions that cause episodic vertigo such as benign paroxysmal positional vertigo and Ménière disease. Other central causes of vestibulopathy, such as vertebrobasilar insufficiency, hydrocephalus, and multiple sclerosis should also be considered, as well as neoplastic diseases and infection.

◆ Evaluation

History

A thorough history focusing on migrainous symptoms is crucial. The IHS criteria for migraines are useful in diagnosis. In addition, a family history of migraine should also be sought. The IHS criteria for migraines are useful in diagnosis (**Table 2.14**). The proposed criteria for migraine-associated vertigo as noted below can be helpful in identifying patients.

1. Episodic vestibular symptoms of moderate severity
2. Migraine according to IHS criteria
3. At least one of the following migrainous symptoms during at least two vertiginous attacks: migrainous headache, photophobia, phonophobia, visual or other auras
4. Other causes ruled out by appropriate investigation

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Table 2.14 International Headache Society (IHS) Diagnostic Criteria for Migraines

Recurrent headaches separated by symptom-free intervals and accompanied by any three of the following:
• Abdominal pain
• Complete relief after sleep
• Nausea and/or vomiting
• Aura
• Hemicrania
• Throbbing, pulsatile quality
• Family history of migraine

Data from International Headache Society. The International Classification of Headache Disorders. 2nd ed. Cephalalgia 2004;24(Suppl 1).

In women, one should inquire about the possibility of perimenstrual migraine with vertigo. Up to 50% of patients will not experience headaches during, or in a consistent temporal relationship to, vertigo symptoms.

Physical Exam

The physical exam in patients with migraine-associated vertigo is usually normal. However, thorough head and neck examinations including neurologic and vestibular exams should be completed to exclude other causes of vertigo.

Other Tests

Vestibular testing can be helpful in separating peripheral from central causes of vertigo. Patients with migrainous vertigo usually have normal ENG tracings, but may have some abnormalities on a rotary chair test. Complete audiologic assessment should be obtained.

Imaging

MRI and CT of the brain are usually normal in cases of migraine-associated vertigo, but are useful in excluding other serious neurologic conditions. There is often considerable value in offering patients reassurance that MRI results are normal.

◆ Treatment Options

Treatment for migraine-associated vertigo centers on control of migrainous attacks by prophylactic or abortive medical management (**Table 2.15**). Prophylactic treatment includes the avoidance of dietary triggers, β-blockers (propranolol 40 mg twice daily), calcium channel blockers (verapamil 80 mg three times daily), and antidepressants. Triptans (sumatriptan 25 mg PO at onset of symptoms) and analgesics (ibuprofen 600 mg) can be used to abort or reduce symptoms when they do occur. Few studies have evaluated the effects of these management strategies on vestibular symptoms; however, there is

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Table 2.15 Management Strategies for Migraine-Associated Vertigo

Prophylaxis	Treatments to Abort Acute Attack
Avoidance of dietary triggers (e.g., aged cheeses, chocolate, beer, red wine, excessive caffeine, aspartame, monosodium glutamate, high salt)	Nonsteroidal antiinflammatories: Ibuprofen 600 mg PO at onset
Beta-blockers: Propranolol 40 mg PO twice daily	For perimenstrual migraine with vertigo: Clonazepam (Klonopin) 0.5–1 mg PO every 8 hours as needed
Calcium channel blockers: Verapamil 80 mg three times daily	Triptans: Sumatriptan (Imitrex) 25–100 mg PO at onset of symptoms, or 4–6 mg SC × 1, or 5 or 20 mg nasal spray × 1 Rizatriptan (Maxalt) 5–10 mg PO × 1 Naratriptan (Amerge) 1–2.5 mg PO × 1 Others
Tricyclic antidepressants: Amitriptyline 25–150 mg PO qhs, Nortriptyline (Pamelor) 25–150 mg PO qhs, Protriptyline (Vivactil) 5–10 mg PO three times daily	Combination: (Treximet 1 tab PO × 1) single-tablet combination of sumatriptan and naproxen sodium

Abbreviations: PO, orally; qhs, before bedtime; SC, subcutaneously.

some evidence that the same prophylactic drugs can be effective. For acute vertiginous episodes, sumatriptan is helpful in some patients, but only for attacks lasting longer than one hour. Patients who develop chronic disequilibrium may also benefit from vestibular rehabilitation therapy. Patients with perimenstrual migraine with vertigo often respond well to clonazepam (Klonopin; Roche Pharmaceuticals, Nutley, NJ) 0.5–1 mg PO every 8 hours as needed.

◆ Outcome and Follow-Up

Avoidance of identifiable triggers is highly successful. If pharmacotherapy is needed, the majority of patients respond favorably to prophylactic medications or triptans.

◆ ICD-9 Codes

346.2 Variants of migraine

386 Vertiginous syndromes and other disorders of the vestibular system

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2.7 Tinnitus

◆ Key Features

- Tinnitus is the perception of sound with no acoustic source external to the head.
- Retrocochlear pathology in unilateral tinnitus must be excluded.
- Vascular anomalies and neoplasms should be excluded in pulsatile tinnitus.

Tinnitus is a common but poorly understood disorder. Its severity can range from insignificant to disabling. Tinnitus is a nonspecific symptom characterized by the sensation of buzzing, ringing, clicking, pulsations, and other noises in the ear. Objective tinnitus, or somatosounds, refers to noises generated from within the ear or adjacent structures. The term "subjective tinnitus" is used when the sound is audible only to the affected patient.

◆ Epidemiology

Fifty million Americans report tinnitus; 12 million seek medical attention. Males report it more frequently than females.

◆ Clinical

History

A thorough audiological history, including infections, surgery, trauma, and family history should be obtained. Review of the patients' current medication list may reveal ototoxic or tinnitogenic medications. Management of tinnitus will benefit from determining the quality of life related to the symptom (memory, sleep, productivity), laterality, and onset sequence.

Differential Diagnosis

Subjective (Nonpulsatile)

Otologic: hearing loss, otosclerosis, Ménière disease
Neurologic: multiple sclerosis, trauma, vestibular schwannoma

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Infectious: Lyme disease, syphilis, otitis, meningitis

Drug-induced: aspirin, nonsteroidal antiinflammatory drugs (NSAIDs), aminoglycoside, furosemide, vincristine, platens

Miscellaneous: TMJ or myofascial, dental, depression, idiopathic

Objective (Pulsatile)

Turbulent flow: glomus tumors, carotid atherosclerosis, benign intracranial hypertension, dural arteriovenous malformations, cerebral aneurysms

Muscle contractions: palatal myoclonus—stapedial, tensor tympani

Spontaneous otoacoustic emissions

◆ Evaluation

Physical Exam

A complete neurotologic exam should be performed. Attention should be paid to the middle ear and CNs V, VII, and VIII. In an oral cavity exam, check for palatal myoclonus and TMJ disease; examine for myofascial etiology. Auscultation of the neck and temporal bone may reveal bruits. Occasionally, the tinnitus may be correlated to the cardiac rhythm with a variety of maneuvers.

Imaging

MRI should be performed in appropriate patients with unilateral tinnitus. CT angiography or MRA is useful for pulsatile tinnitus.

Labs

Order Lyme or syphilis titers if history is suggestive.

Other Tests

Perform an audiogram to determine if there is any associated hearing loss. Tinnitus matching can be performed to quantify intensity and frequency of tinnitus. ABR can be done to assess for retrocochlear pathology.

Pathology

Abnormalities causing tinnitus can occur in any part of the auditory system. Current research using neural imaging supports a theory that subjective tinnitus originates in the central auditory system as opposed to the cochlea.

◆ Treatment Options

The management strategy may benefit from addressing the impact of tinnitus on the quality of life.

Medical

Reassurance, patient education, noise precautions in work or recreation environment, amplification, masking device, noise generator, and tinnitus retraining therapy are often helpful. Complementary and alternative medi-

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cine treatments such as biofeedback, electrostimulation, and nutritional supplements (zinc, vitamin B_{12}) may also help; see Chapter 1.8.8.

Antidepressants: Elavil, gabapentin. Melatonin has been shown in blinded studies to improve tinnitus in patients with trouble sleeping attributed to the tinnitus. Discontinue ototoxic medications (aspirin, NSAIDs, diuretics, chemotherapeutics, quinine).

Surgical

Where appropriate, surgery can be considered for some objective sources of tinnitus, e.g., to treat the underlying lesion.

◆ Outcome and Follow-Up

Predefine objectives of management together. Healthcare professionals have the opportunity to help tinnitus patients learn how to cope with it, how to manage it, and how to treat their tinnitus.

◆ ICD-9 Code

388.3 Tinnitus

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2.8 Cerebellopontine Angle Tumors

◆ Key Features

- The most common tumors are vestibular schwannomas and meningiomas, less commonly epidermoids.
- Treatment options include observation, stereotactic radiosurgery, and surgical excision.
- Surgical approaches are middle fossa, retrosigmoid, and translabyrinthine.

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The cerebellopontine angle (CPA) is a CSF-filled space found at the ventral aspect of the junction between the cerebellum and the pons. Most neoplasms found in this location are benign and are treated similarly, with vestibular schwannomas (also known as acoustic neuromas) and meningiomas being most frequent. Presenting symptoms usually include unilateral progressive SNHL, high-pitched tinnitus, and disequilibrium.

◆ Epidemiology

Vestibular schwannomas are the most common CPA neoplasm. These most commonly arise from the superior vestibular division of CN VIII. Spontaneous yearly occurrence is ~1 in 100,000; roughly 2280 new cases annually in the United States. Most tumors grow slowly (average is 1–2 mm per year). Bilateral tumors can be seen in patients with NF2.

◆ Clinical

Signs and Symptoms

Typical initial symptoms are unilateral hearing loss, unilateral tinnitus, or progressive imbalance or vertigo. Symptoms correlate with tumor size, generally. Large tumors can cause facial weakness, facial numbness, and brainstem compression. Compression of the fourth ventricle may cause hydrocephalus, typically with tumor size >4 cm. Sudden hearing loss may be the presentation in ~10% of cases. Difficulty talking on the phone is a common complaint. Brainstem compressive symptoms with very large tumors are ataxia, headache, nausea, vomiting, diplopia, cerebellar signs, lower cranial nerve palsies (i.e., hoarseness), respiratory depression, and coma.

Differential Diagnosis

The differential diagnosis may include vestibular schwannoma, meningioma, epidermoid, arachnoid cysts, facial nerve schwannoma, trigeminal schwannoma, and metastatic tumor.

◆ Evaluation

Physical Exam

A complete neurotologic exam should be performed. Cranial nerve examination should be performed. Trigeminal, trochlear, and lower CNs may all be involved in large tumors. Check the corneal reflex. A head-shake test may induce brief nystagmus if unilateral vestibular weakness is present.

Imaging

MRI of the head/brain with contrast along with special attention to the internal auditory canals is the gold standard for diagnosis. Vestibular schwannomas and meningiomas are isointense on T1 and T2, while enhancing with contrast on T1. Meningiomas are more sessile, and may display a dural tail. They are rarely centered within the IAC.

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Other Tests

- Audiogram: Unilateral pure tone loss is usually seen. Decreases in speech discrimination are common and usually greater than expected considering pure tones.
- ABR test: There is an approximate 95% sensitivity in detecting compressive tumors. Typically, an increased inter-aural difference in wave V is seen with vestibular schwannoma.
- ENG: Small tumors may cause caloric weakness alone, larger tumors may cause abnormalities in the ocular portions of ENG testing if there is brainstem compression.

Pathology

Vestibular schwannomas demonstrate two typical histologic architectures. Elongated spindles are predominant in either pattern: Antoni A pattern shows dense nests of cells while Antoni B is interwoven and less densely packed.

◆ Treatment Options

See **Table 2.16** for treatment options for a vestibular schwannomas. Tumor size, growth rate, severity of symptoms, hearing levels, anesthetic risk, patient age, and other factors must be evaluated for the individual patient to determine the best option for treatment. The average growth rate of a vestibular schwannoma is ~1 to 2 mm per year, although this can vary. Most tumors display some growth over 3 years. Serial MRI scans every 6 to 12 months can monitor growth rate. Radiotherapy or excision may be required if tumor progresses. NF2 patients require individualized evaluation and treatment planning.

Gamma knife stereotactic radiosurgery can be used to irradiate tumor with less radiation exposure to surrounding tissues than external beam radiation. Typically, facial nerve function is preserved; however, a slow, progressive hearing loss may be seen due to radiation effects on the cochlea or

Table 2.16 Management of Non-Neurofibromatosis Type 2 Vestibular Schwannomas (Acoustic Neuromas)

Tumor Size	Options

Small (≤2 cm diameter), noncystic	• Observation
	  ○ MRI every 6–12 mo until stable, then annually
	  ○ Annual audiometry
	  ○ Treat if >2 mm interval growth
	• Radiotherapy
	• Surgery
	  ○ Translabyrinthine approach if no useful hearing
	  ○ Suboccipital or middle fossa approach if good discrimination score

Large (>2 cm diameter)	• Treat (consider age comorbidities to determine best modality)

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nerve. Continued monitoring for growth is recommended, as some tumors may progress despite radiation. Generally, tumors greater than 2 to 3 cm are not candidates for stereotactic radiosurgery, although the indications for stereotactic radiosurgery may be expanding.

There are three typical surgical approaches: middle fossa, retrosigmoid, and translabyrinthine (**Fig. 2.13**). The preference and experience of the surgeon will factor into the decision regarding the approach.

- The middle fossa approach is appropriate for tumors less than 1.5 cm (mostly intracanalicular). Hearing preservation is possible, and the entire IAC can be accessed. Most commonly used for small, lateral tumors.
- The retrosigmoid (suboccipital) approach can be used for hearing preservation for tumors up to 2 cm. It can also be used for large tumors, without hearing preservation, but only the medial two thirds of the IAC is exposed, and some lateral tumor must be dissected blindly if hearing is to be preserved. Some tumor may theoretically be left behind.
- The translabyrinthine approach can be used for any size tumor, but has no chance of hearing preservation. It has the best exposure of the facial nerve and is the most commonly used approach. It also requires very little if any brain retraction.

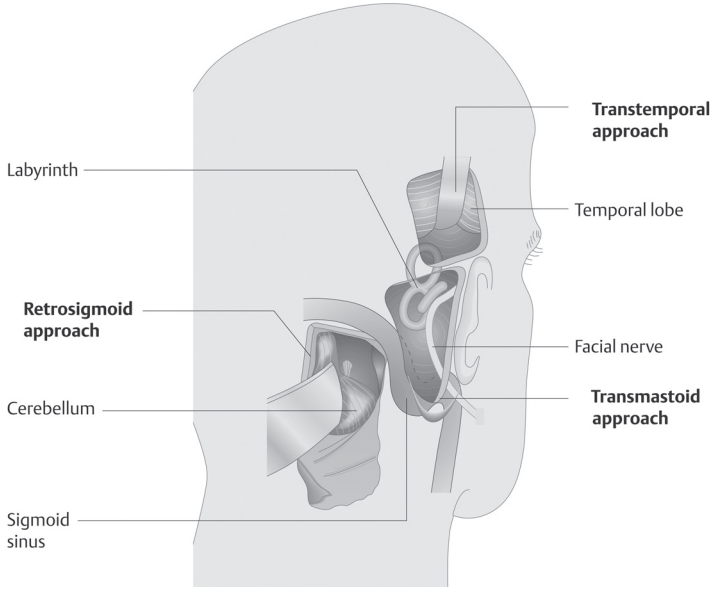

Fig. 2.13 Surgical approaches to the lateral skull base. **(A)** The retrosigmoid approach is intracranial and intradural. **(B)** The transtemporal approach is intracranial and extradural. **(C)** The transmastoid approach is extracranial and extradural. From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:301.)

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NF2 patients with bilateral schwannomas require special consideration and sometimes aggressive management to avoid eventual bilateral deafness. Enlarging tumors may impair function of the facial and cochlear nerves bilaterally. Unlike most sporadic acoustic tumors, NF2 tumors often infiltrate nerve fibers, rather than displace them. Managing multiple cranial neuropathies can be challenging; lower cranial nerve involvement is common. Meningiomas have a slightly better chance of hearing preservation.

Facial nerve neuromas often require a combined middle fossa and transmastoid approach to resect but can be approached translabyrinthine if hearing is not serviceable.

◆ Complications

See **Table 2.17** for timetable and description of possible complications, as well as management issues.

Initial postoperative facial weakness should be treated with steroids and eye protection in the short term. Appropriate eye care is critical to avoid exposure keratitis, which is entirely preventable. A recommended regimen includes hourly artificial tears drops while awake, Lacri-Lube ointment (Allergan, Inc., Irvine, CA) with a square of saran wrap taped over the eye while sleeping ("moisture chamber"). The ointment and wrap can also be used while awake if the patient is noncompliant with the drops regimen. Taping an eye is to be avoided. If the eye opens under the tape, which it likely will do, the cornea can be abraded. Moreover, in the setting of concurrent facial and trigeminal dysfunction, the patient will not feel the pain of the eye drying or abrading. Prolonged facial weakness may warrant an upper eyelid gold weight placement, as well as surgical reanimation. If no function returns after 18 to 24 months, then static reanimation is required (see also Chapter 7.2).

CSF otorrhea, CSF rhinorrhea, or incisional CSF leaks may resolve with lumbar drain placement and pressure dressing. Incisional CSF leak is more likely to respond to this type of conservative management. Continued CSF leaks should be treated with reexploration, middle ear obliteration, and eustachian tube closure.

Unilateral complete hearing loss can be rehabilitated with a CROS aid or a BAHA (see Chapter 2.5.5). These techniques function on the principle of transferring sound from the deaf ear to the hearing ear.

◆ Outcome and Follow-Up

Patients may have significant vertigo following surgical treatment until adequate compensation occurs over several weeks. This is especially true of patients without preoperative vestibular hypofunction who have, therefore, no previous compensation. Short-term vestibular suppressants and vestibular therapy can help improve symptoms. Careful monitoring of facial nerve function and observation for evidence of meningitis or CSF leak is critical. CSF leak rates run from 10 to 15%. Facial nerve preservation rates are very dependent on both surgeon experience and tumor size. Results drop as tumor size increases. Facial nerve preservation can be much more difficult when removing previously irradiated tumors, should the need arise.

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Table 2.17 Timetable of Possible Postoperative Complications Following Craniotomy and Cerebellopontine Angle Tumor Resection

← **ABCs** →
Airway compromise: mucus plugging, ventilator problems, vocal fold paralysis from lower cranial nerve dysfunction.
Bleeding: within the CPA from operative field – contralateral long-tract signs, ↑ICP
Circulation: normotension not maintained

← **Intracranial hemorrhage** →
(Parenchymal, epidural, subdural)

← **Infarct** →
(AICA, cerebellar, cerebral)

CNS edema
← **SIADH/DI** →
(Watch sodium; consider hydrocephalus; RAS suppression; medication effects)

← **Emesis followed by change in mental status** →
(Bleed, pneumocephalus, CSF leak.
Could be prompted by movement, or disorientation in CT scanner)

← **Fever, meningitis** →

←**DVT, PE** →
(TEDS, sequential compression stockings)

TIME: →
Postoperative day #

0	1	2	3	4	5	+
ICU	floor	Start PO intake Start PT				

Comments:

RAS suppression: Edema, increased pressure or displacement can inhibit brainstem reticular activating system function, causing decreased consciousness in the early postoperative period. CPA surgery and cerebellar retraction predispose the patient to edema, *especially 24–72 hours postoperatively*. *Keep Na > 140; consider steroids.*

Increased intracranial pressure: Hemiparalysis with obtundation, a fixed dilated pupil and respiratory distress are the hallmarks of rapidly increasing ICP with peduncular herniation. If the patient does not recover properly from anesthesia or there is any unexpected neurologic abnormality, suspect hematoma, hydrocephalus with increased ICP, or cerebellar infarct. If deterioration is slow, obtain a CT scan; if rapid, go to OR.

Meningitis: Most common at postoperative day 4 or later; marked by postoperative fever, increased headache, malaise, photophobia, nausea. Promptly obtain LP with Gram stain, cell count, glucose, cultures. Treat with IV antibiotics and close monitoring.

CSF leak: There is a 5–17% incidence. Mastoid air cells may be opened intraoperatively during craniotomy or IAC drilling. If CSF rhinorrhea develops, treat with antibiotics, head of bed elevation, ± intermittent LP versus lumbar drain. If copious leakage, prompt mastoidectomy with fat obliteration is indicated.

Abbreviations: AICA, anterior inferior cerebellar artery; CNS, central nervous system; CPA, cerebellopontine angle; CSF, cerebrospinal fluid; CT, computed tomography; DI, diabetes insipidus; DVT, deep vein thrombosis; ICP, intracranial pressure; ICU, intensive care unit; LP, lumbar puncture; PE, pulmonary embolus; PT, physical therapy; RAS, reticular activating system; SIADH, syndrome of inappropriate antidiuretic hormone; TEDS, thromboembolic deterrent stockings.

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Patients undergoing observation or radiotherapy should have repeat MRI scans every 6 to 12 months to watch for tumor growth. Surgical patients should have follow-up MRIs at 1 and 5 years postoperatively. Some surgeons will follow their tumor patients indefinitely.

◆ ICD-9 Codes

225.1 benign neoplasm of cranial nerves
237.72 Neurofibromatosis type 2

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2.9 Superior Semicircular Canal Dehiscence Syndrome

◆ Key Features

- The Tullio phenomenon, autophony, and hyperacusis are findings.
- A third mobile window leads to conductive loss with intact acoustic reflex.
- An absence of intact bone overlying the superior semicircular canal can be seen on high-resolution CT.
- VEMPs have larger amplitudes and decreased thresholds.
- Resurfacing procedure can help severely symptomatic patients.

Superior semicircular canal dehiscence syndrome (SCDS) was first described by Minor in 1998 and is the absence of bone overlying the superior semicircular canal, leading to vestibular and auditory symptoms. Findings with SCDS include an apparent CHL with intact acoustic reflexes, conductive hyperacusis, pulsatile tinnitus, and Tullio phenomenon.

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◆ Epidemiology

The incidence of SCDS is unknown. However, temporal bone studies suggest that the anatomic incidence of dehiscence of the superior canal is 0.5%.

◆ Clinical

Signs and Symptoms

Clinically, signs and symptoms may range from very mild to severe. Patients with SCDS may exhibit some or all of the following:

- Tullio phenomenon – vertigo or dizziness induced by loud sound, with oscillopsia and nystagmus
- Autophony – the person's own speech or other self-generated noises such as chewing is perceived as unusually loud in the affected ear
- Dizziness, disequilibrium, or true vertigo
- Hyperacusis
- Conductive hearing loss, often in the low-frequency range
- Aural fullness
- Pulsatile tinnitus
- Headache

Differential Diagnosis

With a healthy or normal-appearing middle ear, the presence of normal acoustic reflex should help discriminate otosclerosis with stapes immobility from possible SCDS. Obviously, one must exclude other identifiable causes of CHL, such as tympanic membrane perforation, ossicular chain problems, or middle ear effusion. The differential diagnosis for disequilibrium is broad. Noise-induced vertigo raises suspicion for SCDC. Perilymph fistula and an enlarged vestibular aqueduct are possible conditions associated with an inner ear third mobile window. Ménière disease may have some overlapping findings.

◆ Evaluation

History

History should focus on complaints described above, including history of dizziness in loud noise, bothersome autophony, or aural fullness.

Physical Exam

A full head and neck exam is done, including the cranial nerve exam. The evaluation of a patient suspected of having SCDS should include imaging, audiologic assessment, and VEMP testing.

Imaging

High-resolution CT scanning (0.6 mm slice thickness or less) of the temporal bone is obtained and can suggest dehiscence of bone overlying the superior

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semicircular canal when examining coronal images. Dehiscence may be better appreciated if the images can be reformatted in the plane parallel to the superior canal and in the plane perpendicular to the superior canal.

Labs

No specific laboratory abnormality is seen with SCDS. However, the evaluation of the patient with disequilibrium or vertigo may otherwise include serology. For instance, RPR or FTA-ABS may be obtained to rule out syphilis, or Lyme serology may be ordered if in an endemic area or risk factors are present.

Other Tests

Audiologic Assessment

Audiologic assessment should include pure tone testing for air-conduction thresholds from 250 to 8000 Hz, and bone-conduction thresholds from 250 to 4000 Hz. Conductive hyperacusis may be assessed by administering bone-conduction testing at stimulus levels less than 0-dB hearing loss (e.g., −5 to −10 dB). Assessment should also include tympanograms, otoacoustic emissions, and ipsilateral acoustic reflexes.

Vestibular Evoked Myogenic Potential Testing

VEMP testing can be used to provide evidence of altered vestibular function; thus, it can help to determine if a suspected dehiscence of the superior canal, seen on CT scanning, is associated with an alteration of inner ear physiology. Patients with SCDS have larger amplitudes and lower thresholds on VEMP testing. A tone burst is applied via insert earphones and the evoked EMG activity is recorded from the ipsilateral sternocleidomastoid muscle.

Pathology

A thinning of the bone overlying the superior semicircular canal leads to a third mobile window within the inner ear. This alters inner ear mechanics leading to the clinical findings. The precise cause of the dehiscence is unknown.

◆ Treatment Options

Medical

Accurate diagnosis is important, as unnecessary stapes surgery (for the CHL) must be avoided. If the patient has mild symptoms, the identification of a source for the symptoms may provide reassurance, and ongoing observation may be all that is required.

Surgical

If the patient has severe symptoms, surgery to resurface or occlude the dehiscence may be warranted. The surgical candidate should have a correlation of symptoms, exam findings, cervical VEMPs, and imaging. The procedure involves a middle fossa craniotomy; brief retraction of the temporal lobe; localization of the dehiscence, often using computer-assisted CT surgical

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navigation; and plugging the dehiscence with preferred material such as bone pate, surgical glue material, and/or fascia.

◆ Outcome and Follow-Up

A serial hearing testing is recommended. If symptoms progress, reimaging and repeat VEMP testing may be helpful.

◆ ICD-9 Code

386.43 Semicircular canal fistula

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2.10 Otologic Manifestations of Systemic Diseases

◆ Key Features

- Many systemic diseases may affect the cochleovestibular system.
- Otologic manifestations can be the presenting signs of systemic disease.
- A high index of suspicion is required when evaluating otologic complaints.

A diverse group of systemic diseases affect the ear and produce otologic complaints. These conditions can be broadly characterized as infectious/granulomatous, autoimmune, neoplastic, metabolic, disorders of bone, and immunodeficiencies. Otologic manifestations can be either part of the disease progression or the initial findings that can herald a diagnosis. A high index of suspicion is required, therefore, when investigating a patient's otologic complaints.

◆ Infectious/Granulomatous Processes

Tuberculosis (TB)

- *Mycobacterium tuberculosis* via hematogenous, lymphatic or direct from eustachian tube

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- Chronic otorrhea, TM perforations, granulation in middle ear, bone sequestra
- Diagnosis: acid-fast stain of exudates, culture (negative in 70%), PCR
- Treatment: standard anti-TB therapies, surgery for refractory cases

Syphilis

- Spirochetal infection, *Treponema pallidum*, congenital and acquired forms
- Ossicular/temporal bone osteitis in latent syphilis
- Hearing loss is abrupt, bilateral, and progressive in secondary/tertiary syphilis.
- Granulomatous lesions, gummas, affect middle ear and cause TM perforation
- Treatment: benzathine penicillin 2.4 million units every week for 6 to 12 weeks, prednisone 60 mg every other day for 3 to 6 months, tapered slowly

Lyme Disease

- Spirochetal infection, *Borrelia burgdorferi*, transmitted by ticks
- Facial paralysis (especially bilateral), tinnitus, SNHL, otalgia, vertigo
- Treatment: doxycycline 100 mg orally every day for 14 to 21 days

Mumps

- RNA paramyxovirus
- Unilateral, high-frequency hearing loss, tinnitus. Hearing loss is usually permanent, vestibular symptoms resolve over weeks
- Treatment: nonspecific; steroids might be of some benefit

Measles (Rubeola)

- RNA paramyxovirus
- Bilateral, high-frequency SNHL occurs acutely with onset of rash.
- 50% of patients have improvement in hearing loss.

Sarcoidosis

- Multisystem disorder characterized by noncaseating granulomas
- SNHL, vestibular dysfunction, facial nerve paralysis
- Uveoparotid fever (Heerfordt syndrome): facial paralysis, parotitis, uveitis, pyrexia
- Treatment: nonspecific, corticosteroids, cytotoxic agents

Wegener Granulomatosis

- Otologic involvement in 35% of cases, may be the first sign of disease
- Facial/postauricular pain, erythematous auricular swelling
- Otitis media, CHL, SNHL, vertigo, facial paralysis (rare presenting sign)

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- Diagnosis: c-ANCA present in 90% of patients with active disease
- Treatment: corticosteroids, cytotoxic agents

Langerhans Cell Histiocytosis

- Disorders characterized by proliferation of benign histiocytes
- Otologic manifestations in 15 to 61% of cases; may be presenting symptoms
- Otorrhea, postauricular swelling, hearing loss, vertigo, aural polyps, granulation tissue in EAC, TM perforations
- Predilection for mastoid, erosion of posterior canal, secondary infection
- Treatment: corticosteroids, cytotoxic agents

◆ Autoimmune

Relapsing Polychondritis

- Recurrent inflammation of cartilaginous structures with eventual fibrosis
- Tender, red, edematous auricle with sparing of the lobule and external ear canal
- Diagnosis: mainly clinical, biopsy during acute phase is avoided
- Treatment: corticosteroids

Chronic Discoid Lupus Erythematosus

- Limited to skin of head, neck, and chest in 90% of cases
- Well-circumscribed, raised lesions on auricle that are pruritic and slowly enlarge
- Treatment: nonspecific, corticosteroids may be helpful

Polyarteritis Nodosa

- Necrotizing vasculitis of small and medium-diameter arteries
- CHL, SNHL, occasional facial paralysis with hearing loss
- Treatment: corticosteroids, cyclophosphamide

Rheumatoid Arthritis

- Affects ossicular articulations causing CHL
- Treatment: NSAIDs, glucocorticoids, immunosuppressive therapy

◆ Neoplastic

Multiple Myeloma

- Malignancy of plasma cells, B cell derivatives
- Temporal bone frequently involved, may be only manifestation (plasmacytoma)
- Rounded, lytic lesions of the calvaria and temporal bone

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Leukemia

- Leukemic infiltrates affect mastoid, middle ear, petrous apex
- Hemorrhage can often accompany infiltrates in these areas
- Sludging of cells in cochlea may cause ischemic hearing loss

Metastatic Neoplasms

- In decreasing frequency: breast, lung, prostate, skin
- Lesions are usually osteolytic, but can also be osteoblastic (e.g., breast, prostate)
- Most commonly involve internal auditory canal and petrous apex

◆ Metabolic Diseases

Gout

- Deposits of monosodium urate crystals in joints and cutaneous structures (tophi)
- Tophaceous deposits in helical rim are classic and are usually asymptomatic.

Ochronosis

- Lack of enzyme: homogentisic acid dehydrogenase
- Deposition of dark pigments in tissues rich in collagen
- Cartilage of ear is affected: blue, mottled brown macules on auricle

Mucopolysaccharidoses

- Characterized by deficiency of enzymes that degrade mucopolysaccharides
- CHL or SNHL (unknown mechanism)
- Eustachian tube dysfunction and hypertrophy of middle ear mucosa are seen.

◆ Bone Diseases

Paget Disease (Osteitis Deformans)

- Osteolytic and osteoblastic changes affecting axial skeleton in older individuals
- Mixed, progressive hearing loss, tinnitus, vestibular dysfunction
- Facial nerve not involved

Osteogenesis Imperfecta

- Genetic disorder of collagen synthesis manifested by fragile bones
- CHL and SNHL
- No correlation between hearing loss and severity or frequency of fractures
- Can mimic otosclerosis

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Osteopetrosis

- Characterized by an increased bone density
- Affects the endochondral layer of otic capsule and ossicles; poorly pneumatized mastoid compartment
- Recurrent OM, mixed hearing loss, unilateral or bilateral facial nerve palsies

Fibrous Dysplasia

- Benign, chronic disease characterized by replacement of bone with fibrous tissue
- Temporal bone involvement most common in monostotic form
- Painless swelling in mastoid and squamous portions, narrowing of EAC, CHL
- Treatment is symptomatic.

◆ Immunodeficiencies

Primary/Congenital

- Result in recurrent OM often refractory to medical and surgical therapies
- DiGeorge syndrome (cellular immunodeficiency) associated with Mondini deformity

Acquired Immunodeficiency Syndrome

- HIV/AIDS
- Kaposi sarcoma can affect outer ear.
- *Pneumocystis carinii* can cause middle and external ear disease.
- Otosyphilis, tuberculous otitis, fungal infections, and herpes zoster can also occur.

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3 Rhinology

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3.0 Anatomy and Physiology of the Nose and Paranasal Sinuses

The external nose consists of the nasal pyramid (frontal process of the maxilla) with the paired nasal bones forming the dorsum and meeting the frontal bone superiorly at the glabella. Inferiorly are the upper lateral cartilages and lower lateral (alar) cartilages, which contribute to the nasal tip and nasal valves (**Fig. 3.1**).

The internal nasal and sinus anatomy is complex and variable. The midline nasal septum is composed of the quadrangular cartilage, the perpendicular plate of the ethmoid bone, the vomer bone, and the palatine bone, with an overlying mucosal covering. There are four paired sinuses: the maxillary, frontal, ethmoid, and sphenoid (**Fig. 3.2**). The lateral nasal wall consists of the inferior, middle, and superior turbinates. The nasolacrimal duct opens in the inferior meatus. The frontal, anterior ethmoidal and maxillary sinuses drain via the middle meatus. The posterior ethmoids drain via the superior meatus. The sphenoid ostia are near the level of the superior meatus on the anterior wall of the sphenoid sinus. Clinically, the relation of the paranasal sinuses to adjacent anatomic structures is important as it relates to the potential for the spread of infection or an iatrogenic injury. Specifically, the ethmoid roof may be an extremely thin bone along the lateral lamella of the cribriform plate and may vary in its height considerably; intracranial contents lie superiorly. The lamina papyracea separates the orbit from the ethmoids; the orbital spread of infection is discussed in Chapter 3.1.2.

The sphenoid sinus is bounded by the internal carotid artery, optic nerves, and cavernous sinus and sella; an overriding posterior ethmoid (Onodi) cell may risk critical structures. Dehiscence of the bone covering the internal carotid within the sphenoid is relatively common and should be routinely assessed for on a preoperative computed tomography (CT) scan. The frontal

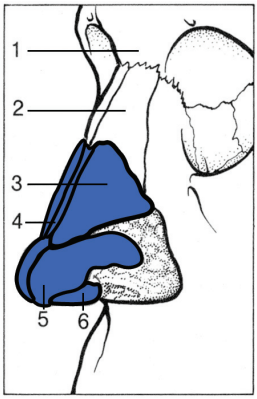

Fig. 3.1 Nasal skeleton. 1. Glabella. 2. Nasal bone. 3. Upper lateral cartilage. 4. Upper edge of the cartilaginous nasal septum. 5. Lower lateral cartilage. 6. Medial crus of lower lateral cartilage. (From Becker W, Naumann HH, Pfaltz CR. Ear, Nose, and Throat Diseases: A Pocket Reference. 2nd ed. Stuttgart/New York: Thieme; 1994:170)

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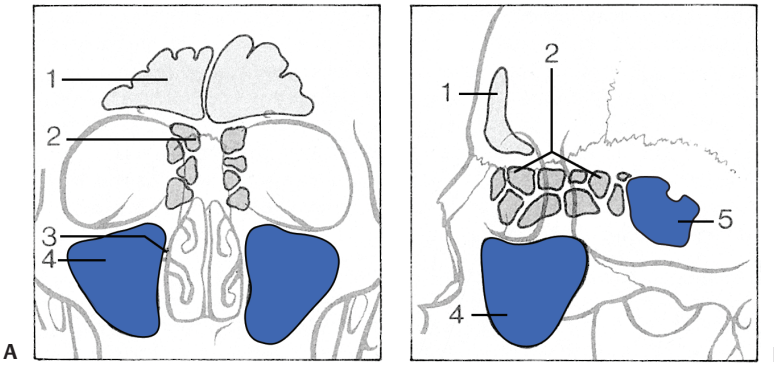

Fig. 3.2 Nasal sinuses: **(A)** frontal section, **(B)** sagittal section. 1. Frontal sinus. 2. Ethmoid sinus. 3. Maxillary ostium. 4. Antral cavity. 5. Sphenoid cavity. (From Becker W, Naumann HH, Pfaltz CR. Ear, Nose, and Throat Diseases: A Pocket Reference. 2nd ed. Stuttgart/New York: Thieme; 1994:175)

sinus is bounded by the orbit and the anterior fossa, and also may be a source of spread of rhinogenic infection. A Haller cell is an anterior ethmoid cell that pneumatizes laterally at the orbital floor and can contribute to maxillary sinus drainage problems. Agar nasi cells are anterior ethmoid cells that pneumatize superiorly and can contribute to frontal sinus drainage problems.

◆ Blood Supply

There is abundant external and internal carotid supply (**Fig. 3.3**). Kiesselbach's plexus (also known as Little's area) of the anterior septum provides superficial anastomoses. External carotid branches supply the nose via the facial artery externally and the maxillary artery internally, including the sphenopalatine artery. Internal carotid branches are supplied via the ophthalmic artery to the anterior and posterior ethmoid arteries. Venous drainage occurs via facial veins as well as ophthalmic veins, which have valveless intracranial connections to the cavernous sinus and therefore relate to intracranial hematogenous spread of infection. Epistaxis is discussed in Chapter 3.1.5.

◆ Innervation

General sensory supply is via the first and second divisions of the trigeminal nerve. Importantly, the nasal tip is supplied via V1 (the first division if the trigeminal nerve). Thus, if possible herpetic lesions involve the nasal tip, ophthalmologic evaluation is indicated to rule out herpes zoster of the eye. Special sensory supply is via the olfactory (first cranial) nerve. Complex autonomic innervation is supplied to mucosa via the pterygopalatine ganglion regulating vasomotor tone and secretion. The vidian nerve contains

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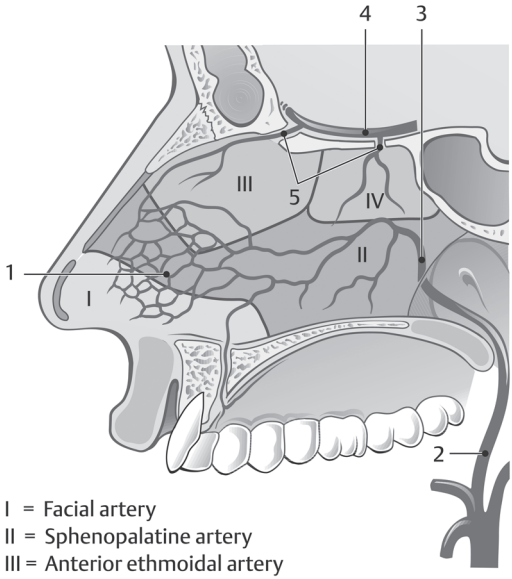

Fig. 3.3 Vasculature of the nasal cavity. 1. Kiesselbach's plexus. 2. Internal maxillary artery. 3. Sphenopalatine artery. 4. Ophthalmic artery. 5. Anterior and posterior ethmoid arteries. I–IV: areas supplied by arteries to the nose. (From Behrbohm H et al. Ear, Nose, and Throat Diseases: With Head and Neck Surgery. 3rd ed. Stuttgart/New York: Thieme; 2009:122)

preganglionic parasympathetic fibers from the greater superficial petrosal nerve (from cranial nerve [CN] VII) and sympathetic fibers from the deep petrosal nerve (from superior cervical ganglion) and synapses at the pterygopalatine ganglion prior to innervating the sinonasal mucosa.

◆ Physiology

Table 3.1 gives a summary of tests for the assessment of sinonasal physiology. Warming and humidification of inspired air, olfactory function, and immune function all are aspects of nasal physiology. The nose and paranasal sinuses play a role in host defenses. In general, mucociliary clearance is a key feature. Specific factors such as secretory immunoglobulin A (IgA), lactoferrin, lysozyme, cytokines, and the complex regulation of cells that mediate immunity are critical to the maintenance of normal sinus function. A detailed discussion of sinonasal physiology is beyond the scope of this book. The presence of infection, inflammation, allergy, neoplasm, or traumatic, iatrogenic, or congenital deformity may all perturb sinonasal physiology and must be considered in the evaluation of the patient with complaints related to the nose.

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Table 3.1 Summary of Tests for Factors Affecting Sinonasal Physiology

Lab Test	Utility (Tests for)
RPR (FTA-ABS)	Syphilis
c-ANCA	Wegener disease
ACE	Sarcoidosis
ANA	Autoimmune disease
ESR	Inflammation
Cultures of sinus drainage	Specific infectious organism
University of Pennsylvania Smell Identification Test (SIT)	Olfactory function
RAST (blood testing for allergen-specific IgE)	Allergy
Skin testing (prick test; SET)	Allergy
Acoustic rhinometry Rhinomanometry	Nasal obstruction; not currently widely used outside of research settings
Ciliary beat frequency Ciliary electron microscopy	Assess ciliary function and anatomy, i.e., for Kartagener syndrome. Typically tested on adenoid or mucosal biopsy.

Abbreviations: ACE, angiotensin-converting enzyme; ANA, antinuclear antibody test; c-ANCA, antineutrophil cytoplasmic antibody; ESR, erythrocyte sedimentation rate; FTA-ABS, fluorescent treponemal antibody absorbed; IgE, immunoglobulin E; RAST, radioallergosorbent test; SET, skin endpoint titration.

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3.1 Rhinologic Emergencies

3.1.1 Acute Invasive Fungal Rhinosinusitis

◆ Key Features

- A rapidly progressive sinonasal fungal infection can be fatal.
- Acute invasive fungal infections occur almost exclusively in immunocompromised or debilitated patients,
- Successful treatment requires early detection, wide surgical débridement, and correction of the underlying predisposing condition.

In the debilitated patient, certain fungal infections can become angioinvasive with tissue necrosis, cranial nerve involvement, and possible orbital or intracranial extension. The most common organisms are *Mucor* or *Aspergillus* species. High-risk patients include those with neutropenia from any cause (e.g., leukemia, bone marrow transplantation), other oncology patients undergoing chemotherapy, chronic immunosuppressive therapy or corticosteroid use, diabetes mellitus and diabetic ketoacidosis, or acquired immunodeficiency syndrome (AIDS). Acute invasive fungal rhinosinusitis is a distinct and rapidly aggressive disease process that is distinguished by its fulminant course from other forms of fungal sinusitis, such as mycetoma, allergic fungal rhinosinusitis, or chronic invasive (indolent) fungal rhinosinusitis.

◆ Epidemiology

Also known as rhinocerebral mucormycosis, acute invasive fungal rhinosinusitis occurs in the at-risk populations described above: patients with hematologic malignancies, patients status post solid organ or bone marrow transplant, diabetics, those on chronic steroid therapy, neutropenic patients, and patients with AIDS.

◆ Clinical

Signs and Symptoms

A high index of suspicion in any at-risk patient is required, as early diagnosis improves prognosis. A fever of unknown origin should raise suspicion, as should any new sign or symptom of sinonasal disease. Facial edema, periorbital swelling, pain, or numbness are common findings. However, the leukopenic patient may be unable to mount a febrile response. Other findings may include epistaxis, headache, mental status change, or crusting/eschar at the naris that can be mistaken for dried blood. One should consider

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unilateral cranial neuropathy, acute visual change, or altered ocular motility in an immunocompromised patient to be acute invasive fungal rhinosinusitis until proven otherwise. A black intranasal eschar on exam is considered pathognomonic for invasive *Mucor*.

Differential Diagnosis

A noninvasive sinonasal infection, such as acute bacterial sinusitis, should be considered. An acute bacterial sinusitis complication, such as orbital cellulitis or intracranial suppurative spread may present similarly. Radiographically similar processes may include squamous cell carcinoma, sinonasal lymphoma, and Wegener granulomatosis.

◆ Evaluation

See **Fig. 3.4** for a diagnostic and treatment algorithm.

Physical Exam

The patient suspected to have acute invasive fungal rhinosinusitis should be seen without delay. The head and neck examination should focus on cranial nerve function and should include nasal endoscopy. Avoid tetracaine spray or other topical anesthetics. Insensate mucosa noted during an endoscopic exam is consistent with invasive fungal infection. Dark ulcers or pale, insensate mucosa may appear on the septum, turbinates, palate, or nasopharynx. Early infection may appear as pale mucosa; the presence of dark eschar has been considered to be pathognomonic. Signs of cavernous sinus thrombosis include ophthalmoplegia, exophthalmos, and decreased papillary responses.

Biopsy of suspicious areas such as the middle turbinate or septal mucosa is required for diagnosis. It is important to obtain actual tissue at biopsy, not just overlying eschar or necrotic debris. These specimens should be sent fresh for immediate frozen section analysis as well as silver stain. Patients may be thrombocytopenic, and although a low platelet count may lead to profuse bleeding after biopsy, the risk of this must be balanced with the high mortality associated with a delay in diagnosis. If necessary, platelet transfusion should be ordered early. As a rule, a platelet count of 50,000 is desired. Acceptable hemostasis can usually be obtained with chemical cautery and Avitene (Davol, Inc., Cranston, RI), Gelfoam (Pfizer Pharmaceuticals, New York, NY), or other hemostatic packing.

Imaging

CT findings may be nonspecific. However, the presence of bone erosion and adjacent soft tissue edema on contrast-enhanced maxillofacial CT strongly suggests the diagnosis if clinical correlation is present. Unilateral edema of the nasal mucosa has also been associated with invasive fungal sinusitis, as well as obliteration of the retroantral fat planes. Both soft tissue and bone windows, as well as high-resolution axial and coronal views are necessary. Magnetic resonance imaging (MRI) is useful to evaluate intracranial extension and extension beyond the paranasal sinuses.

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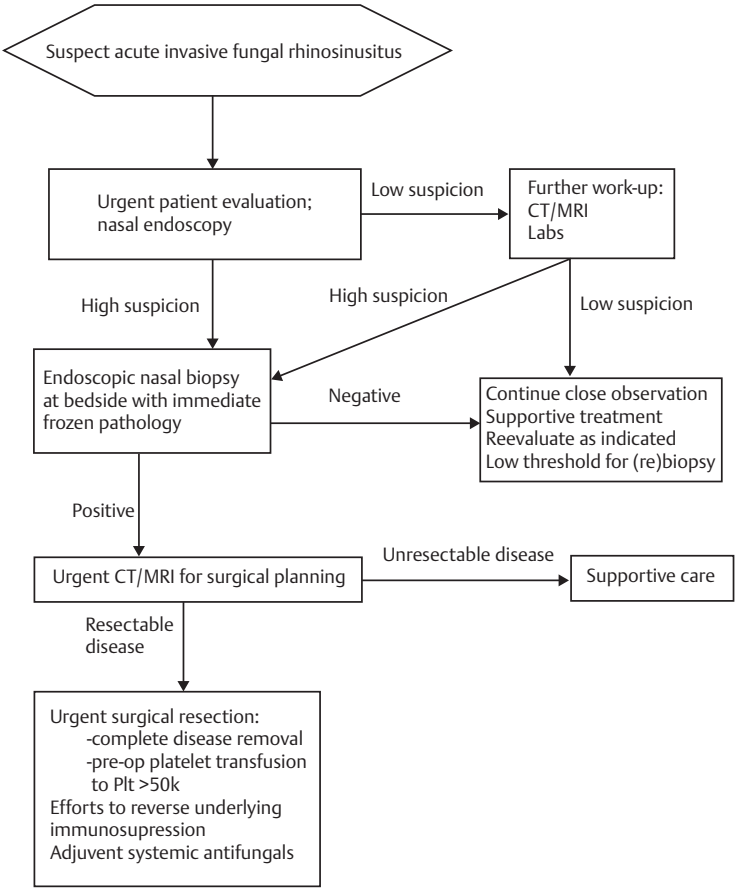

Fig. 3.4 Treatment algorithm for acute invasive fungal rhinosinusitis. Note that there should be a very low threshold to proceed with biopsy, as rapid diagnosis and treatment is critical to patient survival. CT, computed tomography; MRI, magnetic resonance imaging.

Labs

Cultures are inadequate and play no role in the initial diagnosis and management of suspected acute invasive fungal rhinosinusitis. Positive culture results will most likely be available late in the course of the disease. Useful labs to assess risk factors include a complete blood count (CBC), absolute neutrophil count, chemistries, blood glucose and hemoglobin A1C (HbA1c) in the diabetic, and human immunodeficiency virus (HIV) testing with CD4 lymphocyte counts and viral load in AIDS patients.

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Pathology

Biopsy of the middle turbinate or other suspicious lesions with immediate frozen section analysis is the gold standard test to confirm the presence of tissue-invasive fungus. *Mucor* is identifiable within the mucosa as large, irregularly shaped nonseptate hyphae that branch at right angles. *Aspergillus* is identifiable as smaller hyphae that are septate and branch at 45-degree angles. Methenamine silver stain is performed to confirm the diagnosis; however, these results may not be available for several hours.

◆ Treatment Options

This is a surgical emergency: complete surgical resection and the reversal of underlying immune dysfunction are critical. The diabetic patient can be successfully treated with early diagnosis, insulin drip, and wide surgical resection. In the oncology patient, if neutropenia cannot be reversed, mortality is high. Granulocyte-macrophage colony-stimulating factor (GM-CSF) may improve survival. Surgical goal is resection of all involved tissue. This may be accomplished endoscopically in select cases. However, an extended total maxillectomy with orbital exenteration may be necessary in advanced disease. Systemic antifungals as well as intranasal nebulized amphotericin are administered, but should be considered adjuvant therapy.

◆ Outcome and Follow-Up

Prognosis is very poor with intracranial involvement. A bone marrow transplant patient with uncorrectable neutropenia has a poor prognosis. Overall survival in diabetic patients may approach 80% if ketoacidosis is corrected.

◆ ICD-9 Codes

117.7	Zygomycosis (phycomycosis or mucormycosis)
117.3	Aspergillosis
461	Acute sinusitis
473	Chronic sinusitis

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3.1.2 Orbital Complications of Sinusitis

◆ Key Features

- Sinusitis is a common cause of orbital infection.
- Significant morbidity and even mortality can result.
- Orbital extension of sinusitis is most common in pediatric patients.
- Combined otolaryngology and ophthalmology care is required.

Orbital extension of sinonasal disease requires immediate attention, as rapid progression and blindness may occur. Anatomically, the orbit is bounded by all paranasal sinuses and infection may spread to the orbit directly or via retrograde thrombophlebitis. The Chandler classification system is heuristically useful in staging and managing orbital complications of sinusitis (**Table 3.2**). Hospital admission and intravenous antibiotic therapy are required for treatment; surgical drainage is necessary for abscess formation, vision compromise, or lack of improvement with medical therapy.

Table 3.2 Chandler Classification

Stage	Description
I	Preseptal cellulitis
II	Orbital cellulitis
III	Subperiosteal abscess
IV	Orbital abscess
V	Cavernous sinus septic thrombosis

◆ Epidemiology

Orbital complications occur in ~3% of sinusitis cases. This is most common in children but can occur at any age. Subperiosteal abscess is present in ~20% of cases of orbital extension of sinusitis. Cavernous sinus thrombosis is rare. Immunosuppressed patients are at increased risk and require aggressive treatment.

◆ Clinical

Signs and Symptoms

The most common findings are orbital edema, pain, proptosis, and fever. Orbital disease may be the first sign of sinusitis in children. In more advanced cases, there may be gaze restriction and visual acuity change.

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Differential Diagnosis

In the pediatric age group, orbital pseudotumor consists of painful proptosis without a fever or leukocytosis. Orbital rhabdomyosarcoma may present with inflammatory changes in 25% of patients. An ethmoid mucocele may present with proptosis, and CT will reveal an expanded sinus in this instance. Other sinonasal causes of proptosis or orbital edema include allergic fungal rhinosinusitis and neoplasm, as well as iatrogenic injury. Abnormal thyroid state may cause ophthalmopathy.

◆ Evaluation

Physical Exam

Examination requires the combined input of the otolaryngologist and the ophthalmologist. In general, the patient will have a history of preceding sinusitis or current complaints consistent with acute sinusitis. Fever is usually present. Head and neck exam reveals lid or periorbital edema, erythema, and tenderness. In cases of preseptal (periorbital) cellulitis, the remainder of the eye exam is normal. The presence of proptosis, chemosis extraocular muscle limitation, diplopia, or decreased visual acuity suggests orbital cellulitis or subperiosteal abscess. With cavernous thrombosis or intracranial extension, findings may include a frozen globe (ophthalmoplegia), papilledema, blindness, meningeal signs, or neurologic deficits secondary to brain abscess or cerebritis. Superior orbital fissure syndrome is a symptom complex consisting of retroorbital pain, paralysis of extraocular muscles, and impairment of first trigeminal branches. This is most often a result of trauma involving fracture at the superior orbital fissure, but dysfunction of these structures can arise secondary to compression. Orbital apex syndrome adds involvement of the optic nerve.

Imaging

Contrast-enhanced CT scanning with coronal and axial views is the imaging study of choice if there is any suspicion of postseptal involvement (i.e., other than simple periorbital cellulitis). A subperiosteal abscess is identifiable as a lentiform, rim-enhancing hypodense collection in the medial orbit with adjacent sinusitis. The medial rectus is displaced. In the absence of abscess formation, there may be orbital fat stranding, solid enhancing phlegmon, or swollen and enhancing extraocular muscles, consistent with orbital cellulitis. A lid abscess may be present less commonly. Suspicion of cavernous sinus thrombosis is better evaluated with MRI.

Labs

A CBC with differential may be useful. Preoperative labs should be ordered as indicated.

Pathology

In younger children, microbiology is often single aerobes including alpha *Streptococcus*, *Haemophilus influenzae*, or coagulase-positive *Staphylococcus*. In those over 10 years old, organisms are often mixed and may include anaerobes.

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◆ Treatment Options

Medical

All patients should be admitted and treated with serial ophthalmologic exams and intravenous (IV) antibiotics that have good cerebrospinal fluid (CSF) penetration. Generally, a third-generation cephalosporin is used. Oxacillin is often used in children. Older patients are often double-covered with clindamycin for anaerobes. Alternatives include ampicillin/sulbactam, vancomycin, or aztreonam. Antibiotics are adjusted according to cultures, if possible. Systemic steroids are not recommended. Topical nasal vasoconstrictors are useful (i.e., oxymetazoline).

Surgical

A majority of patients with orbital complications require surgery. This is an area of some controversy. Clearly, surgical drainage is required urgently for abscess formation or decreased visual acuity. If the clinical setting allows for close follow-up (i.e., frequent serial ophthalmologic assessment), many clinicians will observe certain cases of preseptal or early postseptal cellulitis. If there is any progression or lack of resolution with medical therapy over 48 hours, surgery is recommended. Surgical drainage may be accomplished endoscopically by experienced surgeons; however, consent for an external ethmoidectomy approach is recommended. Regardless of approach, the abscess should be drained and the underlying sinus disease should be addressed. For cavernous thrombosis, involved sinuses including the sphenoid must be drained; systemic anticoagulation remains controversial.

◆ Outcome and Follow-Up

The natural history of untreated disease (all stages) results in blindness in at least 10%. Most cases of Chandler stage I–IV disease recover well with treatment. There remains up to an 80% mortality rate with cavernous sinus involvement, although new literature reports suggest this figure is high.

◆ ICD-9 Codes

376.01	Orbital cellulites
461	Acute sinusitis
473	Chronic sinusitis
325	Phlebitis and thrombophlebitis of intracranial venous sinuses

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3.1.3 Intracranial Complications of Sinusitis

◆ Key Features

- Intracranial complications due to sinusitis are a life-threatening emergency.
- Management requires a multidisciplinary approach including neurosurgical consultation.
- Complications include meningitis, dural sinus thrombosis, and intracranial abscess.

Sinusitis may result in spread of infection intracranially. Extension may occur via osteomyelitic bone, trauma, or via venous channels. The frontal sinus is commonly the source, although ethmoid or sphenoid sinusitis can lead to intracranial spread. Complications include meningitis, epidural abscess, subdural abscess, parenchymal brain abscess, and cavernous sinus thrombophlebitis.

◆ Epidemiology

This is uncommon. Since the advent of antibiotics, the incidence has decreased dramatically. Currently, probably less than 1% of sinusitus cases are complicated by spread of infection.

◆ Clinical

Signs and Symptoms

The patient with meningitis of a rhinologic origin will manifest signs and symptoms typical of bacterial meningitis. These include high fever, photophobia, nausea and emesis, mental status change, and nuchal rigidity, pulse and blood pressure changes. However, epidural or subdural abscess without meningitis may be more subtle. Fever and headache may be present but are nonspecific. Usually focal signs are absent. Meningeal signs may develop gradually. A parenchymal brain abscess of rhinologic origin (frontal lobe abscess) may initially result in few signs or symptoms. However, this may progress from headache to signs of increased intracranial pressure, vomiting, papilledema, confusion, somnolence, bradycardia, and coma. Cavernous sinus thrombophlebitis results in spiking fevers, chills, proptosis, chemosis, decreased visual acuity and blindness, and extraocular muscle paresis. Infection can rapidly spread to the contralateral cavernous sinus via venous communications.

Differential Diagnosis

Meningitis of nonrhinologic origin must be considered. The incidental finding of paranasal sinus disease on imaging does not necessarily signify a causal relationship.

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◆ Evaluation

Pathogenesis may involve several mechanisms. Extension may occur via osteomyelitic bone. Also, traumatic bone disruption may allow communication of infected sinus contents with dura, for example, after posterior table fracture of the frontal sinus. Also, infection may propagate via venous channels in bone or retrograde venous circulation to the cavernous sinus. General hematogenous spread is possible, especially in a severely immunocompromised host.

Physical Exam

Complete head and neck exam is required with careful assessment of all cranial nerves. Nasal endoscopy may reveal active sinonasal disease and provide mucopus for culture and sensitivities. A neurologic exam including orientation to person, place, and date will reveal any focal deficits and serve as a useful baseline to monitor for any deterioration. The presence of ocular findings or neurologic deficit should prompt ophthalmologic and neurosurgical consultations.

Imaging

Both CT and MRI are usually obtained. Contrast-enhanced CT scanning will provide detail regarding paranasal sinus disease and will reveal evidence of bony dehiscence that may underlie intracranial spread of infection. Intravenous contrast will reveal an enhancing abscess. Gadolinium-enhanced MRI will provide a more sensitive assessment of dural enhancement and intracranial disease but lacks the bony detail necessary for planning sinus surgery.

Lumbar Puncture

Obtain CSF via lumbar puncture (LP) in the patient with possible meningitis, after obtaining a CT scan. There is a risk of acute tonsillar herniation following LP, almost always seen in patients with a noninfectious intracranial process causing a mass effect with localizing signs and papilledema. See **Table 3.3** for interpretation of CSF values. Cell count (tube 3), protein and glucose (tube 2), and Gram stain with culture and sensitivities (tube 1) are ordered. Cultures with sensitivity will guide antibiotic therapy.

Labs

In addition to CSF studies, other useful labs include blood cultures, CBC with differential, chemistries, and prothrombin time (PT), and partial thromboplastin time (PTT).

◆ Treatment Options

Medical

IV high-dose antibiotics are empirically started following LP, and adjusted based on cultures. Broad-spectrum agents with good CSF penetration are utilized, such as ceftriaxone or cefepime. Vancomycin is often added for

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Table 3.3 Interpretation of Cerebrospinal Fluid Findings

	Opening Pressure (cm H_2O)	Appearance	Cells (per mL)	Protein (mg %)	Glucose (% serum)
Normal	7–18	Clear	0 PMNs 0 RBCs 0–5 monocytes	15–45	50
Acute purulent meningitis	Often elevated	Turbid	Few–20k (WBCs mostly PMNs)	100–1000	<20
Viral meningitis, encephalitis	Normal	Normal	Few–350 WBCs (mostly monocytes)	40–100	Normal

Abbreviations: PMNs, polymorphonuclear cells; RBCs, red blood cells; WBCs, white blood cells.

double coverage and for possible methicillin-resistance. Peak and trough levels must be monitored with vancomycin. The role for anticoagulation for cavernous thrombophlebitis remains controversial. Anticoagulation may interfere with the ability to perform intracranial pressure monitoring or craniotomy.

Surgical

The underlying sinus infection is drained either by endoscopic or open approach. If an intracranial abscess is present, this is drained by the neurosurgeon in conjunction with sinus drainage.

◆ Outcome and Follow-Up

Close observation in an intensive care unit (ICU) setting with serial neurologic exams is required postoperatively. Intracranial pressure monitoring may be required.

◆ ICD-9 Codes

320.9 Meningitis due to unspecified bacterium
325 Phlebitis and thrombophlebitis of intracranial venous sinuses
324.0 Intracranial abscess
461.9 Acute sinusitis, unspecified
473 Chronic sinusitis

Further Reading

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3.1.4 Cerebrospinal Fluid Rhinorrhea

◆ Key Features

- CSF rhinorrhea may occur due to iatrogenic injury to the ethmoid roof during sinus surgery.
- CT and MRI, β-2 transferrin assay, radioactive pledget scan, or intrathecal fluorescein with endoscopy can be useful for evaluation.
- The skull base can be repaired with endoscopic instruments from below, or via neurosurgical approach from above.

Anterior skull base disruption can result in drainage of CSF via the nose. The underlying problem may be the result of trauma, iatrogenic injury, or congenital anomaly, or it may arise spontaneously with no obvious specific cause. The management of CSF rhinorrhea related to skull base injury from endoscopic sinus surgery is highlighted. CSF leak from the temporal bone may present with rhinorrhea via the middle ear and eustachian tube.

◆ Epidemiology

This is uncommon. Although the exact incidence of complications is unclear currently, previous estimates suggest a ~0.5% rate of skull base injury with CSF leak following ethmoid surgery.

◆ Clinical

Signs and Symptoms

Patients present with watery rhinorrhea. Often, the drainage can be provoked by leaning the patient forward with the head lowered. Typically, the drainage has a salty or metallic taste. Headache may be reported. Anosmia and nasal congestion may accompany iatrogenic skull base injury with encephalocele.

Differential Diagnosis

The consideration for watery or clear rhinorrhea includes various forms of rhinitis versus CSF drainage. Vasomotor rhinitis typically is elicited by cold temperatures, physical activity, or other specific stimuli.

◆ Evaluation

History

The approach to the patient typically begins with a thorough and complete history. A source of skull base damage may, of course, be quite obvious if there was a suspected iatrogenic injury during preceding endoscopic

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ethmoid surgery. One should note if the drainage is unilateral as well as its duration and severity. Associated complaints such as headache, visual disturbance, epistaxis, and anosmia should be noted. Details of any previous sinonasal surgery, neurosurgery, otologic surgery, or trauma are important. Any previous imaging studies of the head should be obtained, if possible. Previous history of meningitis should be sought. General past history, medication list, social and family history are obtained.

Physical Exam

A complete head and neck exam is performed. CNs I to XII should be tested. A standard test for olfactory function such as the Smell Identification Test (SIT; Sensonics, Inc., Haddon Heights, NJ) is recommended, to document olfactory function prior to treatment. Nasal endoscopy is performed. A 4-mm rigid 30-degree endoscope is ideal. Drainage, masses, edema, purulence, and prior surgical changes are noted. If the injury is iatrogenic, it may be possible to assess the location and size of the skull base defect. Be suspicious of any mass arising medial to the middle turbinate, as encephalocele or esthesioneuroblastoma can arise in this location. Having the patient perform the Valsalva maneuver may result in visible enlargement of a meningocele or encephalocele.

Imaging

Identification of the site of leak may be straightforward or may be difficult. For a known iatrogenic injury, thin-cut axial CT with coronal views will typically verify the site of injury along the ethmoid roof. This scan should be obtained using an image-guidance protocol so that computer-assisted surgical navigation can be used for endoscopic repair. For other situations, CT is useful to study the bony detail of the anterior skull base and sphenoids. A bony dehiscence may be evident. For a patient presenting with a soft tissue mass, MRI is the study of choice to assess for possible meningocele or encephalocele.

Labs

If rhinorrhea fluid can be collected, this should be sent for β-2 transferrin assay. Usually, at least 1 mL is required; the laboratory may require refrigeration or rapid handling of the specimen. The presence of β-2 transferrin suggests strongly that the fluid is indeed CSF.

Other Tests

See **Table 3.4** for a summary of available studies.

Radioactive Pledget Scanning

This study can be done to help confirm and localize a leak site. Small cottonoid 1 × 3 neuropledgets can be trimmed and placed within the nasal cavity in defined locations. Usually, two pledgets are placed per nostril, one anteriorly and one posteriorly, with the string secured to the skin and labeled. Radioactive tracer is then injected intrathecally, typically technetium-99

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Table 3.4 Diagnostic Studies for Suspected Cerebrospinal Fluid (CSF) Rhinorrhea

Study	Role
β-2 transferrin assay	Confirm that drainage is CSF
Radioactive pledget scanning	Confirm presence of CSF leak, provide localizing information
Intrathecal fluorescein	Localize site of leak during nasal endoscopy/endoscopic repair
CT scanning	Assess bony detail of anterior skull base; assess for intracranial hematoma or pneumocephalus following iatrogenic injury
MRI scanning	Assess for anterior skull base encephalocele, meningocele, neoplasm

Abbreviations: CT, computed tomography; MRI, magnetic resonance imaging.

(^{99m}Tc). After suitable time, the pledgets are removed and assayed for radioactivity counts. If there is an active CSF leak, the location of a "hot" pledget can guide exploration and repair.

Intrathecal Fluorescein

The use of a dye to visualize endoscopically the CSF leakage is extremely helpful, both for endoscopic diagnosis and at the time of repair. For the procedure, a lumbar drain is placed and 10 mL of the patient's CSF is collected, after which 0.1 mL of 10% IV fluorescein is diluted into the 10 mL CSF sample. This is reinjected intrathecally slowly over 5 minutes. The fluorescein causes active CSF leakage to appear greenish endoscopically. Intrathecal fluorescein can cause seizures at higher dosage; however, the protocol described here is widely accepted as safe.

◆ Treatment Options

Skull base disruption causing CSF rhinorrhea is managed with surgical repair. Repair of acute iatrogenic injury is discussed separately from other cases.

Repair of Acute Iatrogenic Injury

If the ethmoid roof is injured during sinus surgery, it may be possible to repair the injury. If there is extensive injury, severe bleeding, or obvious intradural injury, it is highly recommended that neurosurgical consultation be obtained, if possible. Concomitant injury to the anterior ethmoid artery can occur, so the orbit should be assessed for lid edema, ecchymosis, and proptosis. If this occurs, lateral canthotomy, IV mannitol, and IV steroids may be required. To repair the skull base defect, good visualization is required. Mucosa adjacent to the leak site is gently reflected away. If possible, a bone graft placed

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intracranially is used. This is recommended if the defect is larger than ~0.5 cm. For this graft, a portion of turbinate bone is ideal. After placing the bone on the intracranial side of the defect, fibrin glue (or similar material) and fascia or other soft tissue is layered on the nasal side of the defect, followed by several layers of absorbable packing material such as Gelfoam. It is helpful if the patient can emerge from anesthesia smoothly, without "bucking" and straining, and without the need for high-pressure bag-mask ventilation following extubation, to minimize chances of causing pneumocephalus. Postoperatively, once stable, a head CT is recommended, to evaluate for intracranial hematoma or pneumocephalus. If the scan reveals hematoma or significant pneumocephalus, neurosurgical consultation is necessary. Otherwise, postoperative management should include head of bed elevation, bed rest for 2 to 3 days, and stool softeners. Most surgeons recommend antibiotic prophylaxis with Ancef (GlaxoSmithKline, Brentford, Middlesex, UK) or clindamycin. The use of a lumbar drain is debated. For a large defect, lumbar drainage to reduce CSF pressure for 3 days can be helpful.

Elective Repair of Other Anterior Skull Base CSF Leaks

The management is individualized. Depending on surgeon experience, many ethmoid or sphenoid sinus leaks can be approached endoscopically from below. Intrathecal fluorescein used intraoperatively is very helpful; the lumbar drain may be used postoperatively to reduce CSF pressure. In other cases, neurosurgical colleagues may approach the skull base defect from above; a pericranial flap can be used to close the defect.

◆ Outcome and Follow-Up

Repair of anterior skull base CSF leaks has a good success rate. Complications can include repeat leakage, infection including meningitis or abscess, encephalocele, anosmia, postoperative intracranial bleeding, or pneumocephalus.

◆ ICD-9 Code

349.81 Cerebrospinal fluid rhinorrhea

Further Reading

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3.1.5 Epistaxis

◆ Key Features

- Management, as for other emergency situations, should begin with ABCs (airway, breathing, circulation) and follow standard protocols for resuscitation and treatment.
- Address predisposing conditions.
- Accurate localizing of bleeding site is required for treatment.

The word "epistaxis" derives from the Greek *epi*, meaning on, and *stazo*, to fall in drops. A nosebleed may present as anterior (bleeding from the nostril), posterior (blood present in the posterior pharynx), or both. Accurate localization of the bleeding site is the key to treatment. It is estimated that >90% of epistaxis cases arise from the anterior nasal septum at Kiesselbach's plexus, also known as Little's area (**Fig. 3.3**). However, the nasal blood supply involves both the internal and external carotid systems and brisk bleeding can arise posteriorly. Major vessels include anterior ethmoid, posterior ethmoid, sphenopalatine, greater palatine, and superior labial arteries.

◆ Epidemiology

Most nosebleeds are self-limited, not requiring medical intervention. An estimated 45 million Americans suffer from at least minor epistaxis. In children nearly all cases are anterior, often due to digital trauma. Over age 40, the incidence of posterior bleeds rises.

◆ Clinical

Signs and Symptoms

Patients will report bleeding from the nares or the mouth. There may be an obvious antecedent nasal trauma, surgery, or foreign body reported. Hematemesis is common. On exam, blood may be fresh or clotted.

Differential Diagnosis

The existence of epistaxis is established on history and exam. A variety of underlying local and systemic conditions should be considered (**Table 3.5**). Most important to exclude is a neoplasm presenting with epistaxis. Consider juvenile nasopharyngeal angiofibroma in any teenage male with a unilateral sinonasal mass and epistaxis.

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Table 3.5 Causes of Epistaxis

Trauma	Maxillofacial fractures Foreign body Nose-picking
Neoplasm	juvenile nasopharyngeal angiofibroma Squamous cell carcinoma Inverted papilloma Mucosal melanoma Other
Systemic disease	Hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu) Wegener granulomatosis Sarcoid Coagulopathies Thrombocytopenia
Drugs	Coumadin, aspirin, Plavix, etc. Chemotherapy Intranasal illicit drug use
Infection	Tuberculosis, syphilis, rhinoscleroma, viral Other

◆ Evaluation

History

In most cases, the approach to the patient begins with a thorough history. The exception to this rule is the patient with severe active bleeding or hemodynamic instability that must first be corrected. Important historical information should include how long nosebleeds have been occurring, their frequency, whether bleeding is typically left or right-sided and anterior or posterior, how long nosebleeds last, and whether packing or cauterization has ever been required. Note any previous nasal or sinus surgeries. If recent sinonasal surgery has occurred, obtaining operative notes may be helpful. General information that is relevant includes a prior history of easy bruising or bleeding; a family history of such problems or known bleeding disorder; bleeding problems with previous surgeries or dental work; history of anemia, malignancy, leukemia, lymphoma, or chemotherapy; other systemic illnesses; or recent trauma. A complete accurate medication list is needed. Specific attention is directed at any recent use of medications that can promote bleeding, such as aspirin, other nonsteroidal antiinflammatory drugs (NSAIDs), coumadin, or Plavix (Bristol-Myers Squibb, New York, NY). Also consider vitamins such as vitamin E, and other supplements or herbs, many of which can promote bleeding. The social history is obtained. Chronic

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alcohol abuse can be related to coagulation disorders from impaired liver synthetic function as well as malnutrition and vitamin deficiencies; illicit intranasal drug use may be causative. In summary, awareness and treatment of systemic conditions may be required to obtain definitive effective management of epistaxis.

Physical Exam

Priorities with any severe epistaxis must focus on the ABCs and on resuscitation. The key to effective treatment is localization of the bleeding source. The head and neck exam should therefore focus on this goal. It is essential to have a nasal speculum and headlight, Frazier tip suctions, and oxymetazoline (Afrin; Schering-Plough Healthcare Products Inc., Memphis, TN) or Neo-Synephrine (Bayer Consumer Health, Morristown, NJ) spray (for decongestion and hemostasis). Topical 2% pontocaine can be used for intranasal anesthetic, if needed. If topical 4% cocaine is available, this is a very effective decongestant and anesthetic, used sparingly. The availability of a 4-mm rigid nasal telescope (i.e., Karl Storz 7200B; Karl Storz, GmbH, Tuttlingen, Germany) and light source is ideal, especially to localize a posterior bleed. Often, the otolaryngologist will need to first remove improperly placed or ineffective packing materials. Removal of clots with suction will facilitate identification of bleeding sites, using the equipment described above.

Imaging

Profuse bleeding in a maxillofacial trauma patient should be managed with emergent angiography. An injury to the extradural portion of the internal carotid cannot be controlled with nasal packs; most of these injuries probably exsanguinate in the field. Also, in recurrent or difficult-to-treat epistaxis, angiography may be diagnostic (to localize a bleeding source and guide definitive therapy such as ethmoid ligation) or therapeutic (to embolize an external carotid source).

Identification of an intranasal mass on exam should prompt CT or MRI scanning to fully evaluate a possible sinonasal neoplasm or inflammatory disease prior to definitive therapy. However, this should not delay acute treatment of active epistaxis.

Labs

Blood work should include a hemoglobin, hematocrit, platelet count, PT and PTT, as well as blood type and screening tests. Bear in mind that hemoconcentration in the underresuscitated patient may yield laboratory results that do not initially reflect the degree of blood loss. Platelet function studies may be helpful, although results may not be rapidly available. Platelet dysfunction is seen with NSAID use and von Willebrand disease.

◆ Treatment Options

A patient presenting to the emergency department with severe epistaxis or any suspicion of hypovolemia should have IV access, continuous cardiac monitoring, and aggressive fluid resuscitation.

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Generalizations

Correction of severe clotting disturbances will facilitate the efficacy of local therapy for epistaxis (i.e., reversal of overcoumadinization with fresh frozen plasma [FFP] or IV vitamin K; platelet transfusion in the thrombocytopenic oncology patient). Spontaneous mucosal bleeding typically occurs in patients with platelet counts $<$20,000. Control of hypertension is essential. Patients with underlying renal failure may need DDAVP (desamino-D-arginine vasopressin) to correct coagulopathy from underlying uremic platelet dysfunction. Some forms of von Willebrand disease also respond to DDAVP or desmopressin.

Specifics

Localize the source, as described above, using oxymetazoline spray, suction, a nasal speculum, and/or nasal endoscopy. Avoid removable packing unless other methods are ineffective. Most bleeds are anterior and can be treated effectively with topical vasoconstrictors and direct pressure and/or simple silver nitrate cautery sticks. A small piece of Gelfoam or Surgicel (Johnson & Johnson, New Brunswick, NJ) placed over the cauterized area is helpful. If bleeding will not respond to this, nasal packing to place pressure over the bleeding site may be required. Gelfoam, Surgicel, with or without topical thrombin, or Floseal are among the absorbable products available that will not require removal but will dissolve with nasal saline administration. Intranasal salt pork is a highly effective packing material.

For refractory bleeding, removable packing is placed. Many types are available, i.e., Merocel (Medtronic XOMED, Inc., Jacksonville, FL) sponges. Rapid Rhino (Brussels, Belgium) products, which are covered with a procoagulant and contain an inflatable balloon, are also effective. Experience has shown that an effective removable pack should be left in place for 4 days to enable healing prior to removal. Any patient with packing in the nose should be placed on systemic antibiotics with good gram-positive coverage, such as cephalexin or clindamycin, for prophylaxis against toxic shock. In the rare situation where properly placed nasal packing results in ongoing hemorrhage via the posterior nares, an Epistat (Medtronic XOMED, Inc., Jacksonville, FL) balloon pack is very effective. This device has a balloon that is inflated in the nasopharynx and a second balloon that provides pressure intranasally, and effectively tamponades a posterior bleeding vessel.

Other formal posterior packing techniques and devices are available as well. Any patient with a posterior pack requires hospital admission with continuous pulse oximetry. A patient failing or rebleeding following these maneuvers is usually sent for angiography and possible embolization. If unavailable, and/or the surgeon is confident that the feeding vessel is identifiable, clipping or cauterization of the sphenopalatine can be performed endoscopically; ligation of the internal maxillary system can be performed via a Caldwell-Luc approach and takedown of the posterior wall of the maxillary sinus; or anterior or posterior ethmoid artery ligation can be performed via an external incision. Recall the 24-12-6 mm rule to locate the anterior ethmoid, posterior ethmoid, and optic nerve along the orbital wall; however, there is substantial variation with these measurements.

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Other Treatments

Therapy for the Osler-Weber-Rendu patient is a challenge. One should rule out pulmonary or intracranial vascular malformations, which may be life-threatening, with appropriate imaging. Avoid packing. Laser [Nd:YAG (neodymium:yttrium-aluminum-garnet), Argon, KTP (potassium titanyl phosphate)] treatments at 1024 nm have been effective. Septodermoplasty remains an option in severe cases.

◆ Outcome and Follow-Up

Ongoing management of underlying medical disorders may be preventive. Moisture with nasal saline and humidification is helpful. Avoid NSAIDs.

◆ ICD-9 Code

784.7 Epistaxis

Further Reading

Cullen MM, Leopold DA. Nasal emergencies. In: Eisele DW, McQuone SJ, eds. Emergencies of the Head and Neck. St Louis, MO: Mosby; 2000:239–245
Wormald PJ. Endoscopic Sinus Surgery: Anatomy, Three-Dimensional Reconstruction, and Surgical Technique. 2nd ed. Stuttgart/New York: Thieme;2008

3.2 Rhinosinusitis

3.2.1 Acute Rhinosinusitis

◆ Key Features

- Rhinosinusitis is an inflammatory condition of the nose and sinuses.
- By definition, signs and symptoms last less than one month.
- The condition resolves with treatment; inadequate treatment may lead to disabling chronic disease.

Acute bacterial rhinosinusitis is frequently managed by primary care providers. The otolaryngologist often sees patients who are inadequately treated or who have recurrent disease. Aggressive care is necessary with immunocompromised patients. Collection of mucopus during nasal endoscopy enables one to obtain data for culture-directed antibiotic therapy. Treatment is usually medical, in the absence of orbital or intracranial complications.

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◆ Epidemiology

True incidence is difficult to establish because of some overlap with other complaints, such as upper respiratory infection or allergy. However, rhinosinusitis is among the most common of all healthcare complaints. Rhinosinusitis (all varieties) is reported to affect 31 million people in United States. An estimated 20 million cases of acute bacterial rhinosinusitis occur annually in the United States. Annual U.S. expenditure related to the primary diagnosis of sinusitis totals approximately $3.5 billion.

◆ Clinical

Signs and Symptoms

A history of acute rhinosinusitis may be suspected based on the presence of major and minor factors. Major factors include facial pain or pressure, congestion or fullness, nasal obstruction, nasal discharge, purulence, or discolored postnasal drainage, hyposmia/anosmia, purulence in nasal cavity on exam, and fever. Minor factors include headache, fatigue, halitosis, dental pain, cough, and ear pain or ear pressure/fullness.

The presence of two major factors, one major with two minor factors, or three minor factors strongly suggests the diagnosis. Fever is relatively specific to acute rhinosinusitis versus other forms of sinonasal disease. Localizing symptoms may suggest specific paranasal sinus involvement, i.e., cheek or upper dental pain with maxillary sinusitis, forehead pain, or frontal headaches with frontal sinusitis. Retroorbital or occipital pain may be seen with sphenoid sinusitis. Pervasive sinus disease can, however, remain occult with nonspecific symptoms.

Differential Diagnosis

In the early phase of the disease (first 10–14 days), the etiology is presumed to be viral. Thus, a typical viral upper respiratory infection is the main alternative diagnosis in the patient with a history of symptoms of less than 2 weeks' duration. Other entities to be considered include allergic rhinitis exacerbation, unrecognized chronic rhinosinusitis, or rare nasal manifestations of systemic disease such as limited Wegener granulomatosis or sarcoid. Other causes of localized symptoms include severe periodontal disease or recurrent migraine, which may include throbbing localized headache as well as nasal congestion. In the immunocompromised patient, a high index of suspicion for invasive fungal rhinosinusitis is critical.

◆ Evaluation

The diagnosis is generally made on the basis of major and minor factors present by history in combination with objective exam findings. Occasionally, radiographic assessment is needed.

Physical Exam

A full head and neck examination is performed, including a cranial nerve exam. It is important to exclude evidence of complicated sinusitis, such

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as orbital or intracranial extension of disease. Therefore, note is made of proptosis, periorbital edema, extraocular motility, tenderness, and meningeal signs. Nasal endoscopy should be performed after topical decongestion. Assessment includes position of the septum, presence of mucosal edema, presence, location, and quality of mucus or purulence, and the presence and quality of polyps or masses. A calcium alginate swab (calgiswab) or suction trap can be easily used to endoscopically obtain a sample of any purulence from the sinus ostia or middle meatus for culture and sensitivities.

Imaging

The diagnosis of uncomplicated acute rhinosinusitis is generally made on history and exam. If the diagnosis is uncertain, thin-section noncontrast coronal and axial CT scanning of the paranasal sinuses is the most useful study. The presence of fluid or soft tissue density opacification of a paranasal sinus is diagnostic. Mucosal thickening without obvious fluid suggests chronic disease. The bony detail of the skull base and orbits should be assessed. Bone erosion or thickening, or the presence of a sinonasal mass suggests other than acute rhinosinusitis and will prompt additional workup.

The utility of plain films is very limited. Disease of the middle meatus, infundibulum, frontal recess, anterior ethmoids and superior nasal cavity is not identifiable. Furthermore, the radiation exposure from a screening sinus CT (i.e., limited cuts) is probably comparable to a plain film series.

Labs

Unless the patient is toxic or immunosuppressed, or one suspects complications of acute rhinosinusitis (orbital or intracranial extension), blood work such as a CBC with differential is not helpful.

Microbiology

It is generally accepted that viral infection predominates for the first 10 to 14 days and then leads to sinus ostia obstruction. Inspissated mucus leads to bacterial infection. The most common organisms are *Streptococcus pneumoniae*, *H. influenzae*, and *Moraxella catarrhalis*, as well as anaerobes and other *Streptococcus* species. Drug resistance is a growing problem.

◆ Treatment Options

Uncomplicated acute rhinosinusitis is treated medically. If symptoms persist beyond 7 to 10 days, bacterial infection is likely and antibiotics are indicated. The typical duration of therapy is 2 weeks, and 4-week courses are often necessary. Whenever possible, antibiotic therapy should be culture-directed.

Adult empiric therapy published guidelines recommend (a) amoxicillin/clavulanate (1.75–4.0 g/250 mg per day; or 875 mg twice daily); (b) amoxicillin (1.5–4 g/d; or 500 mg three times daily); or (c) cefuroxime axetil (250 mg twice daily). In β-lactam–allergic patients, guidelines recommend (a) trimethoprim–sulfamethoxazole double-strength (twice daily); (b) doxycycline (100 mg twice daily); or (c) macrolide therapy (**Table 3.6**). If there is no improvement in 72 hours, or if there has been

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Table 3.6 Adult Empiric Antibiotic Therapy for Acute Rhinosinusitis

Nonallergic	Beta-Lactam Allergic
Amoxicillin (1.5–4 g/d)	Trimethoprim–sulfamethoxazole double-strength (twice daily)
Amoxicillin–clavulanate (1.75–4.0 g/250 mg/d)	Doxycycline (100 mg twice daily)
Cefuroxime axetil (250 mg twice daily)	Macrolide therapy (i.e., clarithromycin 500 mg twice daily)
If no improvement with 72 hours, or recent antibiotic use, consider fluoroquinolone such as moxifloxacin (400 mg/d); other alternatives include clindamycin and ceftriaxone.	

Note. Culture-directed treatment is highly recommended.

antibiotic use within the previous 4–6 weeks, the antibiotic should be switched to gatifloxacin (400 mg daily), levofloxacin (500 mg daily), or moxifloxacin (400 mg daily). Ceftriaxone or clindamycin and rifampin are additional options listed.

In pediatric patients, guidelines recommend (a) amoxicillin/clavulanate (90 mg/6.4 mg/kg per day); (b) amoxicillin (45–90 mg/kg per day); or (c) cefuroxime axetil (generally 30 mg/kg per day divided every 12 hours; maximum 1000 mg daily). In β-lactam–allergic patients, guidelines recommend trimethoprim–sulfamethoxazole (6–10 mg trimethoprim per kg per day divided every 12 hours), or a macrolide such as clarithromycin (15 mg/kg per day divided every 12 hours).

Additional medical therapy involves the use of oral decongestants such as pseudoephedrine (30–60 mg every 6 hours as needed). Caution should be used in patients with prostatic hypertrophy or poorly controlled hypertension. Topical decongestants such as oxymetazoline 0.05% (two sprays each nostril twice daily) can be used for 3–5 days at most to facilitate drainage. Prolonged use will cause rhinitis medicamentosa. Oral antihistamines are indicated only if the symptoms are associated with allergy exacerbation. Topical nasal steroids have been recommended recently to help decrease inflammation, and do play a prophylactic role following resolution of symptoms in patients with recurrent disease. Nasal saline is useful for thinning secretions, as is guaifenesin (600–1200 mg twice daily).

Occasionally, maxillary puncture is necessary to obtain material for culture and/or to relieve severe symptoms. This may be done via the inferior meatus or canine fossa. A common indication for this is an immunocompromised patient on multiple recent antibiotics with an occluded sinus.

◆ Outcome and Follow-Up

Acute rhinosinusitis generally resolves with appropriate medical treatment. Subacute rhinosinusitis progresses from 4 to 12 weeks and usually still resolves. Certain patients may relapse, termed recurrent acute rhinosinusitis,

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with symptoms again lasting less than 4 weeks and recurring four or more times per year. In patients with recurrent disease, CT scanning and more aggressive therapy, including systemic steroids and/or surgery, may be indicated.

Meningitis is the most common intracranial complication from acute rhinosinusitis, often from sphenoid disease. This is managed with IV antibiotics and usually surgical drainage. Orbital and intracranial complications are discussed in detail in Chapters 3.1.2 and 3.1.3.

◆ ICD-9 Codes

461 Acute sinusitis
461.8 Other acute sinusitis
461.9 Acute sinusitis, unspecified

Further Reading

Anon JB, Jacobs MR, Poole MD, et al, for the Sinus And Allergy Health Partnership. Antimicrobial treatment guidelines for acute bacterial rhinosinusitis. Otolaryngol Head Neck Surg 2004;130(1, Suppl):1–45

Thaler ER. Management of acute rhinosinusitis. In: Kennedy DW, Bolger WE, Zinreich SJ, eds. Diseases of the Sinuses: Diagnosis and Management. Hamilton, ON: BC Decker; 2001:149–154

3.2.2 Chronic Rhinosinusitis

◆ Key Features

- Chronic rhinosinusitis is an inflammatory condition of the nose and sinuses.
- By definition, signs and symptoms last 3 months or longer.
- Several subtypes exist, but their exact definitions remain controversial.

Chronic rhinosinusitis (CRS) is defined as a group of disorders characterized by inflammation of the mucosa of the nose and paranasal sinuses of at least 12 consecutive weeks duration. Acute rhinosinusitis has been defined as lasting up to 4 weeks. Subacute rhinosinusitis lasts 4 to 12 weeks. CRS occurs secondary to uncontrolled acute or subacute sinusitis and many times requires surgical intervention. Underlying factors such as allergies, cilia motility disorders, and immunodeficiency disorders should be considered in refractory patients.

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◆ Epidemiology

Rhinosinusitis (all varieties) is reported to affect 31 million people in United States. Chronic rhinosinusitis results in an estimated 24 million physician visits annually in the United States, 90% of which result in a prescription. U.S. annual expenditure related to the primary diagnosis of sinusitis totals approximately $3.5 billion.

◆ Clinical

Signs and Symptoms

As noted in Chapter 3.2.1, major factors include facial pain or pressure, congestion or fullness, nasal obstruction, nasal discharge, purulence, or discolored postnasal drainage, hyposmia/anosmia, purulence in nasal cavity on exam, and fever. Minor factors include headache, fatigue, halitosis, dental pain, cough, and ear pain or ear pressure or fullness. Two major factors, one major and two minor factors, or pus in the nose on nasal examination strongly suggests sinusitis, and treatment should be initiated. Validated quality-of-life assessment tools have indicated that CRS has a very large negative impact on patients.

Differential Diagnosis

Intranasal neoplasia, benign or malignant, may present similarly to inflammatory disease. Neoplasms include papillomas (inverting, cylindrical), squamous cell or adenocarcinomas, salivary tumors, sarcomas, mucosal melanoma, schwannomas, osteomas, angiofibroma, and ethesioneuroblastoma. Other entities to be considered include nasal manifestations of systemic disease such as limited Wegener granulomatosis, Churg-Strauss syndrome, sarcoid, tuberculosis, leprosy, or syphilis. Other causes of localized symptoms include severe periodontal disease or recurrent migraine, which may include throbbing localized headache as well as nasal congestion (primary care providers may label cephalgia as "sinus headaches," independent of the presence of active sinonasal disease). Temporomandibular joint (TMJ) disorders may cause "sinus" headaches. Rhinitis medicamentosa is common and patients do not always admit to use of nasal decongestants. In the immunocompromised patient, a high index of suspicion for invasive fungal rhinosinusitis is critical. In certain clinical situations, consider Churg-Strauss syndrome (vasculitis, asthma, eosinophilia), eosinophilic granuloma spectrum (Langerhans cell histiocytosis), T-cell lymphoma (formerly considered midline lethal granuloma), rhinoscleroma (caused by *Klebsiella rhinoscleromatis*), or rhinosporidiosis (caused by *Rhinosporidium seeberi*) endemic in India and Sri Lanka.

◆ Evaluation

Controversy exists with regard to definitions and diagnoses of all forms of rhinosinusitis. A diagnosis is generally made on the basis of history in combination with objective exam findings and radiographic assessment, usually CT scanning (**Table 3.7**). As listed above, certain major and minor factors, when present, can strongly suggest the presence of disease.

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Table 3.7 Exam Findings in Chronic Rhinosinusitis

CT Findings	Nasal Endoscopy Findings
Mucosal thickening	Discolored nasal drainage
Bone changes	Nasal polyps
Air-fluid levels	Edema or erythema, especially at middle meatus or ethmoid bulla

Physical Exam

A full head and neck exam is performed, including a cranial nerve exam. It is important to exclude evidence of complicated sinusitis, such as orbital or intracranial extension of disease. Therefore, note is made of proptosis, periorbital edema, extraocular motility, tenderness, and meningeal signs. Rigid nasal endoscopy should be performed after topical decongestion. Assessment includes position and integrity of the septum, presence of mucosal edema, presence, location and quality of mucus or purulence, and the presence and quality of polyps or masses. A calgiswab or suction trap can be easily used to endoscopically obtain a sample of any purulence from the sinus ostia or middle meatus for culture and sensitivities. The otolaryngologist possesses unique expertise in nasal endoscopy, and this procedure should be part of the exam of all patients presenting with sinonasal complaints.

Consider olfactory testing (especially in patients who note hyposmia/anosmia as a complaint), such as the Smell Identification Test (Sensonics, Inc., Haddon Heights, NJ), formerly called the UPSIT.

Imaging

Thin-section noncontrast coronal and axial CT scanning of the paranasal sinuses is the most useful study. CT staging systems (i.e., Lund-Mackay) have been proposed and may be useful for research or tracking disease over time. CT findings correlate poorly with patient's symptoms. The presence of fluid or soft-tissue density opacification of a paranasal sinus is diagnostic. Mucosal thickening without obvious fluid suggests chronic disease. The bony detail of the skull base and orbits should be assessed. Bone erosion or thickening, or the presence of a sinonasal mass suggests other than acute (chronic) rhinosinusitis and will prompt additional workup. Fungal disease presenting as allergic fungal rhinosinusitis will appear as heterogeneous density. Evidence of expansile disease with bone thinning is seen with allergic fungal rhinosinusitis, mucocele, and low-grade neoplasms. Bony erosion should raise concern for malignancy, and is also seen with inflammatory disease such as Wegener granulomatosis. Bony thickening is seen with long-standing inflammatory disease.

MRI is helpful for assessing intracranial extension. Fungus may appear as absent signal with both T1 and T2 sequences. However, at present MRI is not recommended as an alternative to CT for routine diagnosis of chronic rhinosinusitis because of its excessively high sensitivity and lack of specificity. The utility of plain films is very limited.

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Labs

Unless the patient is toxic, immunosuppressed, or one suspects complications of rhinosinusitis (orbital or intracranial extension), blood work such as a CBC with differential is not helpful. Bacterial cultures from sinus ostia are useful. The role of fungi is controversial. Fungi are ubiquitous; topical or systemic antifungals are not used unless tissue-invasive fungal infection is present (i.e., in immunosuppressed patients). The role of virus in chronic rhinosinusitis is unclear and has not been well studied.

Microbiology

Inspissated mucus leads to bacterial infection. Importantly, organisms differ from those seen with acute rhinosinusitis. Common microbes include *S. aureus*, coagulase-negative *Staphylococcus*, and anaerobic and gram-negative species. Most infections are polymicrobial. Antimicrobial resistance is common.

Pathogenesis and Classification

Etiology is multifactorial, involving environmental, local host, and general host factors. Pathogenic environmental factors include viral infection, pollution, smoking, and allergy. Smoking is the single most significant factor in recurrent disease; some prominent surgeons will not perform elective sinus surgery on smokers, knowing that disease will recur. Host factors include atopy, immune deficiency, mucociliary clearance problems such as ciliary dyskinesia or cystic fibrosis (probably even subclinical forms of chloride transporter dysfunction), airway hyperactivity, and reactivity to fungus.

Subtypes of chronic rhinosinusitis have been proposed based on inflammatory infiltrate (eosinophilic, neutrophilic, or other) or on various pathophysiologic mechanisms (extrinsic factors, intrinsic factors). The clinical utility of classification has been hampered to some degree because of overlap in signs and symptoms. However, categories include chronic rhinosinusitis with or without polyps; chronic rhinosinusitis with eosinophilic mucin; allergic fungal rhinosinusitis (based on the presence of eosinophils and atopy to fungus); and Samter's triad (ASA triad) of allergy to aspirin, with asthma, and nasal polyps. CRS is extremely common in patients with AIDS.

◆ Treatment Options

Medical

Uncomplicated chronic rhinosinusitis should be treated medically prior to considering surgery (**Table 3.8**). There is considerable debate regarding what constitutes "maximal medical therapy." Treatment is aimed at infection, inflammation, and underlying factors. A course of systemic steroid tapered over one month combined with oral antibiotic therapy has been found useful. Use Medrol 32 mg orally every day for 1 week, then taper to 32 mg every other day for 1 week, 24 mg every other day for 1 week, 16 mg

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Table 3.8 Treatment Strategies in Chronic Rhinosinusitis

Oral steroids	Medrol 32 mg daily × 7 days then taper
Oral antibiotics	Culture-directed whenever possible
Topical nasal steroid sprays	Mometasone, fluticasone, other
Topical nasal steroid irrigation (off-label use)	Pulmicort respules (one respule contents added to one bottle NeilMed Sinus Rinse nasal saline) twice daily
Topical nasal antibiotic irrigation, useful for methicillin-resistant *S. aureus* (off-label use)	2% mupirocin ointment 22 g in 1 L of normal saline using 50 mL per nostril daily twice daily for 10 days
Surgery	Endoscopic (forceps, microdébrider, balloon catheter dilation) Open (trephination, frontal obliteration, other)

every other day for 1 week. Whenever possible, antibiotic therapy should be culture directed. Empiric choices include ceftin 250 to 500 mg orally twice a day and Levaquin (Ortho-McNeil-Jansson Pharmaceuticals, Titusville, NJ) 500 mg orally daily. Antibiotic treatment for CRS may be prolonged. At the end of this treatment, a CT scan is repeated (with image-guidance protocol, if surgery is likely). If symptoms and/or exam and CT findings are not significantly improved, surgery is considered.

Additional useful medical therapy may include guaifenesin 600 mg to 1200 mg orally twice a day and saline nasal rinses. Methicillin-resistant *S. aureus* (MRSA) cultured from nasal endoscopy specimens has been seen at initial otolaryngology visit. Nasal irrigation with mupirocin ointment 22 g in 1 L of normal saline using 50 mL per nostril daily for 10 days appears beneficial (this is off-label use). Topical decongestants such as oxymetazoline 0.05% (two sprays each nostril twice daily) can be used for 3 to 5 days at most to facilitate drainage. Prolonged use will cause rhinitis medicamentosa. Oral antihistamines are indicated only if the symptoms are associated with allergy exacerbation. Topical nasal steroids have little role acutely; however, they play a prophylactic role following resolution of symptoms in patients with recurrent disease. Pulmicort (AstraZeneca Pharmaceuticals, LP, London, UK) respules [one respule contents added to one bottle NeilMed Sinus Rinse (NeilMed Pharmaceuticals, Inc., Santa Rosa, CA) nasal saline] for topical nasal irrigation twice daily has been used in some patients with CRS with polyps as a systemic steroid-sparing approach (this is off-label use).

Surgical

Endoscopic sinus surgery has become the standard technique for addressing nonneoplastic sinonasal disease. It is also being used for selective neoplastic cases. Open techniques may be necessary in certain cases; for example, open drainage of orbital subperiosteal abscess, or frontal sinus trephination

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Table 3.9 Preoperative Review of Coronal CT Scan

Shape and integrity of skull base	Height of ethmoid cells (in relation to top of maxillary sinus posteriorly–number of cells before skull base)
Shape and integrity of orbit	Sphenoid sinus anatomy: septations, carotids, optic nerves
Position of uncinate	Anatomic variations: Onodi cell (overriding posterior ethmoid) Concha bullosa Infraorbital ethmoid
Location of anterior ethmoid artery	Hypoplastic maxillary sinus

in combination with endoscopic surgery. Preoperative review of the coronal CT scan is critical (**Table 3.9**). Pre- and postoperative care is important for success. Pretreat with Medrol 32 mg orally daily for 5 days prior to surgery and taper slowly postoperatively, depending on endoscopic exam. Tobacco cessation is mandatory to long-term success. The role and extent of office postoperative débridement is debated.

Endoscopic surgical instrumentation may include through-cutting forceps or powered microdebriders. A recent advance has been the development of tissue-sparing instruments for balloon sinus dilation over a guide wire (Acclarent, Menlo Park, CA). At the time of this writing, 2-year efficacy data for the use of sinus balloon dilation has been published. Currently, ethmoid disease cannot be addressed with balloon instrumentation.

◆ Outcome and Follow-Up

Chronic rhinosinusitis patients require long-term ongoing care, often including the allergist, rhinologist, and primary physician. Cumulative systemic steroid exposure must be carefully followed to prevent side effects. There is a high rate of revision surgery in CRS patients, especially those with extensive nasal polyposis. It is important for patients to understand that they have a chronic disease condition involving the upper airway, and that curing this chronic condition may not be achievable. However, effectively managing symptoms, minimizing disease morbidity, and improving quality of life are reasonable goals. Orbital and intracranial complications are discussed in detail elsewhere in this book.

◆ ICD-9 Codes

473 Chronic sinusitis
473.8 Other chronic sinusitis
473.9 Unspecified sinusitis (chronic)

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Further Reading

Benninger MS, Ferguson BJ, Hadley JA, et al. Adult chronic rhinosinusitis: definitions, diagnosis, epidemiology, and pathophysiology. Otolaryngol Head Neck Surg 2003;129(3, Suppl)S1–S32

Clerico DM. Medical treatment of chronic sinus disease. In: Kennedy DW, Bolger WE, Zinreich SJ, eds. Diseases of the Sinuses: Diagnosis and Management. Hamilton, ON: BC Decker; 2001:155–168

3.3 Rhinitis

3.3.1 Nonallergic Rhinitis

◆ Key Features

- Many physicians fail to recognize nonallergic rhinitis (NAR), instead treating all rhinitis as allergic.
- Even when allergy has been ruled out, these patients may be diagnosed with "vasomotor rhinitis" (VMR), often a wastebasket diagnosis. Further workup is often not performed.
- Because NAR encompasses a variety of etiologic factors, careful diagnosis and directed therapy is key to satisfactory treatment.

Rhinitis is defined as inflammation of the nasal lining. It is characterized by symptoms of nasal congestion, rhinorrhea, sneezing, and/or nasal itching. Rhinitis is broadly classified into allergic (AR) and nonallergic (NAR) categories, based on skin testing or in vitro tests for allergen-specific IgE. "Mixed rhinitis" refers to the presence of both allergic and nonallergic components and treatment should address both components.

◆ Epidemiology

In the United States, NAR affects 19 million people, and an additional 26 million experience mixed rhinitis. NAR disproportionately affects females suggesting hormonal influences. It more commonly affects people older than 60 years of age.

◆ Clinical

Signs and Symptoms

Often, VMR is associated with nasal obstruction and thickened postnasal drip. In addition, it can presents with copious, watery anterior rhinorrhea, often triggered by changes in temperature, alcohol use, or exposure to odors and aromas.

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Other common symptoms of NAR include sneezing, congestion, and itching, which can be triggered by inhaled agents, cigarette smoke, foods, chemicals, and medications. All can have an irritant effect on the nasal mucosa.

Differential Diagnosis

NAR is diagnosed after AR has been excluded. Stimulation of the nasal mucosa due to endogenous and exogenous agents causes symptoms that characterize rhinitis. In addition, NAR can be exacerbated by hormonal influences such as those experienced during pregnancy, endocrine disturbances such as hypothyroidism, or autonomic dysfunction.

◆ Classification

NAR can be classified into the following subtypes: occupational (irritative–toxic), hormonal, drug-induced, gustatory, inflammatory (eosinophilic), and vasomotor. Rhinitis due to vasculitides and granulomatous diseases (Wegener, sarcoidosis, etc.) is a separate category.

Occupational Rhinitis

Occupational (irritative–toxic) rhinitis is caused by inhalant irritants or toxic agents including chemicals, solvents, and cigarette smoke. Cigarette smoking is the most common cause, and occupational exposure the next leading cause of this subtype. Cessation and adequate protection from these stimuli result in control of symptoms.

Hormonal Rhinitis

The most commonly reported hormonal rhinitis is rhinitis of pregnancy, seen in 22% of nonsmoking women and in 69% of smokers. Rhinitis of pregnancy is most common during the later stages of pregnancy. Rhinitis of pregnancy resolves within 2 to 4 weeks of delivery.

Drug-Induced Rhinitis

Many common medications have physiologic side effects on the nose resulting in nasal congestion. These medications include angiotensin-converting enzyme (ACE) inhibitors, β-blockers, oral contraceptives, antipsychotics, and recently, phosphodiesterase type-5 inhibitors. Some patients may have a non-IgE-mediated sensitivity to NSAIDs and aspirin.

Rhinitis medicamentosa is most commonly associated with prolonged use of topical vasoconstrictive nasal sprays such as oxymetazoline. Prolonged use of these medications results in tachyphylaxis, and cessation of use is associated with rebound nasal congestion, which can be severe and refractory to treatment.

Gustatory Rhinitis

Gustatory rhinitis is characterized by watery, often profuse rhinorrhea that develops with ingestion of food. Anticipation of eating or even the smell of food may precipitate clear nasal drainage. The response is mediated by aberrant parasympathetic stimulation of nasal secretomotor fibers on stimulation of salivary secretomotor activity.

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Nonallergic Rhinitis with Eosinophilia

Nonallergic rhinitis with eosinophilia (NARES) was originally described as a constellation of perennial sneezing attacks, profuse watery rhinorrhea, and nasal pruritus, as well as nasal congestion in patients who showed a lack of evidence for allergy on skin testing or in vitro testing for specific IgE and who had greater than 20% eosinophils on nasal smears.

Vasomotor Rhinitis

VMR is the most common form of chronic NAR. VMR is a diagnosis of exclusion. VMR is widespread, especially in the elderly, and may be frustrating to treat.

◆ Evaluation

History

Family and personal history of atopy or allergies, medications, abnormal hormonal status, pregnancy, smoking, and occupational exposure are noted.

Physical Exam

The diagnosis of rhinitis involves a complete head and neck examination, as well as a nasal endoscopy. The size of the turbinates and the degree of nasal airway impairment are noted. The reversibility of this hypertrophy is gauged after spraying with topical decongestants. The appearance of the nasal mucosa and secretions is evaluates. Allergic mucosa is classically blue-gray and boggy, whereas erythematous, inflamed mucosa is seen in rhinitis medicamentosa, irritative rhinitis, and granulomatous rhinitis.

Labs

AR should be ruled out by skin allergy testing or by in vitro tests quantifying specific IgE levels. NAR eosinophilic sinusitis can be ruled out by nasal wash cytology or by nasal brushings for eosinophils.

Other Tests

Anterior rhinomanometry, acoustic rhinometry, and nasal peak flows are supplementary tests more commonly used as research tools and not routinely used for diagnosis. Other available tests include serum mediator levels, tests for complementary activation, and nasal lavage studies. Nasal lavage studies of the total protein and albumin concentration show significantly higher levels in AR patients as compared with NAR patients and healthy control subjects, likely due to increased vascular permeability.

◆ Treatment Options

Medical

Conservative management should be attempted first. This includes avoidance of irritating substances, cold weather, offending foods, and wine. Regular physical exercise also increases sympathetic input to the nose, correcting for

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the underlying loss of sympathetic tone. Underlying diseases and medications should be evaluated. Topical anticholinergics, antihistamines, and steroids are commonly used to treat NAR.

Azelastine is approved for use in both AR and NAR. Azelastine is an H1-receptor antagonist. It also inhibits synthesis of leukotrienes, kinins, and cytokines. It also suppresses intercellular adhesion molecules expression and superoxide free radical generation. This antiinflammatory effect provides relief in NARES and VMR. The antihistaminic effect decreases mucosal edema, prostaglandin production, and stimulation of irritant receptors.

Anticholinergic medications provide relief from rhinorrhea in NAR. Ipratropium bromide is a topical anticholinergic with uncommon systemic side effects. Nasal steroid sprays can also control nasal congestion and rhinorrhea effectively.

Vasculitides and autoimmune granulomatous rhinitis (Wegener granulomatosis, sarcoidosis, Churg-Strauss disease) are treated with immunosuppressive drugs.

Surgical

The inferior turbinates are lined with vascular mucosa with mucous and serous glands and also contain venous sinusoids surrounded by smooth muscle fibers under autonomic control. Inferior turbinate hypertrophy unresponsive to medical therapy can be treated surgically with turbinate reduction by a variety of techniques.

Vidian neurectomy and sphenopalatine ganglion block have also been used for intractable VMR. Botox injection to the sphenopalatine ganglion has also been recently described.

◆ Outcome and Follow-Up

If left untreated, patients continue to suffer from detrimental effects on quality of life. Lifestyle modifications and pharmacologic interventions can be useful, but some forms of NAR, especially in the elderly, are particularly challenging to manage. Once symptoms are adequately controlled, patients can be managed with semiannual or annual follow-up.

◆ ICD-9 Code

472.0 Chronic rhinitis (excludes allergic rhinitis)

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3.3.2 Allergy

◆ Key Features

- An allergy is an immune response with deleterious effects.
- Emergencies include anaphylactic shock and angioedema.
- Signs and symptoms often involve otorhinolaryngologic manifestations.

The immune system functions to protect a host from foreign antigens. An allergic reaction is an immune response that causes undesirable effects. Examples of allergic disease of importance to the otolaryngologist are allergic rhinitis, angioedema, latex allergy, and anaphylaxis.

◆ Epidemiology

An estimated 17% of the population is affected by allergic disease.

◆ Clinical

Important to the clinical management of allergic disease is an understanding of the four types of allergic reactions, as described by Gell and Coombs (**Table 3.10**). Type I is an immediate IgE-mediated reaction (e.g., urticaria or anaphylaxis); type II is an IgG- or IgM-mediated cytotoxic reaction (e.g., transfusion reaction); type III is a response due to immune complexes, usually IgG (e.g., glomerulonephritis); type IV is a delayed hypersensitivity reaction mediated by T cells (e.g., poison ivy rash, granuloma formation). Inflammatory mediators such as histamine, leukotrienes, and cytokines mediate the effects of an allergic reaction.

Table 3.10 Gell and Coombs Classification of Allergic Reactions

Type	Reaction
I Immediate IgE response, mast cell degranulation	Response within minutes; may include wheezing, urticaria, rhinorrhea, angioedema, anaphylaxis
II Cytotoxic IgG/IgM reaction	Examples: transfusion reaction with hemolysis; hyperacute graft rejection
III Immune complex reaction, IgG	Often delayed; may affect various tissues Example: glomerulonephritis, arthritis
IV Cell-mediated, T-cell response	Example: poison ivy, granuloma formation

Abbreviation: Ig, immunoglobulin.

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Signs and Symptoms

Signs and symptoms may vary widely depending on the allergen and exposure. Nasal effects include sneezing, rhinorrhea, itching, obstructive edema, and supratip crease with so-called allergic salute. Ophthalmologic effects include conjunctival injection, itching, elongated lashes, and allergic shiners. Other airway effects may include chronic mouth-breathing, globus sensation with frequent throat clearing, cobblestone appearance to the posterior pharynx, coughing, and wheezing.

Acute allergic emergencies include anaphylaxis and angioedema. Anaphylaxis may present with rapid onset of bronchospasm, laryngeal edema, cough, stridor, itching, urticaria, tachycardia, nausea, and initial hypertension followed by hypotension and cardiovascular collapse. Death may occur rapidly due to airway obstruction or cardiovascular collapse (shock). Angioedema may present with swelling of the lips, oral cavity, tongue, and/or larynx. Progression may be unpredictable and rapid, causing death secondary to airway obstruction.

Differential Diagnosis

Patients presenting with complaints or findings related to possible allergic etiologies may be complex. Considerations include inhalant allergy, food allergy, side effects of medications such as ACE inhibitors, immune disorders such as C1 esterase inhibitor deficiency, various immune deficiencies, or infection.

◆ Evaluation

Workup must be tailored to the urgency of the situation. In an emergency, evaluation may be limited to a primary assessment, vital signs, and an assessment of the upper airway followed by immediate treatment directed at angioedema or anaphylactic shock. The elective setting enables one to perform a thorough history and physical examination along with appropriate ancillary testing.

Physical Exam

A full head and neck exam is standard. Auscultation of the lungs for wheezing is performed. Note evidence of chronic allergy, such as allergic shiners, supratip nasal crease, as well as turbinate edema, nasal mucus, postnasal drainage, and posterior pharyngeal cobblestoning. As for laboratory testing, a scraping of the inferior turbinate for cytology may suggest allergic disease–presence of eosinophils, or nasal mastocytosis. Consider C1 esterase inhibitor levels in cases of recurrent angioedema.

Other Tests

Specific allergy testing may be performed either via blood sample for RAST or via skin testing. RAST (a radioallergosorbent assay) is useful as a screening for allergen-specific IgE, but is insufficient to begin immunotherapy. Skin testing, to elicit a wheal and flare response to specific allergens, can be performed either via the prick test or the serial dilution endpoint titration (SDET) method, endorsed by the American Academy of Otolaryngic Allergy.

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SDET provides quantitative data useful for safely beginning immunotherapy. Some allergists use the results of RAST testing to prescribe immunotherapy. Details of skin testing are beyond the scope of this book; instead see *Quantitative Skin Testing for Allergy: IDT and MQT* by Bradley Marple and Richard Mabry (see Further Reading on next page).

◆ Treatment Options

Anaphylaxis

Immediate treatment for anaphylaxis includes airway and circulatory support. Airway support with supplemental oxygen, intubation, or a surgical airway may be indicated. Epinephrine 0.3 mg intramuscularly (IM) or subcutaneously (SC) repeated every 10 minutes up to 1 mg is given (adult dose.). Two large-bore IVs should be placed. IV crystalloid bolus, IV diphenhydramine 50 mg, IV H_2 blocker, and IV dexamethasone 8 mg are administered. ICU observation is planned, with further circulatory management with dopamine drip as indicated.

Angioedema

Airway obstruction must be anticipated. IV access and continuous airway monitoring are mandatory. IV diphenhydramine 50 mg, IV H_2 blocker, and IV dexamethasone 8 mg are administered. If airway obstruction is significant, if swelling persists, or if swelling worsens despite medical treatment, elective intubation is performed via an awake fiberoptic nasotracheal technique, with a tracheotomy tray available. Direct laryngoscopy is contraindicated, as sedation and paralysis may lead to an inability to mask ventilate, and tongue edema will interfere with visualization of the glottis.

Chronic Allergy Management

Pharmacotherapy includes H_1 blockers, topical nasal steroids, oral steroid tapers used sparingly, topical nasal cromolyn, leukotriene inhibitors, oral decongestants, and topical decongestants used for a maximum of 3 consecutive days to avoid rhinitis medicamentosa. Immunotherapy for desensitization may be indicated, based on results of skin testing. Environmental measures are critical, in terms of avoidance of exposure to allergen such as dust mites, molds, pets, etc.

◆ Outcome and Follow-Up

Routine follow-up for response to pharmacotherapy and for immunotherapy is indicated. Consultation with a pulmonologist for management of lower airway disease may be helpful.

◆ ICD-9 Codes

995.3 Allergy unspecified
477.0 Rhinitis

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3.4 Inverted Papillomas

◆ Key Features

- An inverted papilloma is a soft tissue neoplasm often arising from the lateral nasal wall.
- It is benign, but can be locally aggressive.
- Malignant degeneration can occur.

An inverted papilloma is considered a benign neoplasm. However, reports suggest that up to 10 to 20% of cases can contain, or degenerate to, in situ or invasive squamous cell carcinoma. Thus, complete surgical removal is the treatment of choice.

◆ Epidemiology

Males are affected three times more frequently than females. The tumor is more common in older adults.

◆ Clinical

Signs

The patient presents with a polypoid unilateral intranasal mass. Any patient presenting with a unilateral nasal mass should raise suspicion for neoplasm. Allergic fungal sinusitis may present unilaterally as well.

Symptoms

The patient has a unilateral nasal obstruction, with or without sinusitis. Rhinorrhea and/or epistaxis may occur. Histologically, an inverted papilloma consists of infolded epithelium that may be squamous, transitional, or respiratory.

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Differential Diagnosis

The differential diagnosis includes an inverted papilloma, fungiform papilloma (often arises from anterior nasal septum), cylindrical papilloma (often arises from lateral wall; rare tumor), minor salivary gland benign or malignant tumors, lacrimal sac tumors, esthesioneuroblastoma, carcinoma such as squamous cell carcinoma or sinonasal undifferentiated carcinoma, mucosal melanoma, chondrosarcoma, angiofibroma, inflammatory nasal polyp, allergic fungal rhinosinusitis. Other tumors include schwannomas, hamartomas, giant cell granulomas, neurofibromas, and chondromyxoid fibromas.

◆ Evaluation

History

A standard history is taken.

Physical Exam

A full head and neck exam is done, with attention paid to the cranial nerves. Nasal endoscopy with a rigid 0- or 30-degree endoscope will be useful to assess the intranasal extent and location of soft tissue mass. After imaging excludes possible encephalocele, a biopsy may be obtained in the office or operating room.

Imaging

CT provides bony detail, and is reviewed with attention to erosive changes at the orbit and skull base. MRI is helpful in assessing soft tissue tumor extent and can distinguish between inspissated secretion and tumor. CT will often overestimate tumor extent. Images should be reviewed in axial, coronal, and sagittal planes.

◆ Treatment Options

Medical

Definitive treatment requires surgery. Radiotherapy is ineffective and may in fact induce carcinoma.

Surgical

Complete tumor excision is required. Inadequate removal can lead to recurrence, and malignant transformation can occur. Therefore, as summarized by Myers et al, the surgical approach for the management of this tumor must allow (1) adequate exposure for complete removal, (2) an adequate view of the cavity for postoperative examination, and (3) acceptable cosmetic and functional results. Two approaches are commonly employed at present: the open approach via lateral rhinotomy for medial

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maxillectomy, and the endoscopic approach. Although there has been ongoing debate, most surgeons agree that the procedure utilized should be individualized to the size and location of the tumor, and that endoscopic removal can be done in a manner that fulfills the above criteria, with a recurrence rate no higher than that for an open approach, in appropriate cases. A midface degloving approach can also be used, especially for tumors located inferiorly, i.e., the area of the premaxilla, nasal septum, or inferior turbinate.

Regardless of approach, intraoperative frozen pathology should be used to ensure that margins are negative for evidence of residual tumor.

Preoperative Planning

Imaging is reviewed. CT provides bony detail, and is reviewed with attention to erosive changes at the orbit and skull base. MRI is helpful for assessing soft tissue tumor extent, and can distinguish between inspissated secretion and tumor. CT will often overestimate tumor extent.

A biopsy is required prior to surgery, after imaging is reviewed. Do not biopsy a possible encephalocele; review the MRI. Many surgeons perform biopsy in the operating room combined with endoscopic assessment of the tumor, given concerns for possible hemorrhage following thorough biopsy. If the biopsy indicates malignancy, an open approach to wide resection is employed if surgery is to be done; radiation or chemoradiation may be options, depending on histology.

Open Medial Maxillectomy

A temporary tarsorrhaphy stitch with 5-0 nylon is used to protect the eye. A lateral rhinotomy incision is performed, from just above the medial canthus, along the nasal facial groove, around the ala, and, if needed, the lip may be split. The periosteum is elevated as is the periorbita. The anterior and posterior ethmoid arteries are left as skull base landmarks, indicating the superior extent of dissection. Anterior antrostomy is performed, avoiding injury to infraorbital nerve. Osteotomies are made along the nasal bone, along the floor of the nose, below the frontoethmoid suture, and at the junction of the lamina papyracea and orbital floor. Posterior attachments are divided with heavy scissors, removing the lateral nasal wall tissue block. Internal maxillary artery branches may require control for hemostasis. Mucosa is stripped from the maxillary, ethmoid, and sphenoid sinuses, which are opened. The lacrimal sac is opened and sutured to surrounding tissue. The cavity is packed with Gelfoam and antibiotic gauze, and the wound is closed.

Endoscopic Tumor Removal

The following conditions are necessary for endoscopic tumor removal: known histology, adequate imaging studies, surgeon training/experience, and adequate instrumentation. The informed consent must discuss possible conversion to external and/or transoral open procedure. The use of

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intraoperative computer-assisted surgical navigation (image-guidance) is often helpful for endoscopic tumor resection. The techniques used in standard endoscopic sinus surgery are employed, with the goal of complete tumor removal along with a margin of healthy tissue. The nose is topically decongested; an initial rigid endoscopic exam is done for tumor assessment. Local injections are performed using 1% lidocaine with 1:100,000 epinephrine into the sphenopalatine region, middle turbinate insertion, and into tumor. Resection is performed with thru-cutting instruments; the powered microdébrider is often helpful. It is possible (and typical) to remove the tumor in numerous pieces without compromising the surgery. Often, a tumor stalk can be followed to a precise site of attachment. The bone underlying this attachment site should be abraded with a diamond drill if possible. If the attachment involves a turbinate, a portion of the turbinate can be resected. Similarly, the medial wall of the maxillary sinus can be widely removed. If needed, an endoscopic medial maxillectomy may be performed. Bone cannot be examined by frozen section, although mucosa can be sent. If lateral maxillary sinus tumor extension cannot be reached endoscopically, a Caldwell-Luc anterior antrostomy can be used. Steps are taken to ensure adequate sinus drainage pathways are present. Standard packing, antibiotics, and routine endoscopic sinus surgery postoperative care are used.

An advantage of endoscopic removal is that the exact tumor attachment site is identified and can be followed endoscopically in the office for surveillance for tumor recurrence.

◆ Complications

Major surgical complications can include orbital injury, optic nerve injury, skull base injury with CSF leak, and hemorrhage.

◆ Outcome and Follow-Up

An antibiotic such as ceftin 250 mg orally twice a day or clindamycin 300 mg orally three times a day is used until any packing is removed, usually at postoperative day 3 or 4. Narcotic analgesic such as oxycodone–acetaminophen (APAP; Percocet, ENDO Pharmaceuticals, Chadds Ford, PA) 5/325 1 to 2 tabs orally every 6 hours as needed is prescribed. Patients are seen in 1 week and then 3 weeks for gentle cavity débridement. Nasal saline irrigation (i.e., NeilMed Sinus Rinse) is done twice daily. Patients are typically seen every 3 to 6 months for tumor recurrence surveillance. Overall recurrence rates as high as 20% have been reported.

◆ ICD-9 Code

212.0 Benign neoplasm of the nasal cavities

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3.5 Anosmia and Other Olfactory Disorders

◆ Key Features

- Olfactory sensory neurons situated in the nasal cavity comprise the first cranial nerve (CN I) and relay olfactory stimuli to the olfactory bulbs of the brain.
- Anosmia, or olfactory loss, can be considered to be conductive (secondary to a process causing nasal obstruction), sensorineural (secondary to a process affecting olfactory neurons or central pathways), or mixed.

The most common causes of olfactory sensory loss are rhinosinusitis, head trauma, or postviral anosmia. Presbyosmia, or age-related decline in olfactory function, is well documented in patients over age 65. Less commonly, effects of drugs or systemic illness have been reported to adversely impact olfactory function.

Anosmia is an absence of olfactory function. Hyposmia (or microsmia) is a decrease in olfactory function. Parosmia is an altered olfactory perception in the presence of stimulus, usually considered foul. Phantosmia is an olfactory perception in the absence of stimulus.

◆ Epidemiology

Approximately 2 million Americans suffer from chemosensory dysfunction. Olfactory loss is present in 1% of those under age 60, but in $>$ 50% of those over 60 years of age. Presbyosmia is present in most of those $>$ 80 years of age.

◆ Clinical

Signs

In cases of conductive anosmia, nasal endoscopy will usually reveal evidence of obstructive disease, such as mucosal edema, inflammation, mucopus,

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nasal polyps, or other intranasal mass. In sensorineural anosmia, there may be no obvious exam findings. However, intracranial lesions often cause additional neurologic defects such as altered mental status, urinary incontinence, or seizures. Neoplasms such as an olfactory groove meningioma may cause Foster-Kennedy syndrome (ipsilateral anosmia, optic atrophy and central scotoma, contralateral papilledema). Objective olfactory testing will reveal the sensory loss.

Symptoms

Many patients are unaware of significant olfactory loss. Those who rely on olfaction, such as cooks, perfumers, firefighters, or chemical workers are very bothered by hyposmia or anosmia. Acute loss may follow coup/contrecoup injury from even minor head trauma. Fluctuating loss is common with chronic rhinosinusitis. Many anosmics mistakenly complain of loss of taste (see Chapter 3.6).

Differential Diagnosis

Obstructive intranasal lesions include polyps, infectious or inflammatory disease, and neoplasia, including Schneiderian papilloma, carcinoma, esthesioneuroblastoma, mucosal melanoma, and metastases. In the absence of obstruction, a sensorineural loss is most often posttraumatic or postviral; thus the history should be corroborative. One should also consider intracranial lesions such as meningioma, neurologic disease such as multiple sclerosis or Alzheimer disease, medication side effects, or toxic exposure. Iatrogenic causes such as previous nasal or sinus surgery or prior radiation therapy should be considered. Congenital causes of anosmia include Kallmann syndrome (hypogonadotrophic hypogonadism with anosmia); if suspected, these patients should have an endocrinology consultation. Smoking has also been associated with decreased olfaction, as has the use of over-the-counter intranasal zinc-containing preparations.

◆ Evaluation

Objective Olfactory Testing

Validated and easily administered tests of olfactory discrimination are readily available, such as the Smell Identification Test (Sensonics, Inc., Haddon Heights, NJ). This is a "scratch-and-sniff" multiple choice 40-item test that detects anosmia, hyposmia, and malingering and provides scores with age-adjusted norms. More sophisticated tests of olfactory threshold for specific odorants are less commonly employed outside of research centers.

Imaging

In patients without an obvious conductive etiology or history for postviral or posttraumatic anosmia, a head CT or MRI scan is recommended to rule out an occult skull base or intracranial neoplasm. Thin-section coronal CT with bone algorithms provides excellent detail of the frontoethmoidal and cribriform plate

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region. Bone dehiscence or soft tissue mass suggests a lesion (i.e., encephalocele, esthesioneuroblastoma). MRI is the test of choice for intracranial lesions.

◆ Treatment Options

Conductive Anosmia

Medical

Medical management for infectious or inflammatory sinonasal disease includes topical nasal steroids, systemic steroid tapers, and oral antibiotics. Oral steroids in combination with culture-directed antibiotics for 4 weeks are considered by many to constitute maximal medical therapy.

Surgical

Endoscopic sinus surgery, with or without septoplasty, can be effective for relieving obstructing disease such as polyposis. Endoscopic or craniofacial approaches to sinonasal or anterior skull base tumors may be indicated.

Sensorineural Anosmia

At present, there is an absence of effective treatment options for postviral anosmia, posttraumatic anosmia, or presbyosmia. Many cases of postviral anosmia or posttraumatic anosmia may recover over 1 to 2 years, but this is difficult to predict. Many patients report parosmias early during recovery. Empiric trial of steroids is employed by many clinicians, and steroid-responsive anosmia is well documented. However, this must be weighed against the risks of steroid use. Oral zinc therapy is often recommended, but the available evidence does not support its use. Appropriate referral for suspected neurologic or systemic disease is imperative.

◆ Outcome and Follow-Up

It is critically important to educate the anosmic patient regarding the importance of working smoke detectors, natural gas detectors, and the labeling and dating of foods to avoid serious injury or death.

◆ ICD-9 Codes

781.1 Disturbances of sensation of smell and taste
951.8 Injury to other specified cranial nerves

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3.6. Taste Disorders

◆ Key Features

- The sense of taste is mediated by cranial nerves (CNs) VII, IX, and X.
- Most complaints of taste dysfunction are in fact olfactory disorders.
- Medication effects are the most commonly identifiable cause of taste dysfunction.

Taste or gustation is a proximal chemical sense. Unlike in olfaction (see Chapter 3.5), for taste the primary source of stimulus must be in physical contact with the patient. The qualitative descriptors elicited by stimulation of taste receptors include sweet, sour, salty, bitter, and umami (a Japanese word that roughly equates to “savory”).

◆ Anatomy

Epithelial taste receptor cells are located on the anterior and posterior tongue, the soft palate, and in the larynx. Taste receptor cells are arrayed in sensory end organs, the taste buds, and synapse on primary sensory neurons of the facial nerve (CN VII), the glossopharyngeal nerve (CN IX), or the vagus nerve (CN X). Taste innervation is therefore redundant; that is, a lesion of a single cranial nerve would not abolish all taste input. Taste receptor cells in taste buds turn over and are replaced by proliferative basal cells. This indicates that certain clinical conditions that damage or destroy taste receptors may cause only temporary dysfunction since the system has a capacity for repair.

◆ Epidemiology

Taste loss, whether total (ageusia) or partial (hypogeusia), is much less common than olfactory loss. In a study of a large series of patients with chemosensory complaints, only 4% were found by objective testing to have a true taste deficit; the remainder exhibited olfactory deficits. This reflects the

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fact that the experience of flavor is mediated by the synergistic stimulation of the olfactory system, the oropharyngeal somatosensory system, and the actual taste receptors of the gustatory system.

◆ Clinical

Taste dysfunction may be a loss or a phantom or altered perception (dysgeusia). Partial loss is most common, and losses are usually quality or compound specific, rather than involving all taste sensation. Causes of taste dysfunction can be categorized as either drug/toxin effects or, less commonly, disease effects such as via periodontal disorders, neurologic disorders, nutritional disorders, infections, or endocrine disorders. Radiation therapy for head and neck cancer (see Chapter 5.2.2) can be another cause of taste dysfunction. Dysgeusia may (rarely) be a seizure manifestation. Iatrogenic injury to the chorda tympani during middle ear surgery can lead to gustatory complaints, such as a persistent metallic taste in the mouth, which gradually subside in most cases. Taste sensation has been shown to decline with age.

◆ Evaluation

History and physical exam should be directed at determining whether the complaint truly relates to a taste disorder, or (as is much more common) to an olfactory disorder. Objective testing for olfactory function such as the Smell Identification Test (Sensonics, Inc., Haddon Heights, NJ) can be helpful in this regard. Standardized objective testing for taste receptor function is not widely used. It is important to note that dysgeusia is usually described as one of the taste sensations (sweet, sour, etc.) or as metallic. If described as foul or rotten, this should alert the clinician to the possibility of an olfactory disorder (see Chapter 3.5).

◆ Treatment Options

If a taste disorder is identified, management is generally directed at removing or treating underlying causative factors and at supportive measures. Treating local infections such as oral thrush or periodontal disease may be helpful. Stopping possible offending systemic medications, if feasible, may be beneficial.

◆ Outcome and Follow-Up

Good oral care, hydration, nutrition, and xerostomia management with sialogogues are generally supportive measures. Radiation-induced taste dysfunction often recovers gradually over many months.

◆ ICD-9 Code

781.1 Disturbances of sensation of smell and taste

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3.7 Rhinologic Manifestations of Systemic Diseases

◆ Key Features

- Nonspecific sinonasal symptoms may be associated with a systemic disease.
- The symptoms are often managed medically to treat underlying disease.
- Radiographic and laboratory evaluation is helpful.

A broad spectrum of infectious, autoimmune, and neoplastic diseases may cause nasal obstruction as well as cosmetic deformity (**Table 3.11**).

◆ Epidemiology

Certain conditions are common in specific geographic areas, and should be considered if appropriate. Examples include rhinosporidiosis in Sri Lanka or India, and rhinoscleroma in Central America.

◆ Clinical

Signs and Symptoms

Findings with systemic disease processes involving the sinonasal region may be nonspecific and include nasal obstruction, pain, epistaxis, facial numbness, or rhinorrhea. Signs and symptoms of an underlying systemic condition, if active, may be elucidated by a thorough review of systems (i.e., fevers, chills, weight loss, dyspnea, and hematuria).

Differential Diagnosis

Systemic diseases that can involve the nose or sinuses include granulomatous disease such as sarcoidosis or Wegener granulomatosis and histiocytosis X; infections disease such as syphilis or mycobacteria (both tuberculosis and leprosy). Rhinoscleroma is seen in Central America; rhinosporidiosis is seen in India and Sri Lanka. Neoplastic disease such as T-cell lymphoma

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Table 3.11 Systemic Diseases with Sinonasal Manifestations

Condition	Diagnostic Findings	Treatment
Wegener disease	Septal crusting, perforation, chronic sinusitis, "saddle deformity" (+) c-ANCA Biopsy: granuloma, vasculitis CXR; urinalysis important	Rheumatology consult Systemic steroids, cyclophosphamide, methotrexate, or trimethoprim-sulfamethoxazole
Sarcoidosis	Elevated ACE level Hilar adenopathy on CXR Nasal edema, crusting, pain, obstruction	Systemic steroids
Syphilis	(+) VDRL or RPR, FTA-ABS Nasal erosion at mucocutaneous junction, mucus, scabbing, obstruction, rarely septal smooth mass or perforation	Benzathine penicillin parenteral, or tetracycline
Rhinoscleroma	Africa, Central and South America travel Catarrhal, atrophic, granulomatous, fibrotic stages Biopsy: Mikulicz cells with intracellular Gram (−) organism	Débridement Long-term antibiotics such as rifampin, tetracycline, or streptomycin
Rhinosporidiosis	Sri Lanka, Southern India; Nasal obstruction, rhinorrhea, epistaxis, tumor-like nasal lesions; Light microscopy demonstrates organism, *Rhinosporidium seeberi*	Surgical débridement Cauterization of margins Steroid injections for recurrence
Churg–Strauss disease	Asthma, sinusitis, eosinophilia > 10%, histologically proven vasculitis, mononeuritis multiplex	Systemic steroids Cyclophosphamide Consider sinus surgery for persistent disease
Relapsing polychondritis	3 or more of the following: bilateral auricular chondritis, seronegative arthritis, nasal chondritis, ocular inflammation, audiovestibular injury May involve larynx Elevated ESR, (+) immune complex deposition on biopsy	Systemic steroids Cyclophosphamide, azathioprine, methotrexate, dapsone considered
Lethal midline granuloma	Now considered to be angiocentric T-cell lymphoma Destructive midline nasal lesion	Radiotherapy

Abbreviations: ACE, angiotensin-converting enzyme; c-ANCA, antineutrophil cytoplasmic antibody; CXR, chest x-ray; ESR, erythrocyte sedimentation rate; FTA-ABS, fluorescent treponemal antibody absorbed; RPR, rapid plasma reagin; VDRL, Venereal Disease Research Laboratory test.

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(midline lethal granuloma) affects the nasal cavity. Inflammatory/immune system disorders affecting the lower respiratory tract and sinuses, such as Churg-Strauss disease, should be considered in severe asthmatics with severe sinusitis. HIV disease can predispose to sinonasal infections; adenoid hypertrophy in adults should raise suspicion for HIV. Inflammation of nasal cartilage can be seen with relapsing polychondritis.

◆ Evaluation

Physical Exam

A full head and neck examination and a cranial nerve exam are performed. It is important to exclude evidence of complicated sinusitis, such as orbital or intracranial extension of disease. Therefore, note is made of proptosis, periorbital edema, extraocular motility, tenderness, and meningeal signs. Nasal endoscopy should be performed after topical decongestion. Assessment includes position of the septum and presence of perforation, presence of mucosal edema, presence, location and quality of mucus or purulence, and the presence and quality of masses. A calgiswab or suction trap can be easily used to endoscopically obtain a sample of any purulence from the sinus ostia or middle meatus for culture and sensitivities. Assess for adenoid hypertrophy or nasopharyngeal masses.

Imaging

Thin-section noncontrast coronal and axial CT scanning of the paranasal sinuses is the most useful study. Staging systems have been proposed and may be useful for research or tracking disease over time. Mucosal thickening or fluid levels are seen easily. The bony detail of the skull base and orbits should be assessed. Bone erosion, thickening or the presence of a sinonasal mass suggests other than acute rhinosinusitis and will prompt additional workup. Fungal disease will appear as heterogeneous density. Evidence of expansile disease with bone thinning is seen with allergic fungal rhinosinusitis, mucocele, and low-grade neoplasms. Bony erosion should raise concern for malignancy, and is also seen with inflammatory disease such as Wegener's. Bony thickening is seen with long-standing inflammatory disease. MRI is helpful for assessing intracranial extension. Fungus may appear as an absent signal with both a T1 and T2 MRI sequence. The utility of plain films is very limited.

Labs

Laboratory studies are an important component of the workup. The ACE level may be elevated with sarcoidosis; the antineutrophil cytoplasmic antibody (c-ANCA) is elevated with active Wegener disease; a purified protein derivative test may indicate mycobacterial disease; a venereal disease research laboratory (VDRL) test can exclude syphilis; erythrocyte sedimentation rate and antinuclear antibody test may indicate rheumatic inflammatory disease. A chest x-ray and urinalysis may be informative for Wegener disease. Consider HIV testing.

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Pathology

A biopsy of an intranasal mass may be helpful. However, one must exclude the possibility of an encephalocele or a highly vascular lesion such as an angiofibroma; thus imaging before biopsy is prudent. A biopsy of the margin of a septal perforation may reveal granuloma or vasculitis, or neoplasm, but frequently reveals only necrotic tissue or inflammation.

◆ Treatment Options

Medical

Medical therapy directed at the underlying systemic condition is, in general, the treatment of choice. Specific therapy depends upon the diagnosis. Wegener's disease is treated in consultation with a rheumatologist with systemic steroids, cyclophosphamide, methotrexate, or trimethoprim–sulfamethoxazole. Sarcoidosis is treated with systemic steroids. Infectious processes are managed with appropriate antibiotic therapy, ideally based upon cultures and sensitivities. Rhinoscleroma is due to *Klebsiella rhinoscleromatis* and may require aminoglycoside treatment.

Surgical

Surgical treatment of chronic rhinosinusitis due to inflammatory conditions such as Wegener disease is best performed following systemic antiinflammatory therapy, once disease is relatively quiescent, if possible. The same principle applies to surgical correction of destructive septal lesions or "saddle nose" deformity. Rhinosporidiosis may require surgical débridement.

◆ Outcome and Follow-Up

Depends upon specific diagnosis.

◆ ICD-9 Code

473.9 Unspecified sinusitis (chronic)

Further Reading

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4 Laryngology and the Upper Aerodigestive Tract

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4.0 Anatomy and Physiology of the Upper Aerodigestive Tract

The upper aerodigestive tract is composed of the nasal cavity, nasopharynx, oral cavity, oropharynx, hypopharynx, larynx, trachea, and esophagus. The complex anatomy and physiology supports basic functions in respiration, phonation, deglutition, and the special sense apparatus for the olfactory and gustatory systems. Important aspects of anatomy and physiology are reviewed here. Nasal and paranasal sinus anatomy and physiology are covered in Chapter 3.0.

◆ Oral Cavity

General

The vestibule includes the mucosal surface of the lips, buccal mucosa, and buccal/lateral surfaces of the alveolar ridges. The remainder of the oral cavity includes the more medial structures including the hard and soft palate, mobile tongue (anterior two thirds), and the oral floor. The oral floor contains the sublingual salivary glands and the openings of Wharton's ducts draining the submandibular glands are found throughout on either side of the midline. The frenulum attaches the anterior tongue to the midline oral floor. Minor salivary glands coat the oral cavity and pharynx.

Musculature

The vestibule includes the orbicularis oris, various levators and depressors, as well as the buccinator. Tongue musculature involves both intrinsic muscles and extrinsic muscles, including the genioglossus, hyoglossus, and styloglossus, all of which are innervated by the hypoglossal nerve.

Blood Supply

The lingual artery is the primary blood supply to the tongue. The external facial artery supplies the vestibule, via superior and inferior labial branches. The greater and lesser palatine foramina in the lateral hard palate house the greater and lesser palatine arteries.

Lymphatic Drainage

Primarily to submental, submandibular, and facial nodes of level 1, while the anterior tongue lymphatics drain to upper jugular nodes of level 2, often bilaterally.

Nerve Supply

The hypoglossal is the motor supply to the tongue. The lingual nerve provides sensation, and taste fibers of the chorda tympani, to the anterior two thirds of the tongue. The facial nerve is motor supply to the orbicularis oris. General sensation to the buccal mucosa is via the second division of the trigeminal nerve (V_2).

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Physiology

Taste is supplied via the chorda tympani of cranial nerve (CN) VII to the anterior two thirds of the tongue, and CNs IX and X innervate taste buds of the posterior tongue and the base of the epiglottis, respectively. On average, 1500 mL of saliva is produced daily. Detailed swallowing physiology is beyond the scope of this handbook. Briefly, swallowing is divided into active and passive phases. The active phases include a preparatory phase that involves salivation and mastication, and a second oral phase that involves bolus propulsion posteriorly. In the passive phase, CNs IX and X control involuntary laryngeal protective mechanisms and peristalsis.

◆ Pharynx

General

The pharynx extends from the skull base to C6 and is divided into the nasopharynx superior to the palate, the oropharynx extending from the palate to the hyoid and from the circumvallate papillae anteriorly, and the hypopharynx inferior to the hyoid, including the piriform sinuses, posterior wall, and postcricoid region (**Fig. 4.1**).

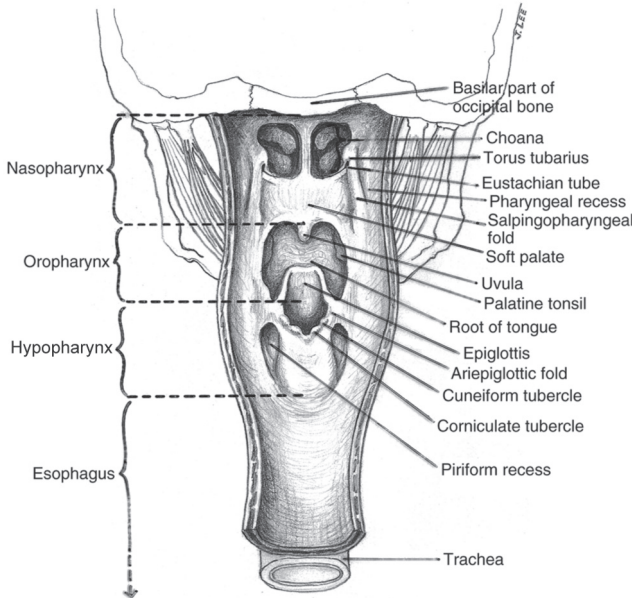

Fig. 4.1 Opened posterior view of the pharynx, demonstrating the boundaries of the nasopharynx, oropharynx, and hypopharynx. (From Van de Water TR, Staecker H. Otolaryngology: Basic Science and Clinical Review. Stuttgart/New York: Thieme; 2006: 553.)

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The cervical esophagus extends inferiorly and the laryngotracheal complex sits anteromedially. Waldeyer's ring of lymphoid tissue includes the adenoids of the nasopharynx, the palatine tonsils of the oropharynx, and the lingual tonsil lining the base of tongue. Taste buds and minor salivary glands exist in this region as well. The eustachian tubes open in the lateral nasopharynx.

Musculature

The superior, middle, and inferior constrictors surround the pharynx, enveloped by the visceral layer of cervical fascia. The palatopharyngeus and stylopharyngeus are supportive. The palatoglossus and palatopharyngeus form the tonsillar pillars.

Blood Supply

The lingual artery supplies the tongue. The tonsils are supplied by external carotid branches via the facial artery, lingual artery, lesser palatine artery, descending palatine artery, and ascending pharyngeal artery.

Lymphatic Drainage

Rich bilateral drainage supplies the base of tongue and pyriforms and drains to levels 2 through 4. The tonsils drain primarily to the jugulodigastric region.

Nerve Supply

Palatine tonsils have sensory supply from CN IX and the lesser palatine nerve. Referred otalgia is common. CNs IX and X supply motor and sensory innervation to the hypopharynx.

Physiology

Swallowing is discussed above.

◆ Larynx

General

The larynx can be considered to be a complex valve, which can regulate airflow. It is a dynamic organ that is involved with the respiratory/vocal system and the digestive tract because of its position in the pharynx. Its lumen continues superiorly with the pharynx and inferiorly with the trachea; posteroinferiorly it is separated from the pharyngoesophageal lumen. The larynx is divided into the supraglottis, which includes the epiglottis, arytenoids, aryepiglottic fold, false vocal fold, and ventricle; the glottis, which is 1 cm inferiorly and includes the true vocal folds; and the subglottis, which extends inferiorly to the inferior border of the cricoid cartilage ring (**Fig. 4.2**).

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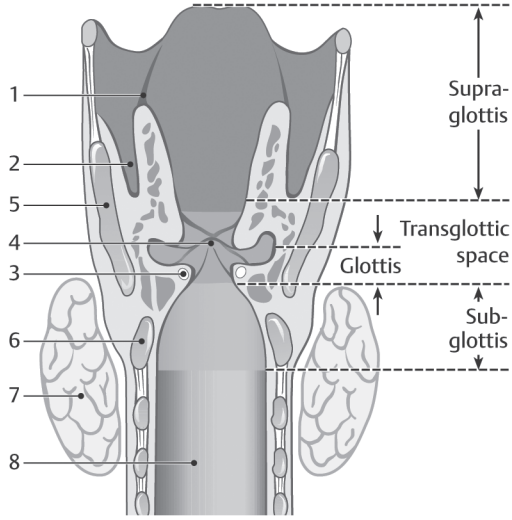

Fig. 4.2 Compartments and individual structures in the larynx. 1. The aryepiglottic fold, forming the boundary between the larynx and hypopharynx. 2. The piriform sinus, which belongs to the hypopharynx. 3. Vocal ligament. 4. Anterior commissure. 5. Thyroid cartilage. 6. Cricoid cartilage. 7. Thyroid gland. 8. Trachea. (From Behrbohm H et al. Ear, Nose, and Throat Diseases: With Head and Neck Surgery. 3rd ed. Stuttgart/New York: Thieme; 2009:293.)

Skeleton

Three unpaired cartilages form the main laryngeal structure (**Fig. 4.3**). These are the epiglottis, the thyroid cartilage (from the Greek *thyreos*, meaning shield), and the cricoid (from the Greek *krikos*, meaning ring). Three paired cartilages compose the remainder of the laryngeal skeleton: the arytenoids, the corniculate, and the cuneiform. Anterosuperiorly, the larynx articulates with the hyoid bone and inferiorly it joins the trachea. Posteriorly, the larynx meets the muscular wall of the pharynx, with the cervical vertebrae posterior to this layer. The thyroid and cricoid cartilages are hyaline cartilage, which may ossify with age. The inferior horns of the thyroid cartilage articulate with the cricoid cartilage; the paired arytenoids articulate with the cranial border of the cricoid lamina. Both of these articulations are synovial joints.

Soft Tissue

Externally, the important membranes include the thyrohyoid membrane, the cricothyroid membrane, and the cricotracheal ligament. Internally, the membranous lining of the larynx is the quadrangular membrane

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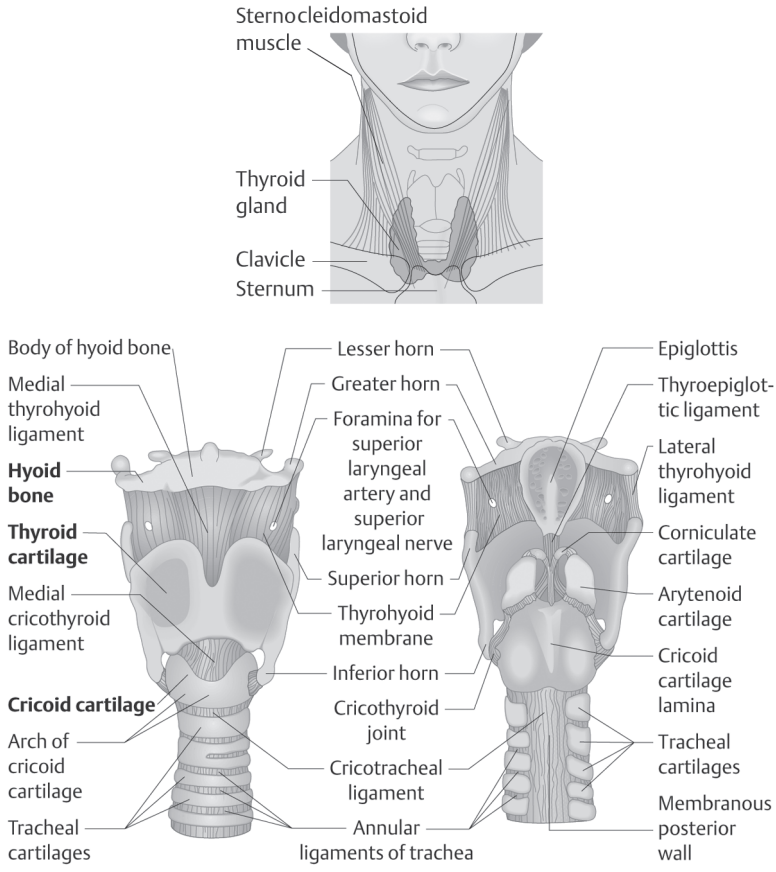

Fig. 4.3 Anatomy of the laryngeal bones and cartilage. (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:338.)

superiorly, extending to the vestibular fold or false vocal fold; and the cricovocal membrane or conus elasticus, extending from the true vocal fold inferiorly. Paired aryepiglottic folds define the opening into the laryngeal lumen superiorly. Laterally and inferiorly is the piriform sinus, which funnels food and liquid into the esophagus. The paired vocal folds extend from the vocal process of the arytenoids dorsally to the thyroid cartilage ventrally at the anterior commissure. The structure of the vocal folds includes the vocal ligament, lateral cricothyroid ligament, median cricothyroid ligament, the vocalis muscle (thyroarytenoid), and the mucosal covering.

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Musculature

The posterior cricoarytenoid exclusively opens (abducts) the vocal folds. The lateral cricoarytenoid and transverse arytenoid (interarytenoid) closes the vocal folds, along with the thyroarytenoid. The thyroarytenoid and the cricothyroid place the vocal folds under tension.

Blood Supply

Arterial supply to the supraglottis arises from the external carotid via the superior laryngeal artery. The inferior laryngeal artery supplies the subglottis arising from the subclavian artery via the thyrocervical trunk. Venous drainage is to the internal jugular and to the brachiocephalic vein.

Lymphatic Drainage

The supraglottis has rich bilateral lymphatic drainage, with connections via the preepiglottic space. The glottis has little lymphatics. The subglottis also has rich bilateral drainage via paratracheal and pretracheal channels. The lymphatic drainage influences the frequency of metastatic spread of laryngeal carcinomas based on the sites of involvement of the primary tumor.

Nerve Supply

The vagus supplies the larynx. The superior laryngeal nerve has an external branch providing motor function to the cricothyroid muscle, and an internal branch providing sensation to the supraglottis and glottis. The recurrent laryngeal nerve provides motor supply to all other internal laryngeal muscles. On the left, the recurrent nerve passes around the aortic arch; on the right, it passes around the subclavian artery. Both recurrent nerves then ascend along the tracheoesophageal groove to enter the larynx at the inferior cornu of the thyroid cartilage. Importantly, the recurrent laryngeal nerve may branch in the neck prior to entering the larynx. (A nonrecurrent right laryngeal nerve may occur if an aberrant subclavian artery is present.)

Physiology

Briefly, the basic functions of the larynx include airway protection, speech, and respiration. The larynx acts as a sphincter, in concert with pharyngeal structures to prevent airway aspiration. This is facilitated via epiglottic tilt, contraction of the aryepiglottic folds, false vocal folds, true vocal folds and adductors. Detailed discussion of the physiology of phonation is beyond the scope of this book.

◆ Esophagus

General

The esophagus lies posterior to the trachea. The esophageal opening is ~15 cm from the upper incisors at the lower border of the cricoid (C6) and the gastroesophageal junction is ~40 cm in adults. The upper esophageal

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sphincter is formed by the cricopharyngeus muscle at the esophageal opening. A middle physiologic sphincter exists at ~25 cm due to the aortic arch and left bronchus. The lower esophageal sphincter is at the gastroesophageal junction. The esophageal wall has a mucosa, submucosa, muscle layer, and outer fibrous adventitia. There are external longitudinal muscle fibers and internal circular fibers. The superior third is striated muscle, the middle third is mixed, and the inferior third is smooth muscle. The mucosa is stratified squamous, with a transition to columnar epithelium at the junction with the stomach.

Blood Supply

Superiorly, supply is via branches from the inferior thyroid artery originating from the thyrocervical trunk. In the thorax, supply is segmental via branches of the thoracic aorta or via intercostals vessels. Inferiorly, there are branches from the left gastric artery. There is a venous plexus and drainage parallels supply generally.

Lymphatic Drainage

The cervical lymphatic system includes the deep cervical and paratracheal nodes; the thoracic lymphatics are composed of the posterior mediastinal and tracheobronchial nodes. The abdominal lymphatic system incorporates the left gastric and celiac nodes.

Nerve Supply

The esophagus is served by the glossopharyngeal and vagus nerves and the sympathetics. The myenteric plexus of Auerbach lies between the longitudinal and circular muscle layers.

Physiology

The esophageal phase of swallowing occurs as the bolus passes the upper esophageal sphincter. Primary peristalsis has an initial rapid inhibitory phase followed by a longer wave of contraction. The lower esophageal sphincter relaxes to open for 6 to 8 seconds. There is also a secondary peristaltic wave in a rostral to caudal progression.

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4.1 Laryngeal and Esophageal Emergencies

4.1.1 Stridor

◆ Key Features

- Stridor is an indication of airway compromise and should be considered an emergency.
- The priorities are to identify the site of obstruction, restore adequate ventilation, and address the underlying cause.

Stridor is an exam finding defined broadly as noisy breathing due to partial upper airway obstruction, and is usually high-pitched and harsh. This is to be distinguished from wheezing, which is noise due to reversible collapse of bronchioles of the lower pulmonary airway; and from stertor, a sonorous noise that is due to collapse or obstruction at the upper pharynx, such as snoring.

◆ Clinical

The stridulous patient must be evaluated without delay, as loss of airway may progress rapidly.

Signs and Symptoms

In the adult, development of stridor may be acute or chronic. Symptoms are often nonspecific and variable. Associated symptoms will vary depending on etiology and may include dyspnea, pharyngodynia, dysphagia, odynophagia, and anxiety. Signs include audible noise with breathing and may include fever, cough, hemoptysis, and retractions. Depending on severity and acuity, the patient may be in distress, hypoxic, and may also display dysphonia.

Differential Diagnosis

The causes of stridor are numerous. It is useful to categorize based on the anatomic level of obstruction. As a generalization, inspiratory stridor correlates with supraglottic obstruction, expiratory stridor correlates with intrathoracic obstruction (trachea), and biphasic stridor suggests glottic or subglottic obstruction (**Table 4.1**). Differential diagnosis includes an obstructing neoplasm, infection with edema (i.e., supraglottitis), allergy/angioedema, foreign body, traumatic injury to the airway (i.e., thyroid cartilage fracture, large hematoma), and bilateral vocal fold immobility (i.e., postoperative iatrogenic injury). Other causes include subglottic stenosis, tracheal stenosis, and tracheomalacia.

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Table 4.1 Stridor Localization by Anatomic Site

	Retractions	Stridor*	Voice	Feeding
Naso/ oropharynx	Minimal†	Stertor†	Normal	Normal†
Supraglottis	Marked and severe	Inspiratory and high pitched	Muffled	Abnormal
Glottis/ subglottis	Mild to severe	Biphasic and intermediate pitched	Normal to very abnormal (barking cough)	Normal
Intrathoracic trachea	Mild to severe	Expiratory and low pitched	Normal (seal-like cough)	Normal

Source: Van de Water TR, Staecker H. Otolaryngology: Basic Science and Clinical Review. Stuttgart/New York: Thieme; 2006:214.
*The quality of the airway noise.
†Unless associated with complete nasal obstruction in a neonate.

◆ Evaluation

One must consider an acutely stridulous patient as a potential airway emergency; prompt evaluation is warranted.

Physical Exam

In critical care algorithms, the ABCs are followed: airway, breathing, circulation. Stridor is a reflection of airway narrowing and is therefore a priority. If the stridulous patient is not ventilating adequately (hypoxic or retaining CO_2), rapid intervention is critical. Establishment of the airway is discussed below.

History is important in guiding the exam: the timing of onset; known diagnoses, such as history of angioedema or head and neck cancers; previous head and neck surgeries (thyroid surgery, previous tracheotomy); trauma; possible foreign body aspiration; current upper respiratory infection; history of intubations; etc.

On exam, vital signs; pulse oximetry; possibly arterial blood gases; phonation; an oral and pharyngeal exam; and a neck exam for masses, edema, crepitus, or tenderness are important. Unless the adult patient is unstable or not adequately ventilating, a flexible fiberoptic nasopharyngolaryngoscopy is usually safe and extremely helpful. This exam will reveal an estimation of glottic airway diameter, vocal fold mobility, any sites of edema or mass, or the presence of an obstructing laryngeal foreign body. Pediatric patients may selectively undergo flexible fiberoptic examination. Caution must be used as the examination can precipitate further airway compromise.

Imaging

Plain films of the neck—posteroanterior (PA) and lateral views—may be informative but are being replaced by computed tomography (CT) or magnetic resonance imaging (MRI). In general, one should not send a patient

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with airway compromise for CT or MRI unless the airway has been secured; loss of the airway during imaging can be disastrous.

Labs

An arterial blood gas (ABG) test may be helpful in determining adequacy of ventilation. Less acutely, a complete blood count (CBC) with differential may be useful in a patient with infection, such as supraglottitis.

◆ Treatment Options

The goals of treatment are (1) to determine the site(s) and degree of obstruction; (2) to stabilize the airway by forced ventilation, intubation, or surgical bypass of the site of obstruction; and (3) to treat the underlying cause.

In an unstable, poorly ventilating patient, the airway must be secured. If possible, the operating room is the safest place to do this. One should approach the airway problem algorithmically, thinking ahead about possible problems (with a plan B and plan C). The algorithm will differ depending on etiology and the patient. There are useful generalizations, however. (1) It is absolutely critical that the otolaryngologist clearly and authoritatively take control of the airway management. (2) An upright, awake, spontaneously breathing patient is usually the safest situation. (3) One must be prepared for the possibility that sedating and paralyzing a stridulous patient for intubation may result in an inability to mask ventilate, precipitating an emergency. (4) With angioedema, tongue swelling usually precludes the ability to perform direct laryngoscopy.

If intubation is deemed to be impractical (i.e., fiberoptic laryngoscopy indicates that an endotracheal tube will not pass through a narrowed airway), awake tracheotomy is performed. In an emergency where the surgical airway must be most rapidly established, cricothyroidotomy is indicated.

Unless the fiberoptic laryngoscopy suggests otherwise, an awake fiberoptic nasotracheal intubation is often the procedure of choice (if the patient requires intubation). As a backup plan, one should have a Holinger laryngoscope, velvet-eye laryngeal suction, and Eschmann stylet assembled and ready to use. Often, the otolaryngologist can easily intubate a patient with these instruments. A ventilating rigid bronchoscope is also very helpful, if available. In these cases, the patient should be maintained with spontaneous ventilation; if the patient has airway masses or stenosis, then ventilating bronchoscopy can be diagnostic and therapeutic. In addition, a tracheotomy tray should be open and ready to use. Injecting the soft tissue over the cricothyroid membrane with 1% lidocaine and 1:100,000 epinephrine ahead of time will result in vasoconstriction and a much drier operative field if emergency cricothyroidotomy or tracheotomy becomes necessary.

Other strategies for difficult intubation include retrograde intubation by placing a needle and guide wire (from a central line kit) into the cricothyroid membrane or trachea and passing the guide wire up and out of the mouth. An orotracheal tube may then be blindly passed over the guide wire and into the trachea. There are other techniques, such as fiberoptic intubation through a laryngeal mask airway, video direct laryngoscopy, intubating

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endoscopes (e.g., Shikani optical stylet), or the use of a lighted stylet to be blindly introduced into the trachea.

Medically, there are helpful strategies to "buy time" or assess response to medical therapy if a patient can maintain ventilation. The patient is maintained in an intensive care unit with continuous pulse oximetry monitoring. Humidified oxygen (i.e., via face tent) will help minimize secretions. Heliox (typically 79% helium/21% oxygen mixture) has been advocated as a short-term intervention to help maximize ventilation while definitive intervention is planned. The gas functions by reducing the viscosity of the inspired air, thus reducing the mechanical work of breathing in the narrowed airway. It can be used while medical intervention is taking effect; this is an excellent means of avoiding intubation. Intravenous (IV) steroids (i.e., dexamethasone 8 mg IV every 8 hours) may reduce airway edema. In some situations, appropriate medical treatment of the underlying problem, such as infection or angioedema, can obviate the need for intubation or surgical airway.

◆ Outcome and Follow-Up

After securing the airway, appropriate management directed at the underlying problem is undertaken. This may include biopsy, treatment of infection, and laboratory or radiographic workup.

◆ ICD-9 Codes

786.1 Stridor

748.3 Other anomalies of larynx, trachea, and bronchus

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4.1.2 Laryngeal Fractures

◆ Key Features

- Airway obstruction can develop rapidly.
- Evaluate for concurrent injuries such as pneumothorax or esophageal or vascular injury.
- The treatment goals are to ensure an adequate airway, to maintain voice quality, and prevent aspiration.

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Laryngeal fractures can be mild, nondisplaced, and isolated. They may also be severe, displaced, with airway compromise and concomitant injuries to other structures of the head, neck, and chest. A fiberoptic laryngeal exam and a CT scan will usually direct care appropriately.

◆ Epidemiology

Five to 10% of traumatic injuries involve the neck. Penetrating neck injuries are more common than blunt trauma (see also Chapter 5.1.1.4).

◆ Clinical

Patients usually present with a history of blunt trauma to the anterior neck. Common mechanisms include assault, a strangling or hanging attempt, and vehicular accidents including automobiles, snowmobiles, motorcycles, all-terrain vehicles, and bicycles. A gunshot wound to the neck can result in a cricoid or thyroid fracture alone or in combination with other injury.

Signs and Symptoms

Signs and symptoms may include dysphonia, aphonia, stridor, cough, hemoptysis, dysphagia, and pain. A hallmark of severe injury is subcutaneous emphysema, as well as loss of the normal prominence of the thyroid cartilage.

Differential Diagnosis

Differential diagnosis is based on severity. The injury may be confined to soft tissue, or may include cartilage fracture, mucosal disruption, or additional injuries to the pharynx, esophagus, or major vessels.

◆ Evaluation

Physical Exam

A fiberoptic exam and CT are diagnostic. The injury may be occult. A delay in diagnosis contributes to mortality. Trauma algorithms are followed, including ABCs. In the presence of laryngotracheal cartilage disruption, intubation is generally contraindicated. An emergency airway is established via a tracheotomy. Transtracheal jet ventilation can be extremely useful while the definitive airway is being established. The remainder of the head and neck exam is directed at determining additional injuries. With a questionable laryngeal fracture, flexible fiberoptic laryngoscopy is performed. The presence of obvious endolaryngeal mucosal disruption, bleeding, or cartilage derangement suggests the injury.

Labs

Laboratory tests are not routinely indicated. When following an inpatient after a laryngeal fracture, a CBC with differential may be useful to monitor for infection and the development of mediastinitis or sepsis.

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Other Tests

Once the airway is deemed stable or stabilized, imaging should be performed. A CT scan of the neck viewed with bone windows provides excellent detail of the laryngeal framework. A chest x-ray is mandatory to evaluate for pneumothorax. A cervical esophagogram is recommended to evaluate for occult esophageal injury.

◆ Treatment Options

The acute treatment goal is maintaining or establishing the airway, as discussed above. The long-term treatment goals include maintaining an adequate airway, a satisfactory voice, and the ability to swallow without aspiration.

Larynx trauma can be categorized as mild or severe. Mild injuries include laryngeal edema, simple endolaryngeal lacerations with minimal cartilage exposure, and nondisplaced fractures. If severity is unclear, an examination under anesthesia with direct laryngoscopy and esophagoscopy is recommended. Patients with mild injuries can usually be managed nonoperatively. Treatment must include observation in a monitored bed (i.e., continuous pulse oximetry), along with intravenous (IV) steroids [Decadron (Merck & Co., Inc., Whitehouse Station, NJ) 8 mg IV every 8 hours $\times$ 3 doses], humidified air/O_2, and a soft diet.

Patients with severe mucosal injuries and/or displaced or comminuted cartilage fractures are managed surgically with low tracheotomy followed by exploration and repair. Fractures should be repaired early, ideally within 24 hours. The basic goals include closure of tracheal defects, coverage of exposed cartilage, removal of devitalized cartilage, and reduction of cartilage fractures. After tracheotomy, direct laryngoscopy and esophagoscopy are performed. If the vocal folds are significantly displaced, laryngofissure is performed with repair of mucosa. Fractures are reduced and reapproximated with Prolene (Ethicon, Somerville, NJ) suture, wire, mesh, or plating. If possible, external perichondrium should be closed. Stenting may be necessary, to prevent stenosis or anterior webbing. Indications for stenting are an area of controversy.

◆ Outcome and Follow-Up

Posttreatment follow-up includes ongoing assessment of the airway and swallowing. A barium swallow study with speech-language pathology for evidence of aspiration is important. Consideration of tracheotomy decannulation is based on standard criteria. Further surgical management of webbing or stenosis may be required, as well as long-term use of a T-tube. Patients may require voice therapy.

◆ ICD-9 Codes

807.5 Fracture of the larynx and trachea, closed
807.6 Fracture of the larynx and trachea, open

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4.1.3 Caustic Ingestion

◆ Key Features

- Perform an airway assessment and intubation if there is any compromise.
- Identify the ingested substance.
- Do an early endoscopy.

◆ Epidemiology

The incidence of caustic ingestion is estimated at 5000 to 15,000 cases annually in the United States: 53% of cases occur in children 6 years of age or less. However, only ~3% of deaths secondary to caustic ingestion occur in young children.

◆ Clinical

The patient with caustic ingestion presents with varying findings depending on the type of substance ingested and the quantity.

Signs and Symptoms

With moderate to severe injury, symptoms include oral, neck, and chest pain, and dysphagia. Young patients may drool, and older patients may spit secretions or refuse to swallow. Respiratory problems range from coughing and wheezing to stridor and respiratory distress.

Differential Diagnosis

The injury may range from mild to severe as a result of acid versus alkaline ingestion.

◆ Evaluation

It is important to obtain information about the substance ingested. Alkaline substances (lye, ammonia, soaps) cause liquefaction necrosis and deep

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penetrating injuries to the pharyngeal and esophageal wall. Ingestion of lye (drain cleaner) accounts for ~60% of cases. Acids cause a mucosal surface coagulative necrosis, and deep tissue injury is uncommon. Household bleach is usually less than 5.25% sodium hypochlorite and, therefore, usually causes only mild mucosal irritation.

Physical Exam

Assessment of the airway is the first priority. If there is evidence of airway compromise, the airway should be secured by intubation before edema worsens. This is especially important in children. A head and neck exam and skin survey for caustic burns from the spilled substance are indicated, followed by assessment of the oral and pharyngeal mucosa for injury. Blind nasogastric tube placement is avoided; emesis is not induced; charcoal gastric lavage is not indicated. In the past, the timing of endoscopy was controversial. Currently, esophagoscopy within 24 to 48 hours is recommended. Damage, in terms of an estimate of the depth of injury, is assessed at the proximal extent of injury. Additional advancement of the rigid endoscope is not performed, to avoid further injury.

Labs

Routine preoperative labs are obtained if surgery is planned. A CBC is followed if there is suspicion of mediastinitis.

Other Tests

Acutely, a contrast swallow study is not routinely indicated. However, contrast swallow studies are often used to assess the progression of stricture formation during recovery.

◆ Treatment Options

Nonoperative therapy includes hydration, acid-blocking medications, and observation. Steroids do not seem to reduce the incidence of stricture formation; some authors have suggested that they may mask the signs or symptoms of perforation. Antibiotics are often given empirically until perforation has been ruled out. An endoscopy is performed to grade the injury (**Table 4.2**). In general, the scope should not be passed beyond any segments of necrosis or concern for transmural injury. A thoracic surgery consultation is obtained for management of perforation or high-grade injuries. There has been a trend toward early surgical intervention by thoracic surgeons (within 36 hours) to treat deep esophageal injuries. For evidence of a high-grade injury on endoscopy, a feeding tube should be placed under direct vision.

◆ Outcome and Follow-Up

Mortality ranges from zero to 8% overall. Complications including mediastinitis, perforation, and stricture formation range from 10 to 20%. Stricture development generally begins at week 3 or 4; a contrast swallow study or a follow-up endoscopy is helpful at this time.

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Table 4.2 Grading of Esophageal Caustic Burns by Endoscopic Findings

1st degree	Erythema and punctuate hemorrhages Superficial mucosal injury
2nd degree	Ulceration, exudates, pseudomembranes Partial-thickness injury
3rd degree	Eschar, perforation into or thru muscular layer Full-thickness injury

◆ ICD-9 Codes

947.0 Burns of the mouth and pharynx
947.2 Burns of the esophagus

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4.1.4 Laryngeal Infections

◆ Key Features

- Infections of the larynx may be acute or chronic in nature.
- Symptoms and signs include hoarseness, pain, dysphagia, cough, dyspnea, stridor, and signs of systemic illness.
- Therapy is directed at the causative organism and is supportive of any airway issues present.
- Laryngoscopy with biopsy and/or culture may be necessary as some infections may mimic neoplasms, or require this information for a definitive diagnosis.

Laryngitis is a term used to describe any inflammatory condition of the larynx. Infectious laryngitis may be caused by viral, bacterial, mycobacterial, fungal, and even protozoan organisms. History and physical examination, including laryngoscopy, are instrumental in establishing the diagnosis. The

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immunologic state of the patient, travel history, and exposure to illness are important factors to address in the history.

◆ Epidemiology

Viral laryngitis is the most common type of laryngitis. Incidence is difficult to establish as most patients do not seek out care for acute laryngitis issues. Secondary to airway size, laryngitis may present in children with airway symptoms; adults more commonly present with hoarseness and pain complaints.

◆ Clinical

Signs and Symptoms

Viral Laryngitis

Viral laryngitis usually accompanies a viral upper respiratory infection (URI) with systemic viral syndrome. Symptoms include hoarseness, with pitch breaks, and decreased pitch; cough; and pain. Examination shows erythema and edema of the vocal folds. Agents include those causing URIs, with rhinovirus being most common. Treatment is symptomatic as this should be a self-limited disease. Therapy includes voice rest, hydration, humidification, cough suppressants, and expectorants.

Croup

Croup (laryngotracheitis) is a viral infection seen in children characterized by stridor, a "barky" cough, and fever. This illness is seen primarily in winter, but may occur year-round. The severity of the illness varies widely, and is based on the degree of subglottic edema. Parainfluenza viruses 1 and 2 and influenza A are the most common etiologic agents. The most important assessment of these patients is evaluating the respiratory status for impending airway intervention and support. Initial home management involves humidification or steam shower, although this is not evidence based. Racemic epinephrine may be useful to those children with respiratory distress, serving to rapidly decrease airway edema. Admission is no longer thought to be absolutely necessary after this treatment, as the "rebound" concern is rare. Corticosteroids have a clear role in treatment of this condition. Airway intervention including intubation or tracheotomy may be necessary if respiratory decompensation occurs, with respiratory fatigue, hypercarbia, inadequate oxygenation, or worsening neurologic status. For intubated children, an air leak should develop and indicate extubation potential, usually within 2 or 3 days.

Bacterial Laryngitis

Bacterial laryngitis is far less common than viral etiologies. Supraglottitis may involve the entire supraglottis, or more focally the epiglottis (epiglottitis). The incidence of epiglottitis in children has dramatically fallen since the introduction of the *Haemophilus influenzae* type B vaccine. Adults typically have more diffuse supraglottitis. Patients relate sore throat, fever, odynophagia, muffled voice, and dyspnea. Children may have drooling. Patients may sit in a tripod position, with the torso leaning partially forward, arms positioned at

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the sides just in front of the torso, and neck extended. Diagnosis is confirmed by the swollen, erythematous epiglottis or supraglottis seen on flexible laryngoscopy. In children, the diagnosis may best be made by lateral neck film and a "thumbprint" sign representing a swollen epiglottis, as manipulation of the airway with flexible endoscopy may precipitate airway compromise. Although historically *H. influenzae* is the most common organism in children, other organisms such as *Streptococcus pneumoniae*, *Staphylococcus aureus*, β-hemolytic *Streptococcus*, *Klebsiella pneumoniae,* and parainfluenza virus may be etiologic agents. In severe cases, a secure airway should be established, potentially in an operating room setting. Less emergent airways may be managed with hospital admission and close observation. Adults are less frequently in need of airway interventions than children. Treatment should include antibiotics that cover β-lactamase–producing *H. influenzae*, such as second- or third-generation cephalosporin, as well as corticosteroids. Epiglottis abscess is a rare complication of supraglottitis.

Fungal Laryngitis

Although fungal infections occur more commonly in the immunocompromised patient, fungal laryngitis may be seen in many varied patient populations. These infections may present as leukoplakia, either focal or diffuse. Risk factors for fungal laryngitis include those with diminished systemic immune response (e.g., immunocompromised states, diabetes, HIV, immunomodulating medications, and severe nutritional deficiencies), or depressed local immune function (e.g., prior local radiation therapy, corticosteroid inhalers, smokers). *Candida* sp. are the most common organisms, but blastomycoses, histoplasma, aspergillus, cryptococcus, and coccidiomycosis may be involved. Secondary to the infrequency of these infections and their presentation as leukoplakia, biopsy may be done. Some infections can show pseudoepitheliomatous changes and can even be mistaken for squamous cell carcinoma. Special fungal stains may be necessary. Systemic antifungal agents appropriate for the causative fungal organism are necessary. Topical antifungal medications are not curative, as they require direct contact with the organism, and the larynx is protected from exposure through protective-airway mechanisms.

Tuberculosis

Tuberculosis of the larynx may be seen with pulmonary infection (historically one of the most common laryngeal infections), but may be experienced as an isolated infection in 20 to 40%. Risk factors include exposure in endemic areas, immunocompromised states, and nursing home environments. Patients typically report hoarseness, dysphagia, odynophagia, and cough. The larynx typically is edematous and hyperemic in its posterior third, with some exophytic granular areas. Biopsy, chest radiography, and sputum cultures may be necessary for diagnosis. Treatment is with antimycobacterial drugs, with cultures helpful given the significant drug resistance that may be present.

Actinomycosis

Actinomycosis is an unusual laryngeal infection, and is known to more commonly affect the oral cavity. The fastidious, anaerobic organism is felt

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to be an intermediate, with features of bacteria and fungi. Infections may manifest as edema, abscess, and ulceration. Biopsy may demonstrate the pathognomonic sulfur granules. Treatment typically is with high-dose penicillin given IV or orally depending on the disease severity. Clindamycin is an option for penicillin-allergic patients.

Syphilis

Luetic infections were once much more common than today. Syphilis may be present in primary, secondary, or tertiary stage. Laryngeal involvement is rare and may be a component of the secondary or tertiary (gumma) phase. Caused by the *Treponema pallidum* spirochete, these infections may lie dormant for prolonged periods of time. Laryngeal involvement may present as a diffuse hyperemia, ulceration, or maculopapular mucosal rash. Laryngeal syphilis may cause fibrosis, chondritis, and scarring or stenosis. Serologic testing should include venereal disease research laboratory (VDRL) testing (100% sensitivity in secondary syphilis) with confirmation using fluorescent treponemal antibody absorption (FTA-ABS) and the microhemagglutination assay for *T. pallidum* (MHA-TP). Penicillin G is the primary drug in treatment.

Leprosy

Leprosy or Hansen disease, caused by *Mycobacterium leprae*, is very rare in the United States but is seen more often in the Indian subcontinent and Africa. Nasal infection is the primary site of infection in the head and neck, with the larynx being second. Infection favors the supraglottic, with symptoms including hoarseness, muffled voice, odynophagia, and cough. Examination may reveal erythema, nodularity of the mucosa, and ulceration. However, often the patient has minimal pain, despite the appearance of the tissue. Scarring may develop that leads to stenosis and airway obstruction. Biopsy may reveal "foam cells," with copious *M. leprae* within these cells. Long-term medical therapy is necessary, even 5 to 10 years, with dapsone and rifampin.

Other

The larynx may be involved in other systemic infections, particular viral infections such as mumps, measles, or varicella (chickenpox).

Differential Diagnosis

There are a number of causes of laryngeal infection:

- Laryngeal neoplasm
- Airway foreign body
- Autoimmune diseases
- Allergic reaction
- Angioneurotic edema

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- Subglottic stenosis
- Laryngopharyngeal reflux

◆ Evaluation

History

The key in evaluation is primarily history, including time course of the condition, exposures, comorbidities, and past medical history.

Physical Exam

A complete, thorough head and neck examination, including flexible nasolaryngoscopy, should be performed. The lungs should also be auscultated to evaluate for concomitant pulmonary issues.

Imaging

The role of imaging is limited. CT may be helpful if an abscess is suspected, or to aid in evaluating the complication of airway stenosis. If tuberculosis is suspected, chest radiography should be ordered.

Labs

Laboratory testing may include a CBC or specific serology as appropriate. In some cases (e.g., epiglottitis), laboratory testing would be contraindicated initially. Tissue biopsy and culture may be necessary to confirm a diagnosis for several reasons. First, many of these infections are uncommon; therefore, clinical experience is limited in recognizing the entity definitively. Second, the laryngeal findings may mimic squamous cell carcinoma grossly; the onus is on the clinician to evaluate for this issue. Lastly, given that the medical therapy may be prolonged and not without side-effects and medication interactions, a tissue culture assists in quality care.

◆ Treatment Options

Treatment of laryngeal infections is based on the causative agent. Viral laryngitis is managed with supportive measures. Pharmacologic treatment should be tailored to the organism implicated in the infection.

◆ Outcome and Follow-Up

Follow-up with laryngoscopy should be used to assess resolution of the infection. The frequency of these evaluations is determined by the severity of the infection, and the expected time to resolution.

◆ ICD-9 Codes

464.0 Acute laryngitis and tracheitis, no obstruction
464.3 Epiglottitis
464.4 Croup

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4.2 Neurolaryngology

◆ Key Features

- Neurolaryngeal examination is a key component in the evaluation of any voice disorder.
- Laryngeal dysfunction may be an early sign of systemic neurologic conditions, even before the manifestation of other symptoms.
- In patients with neurologic dysfunction, the other key laryngeal functions beyond phonation should also be assessed (i.e., respiration, airway protection during deglutition).
- Laryngeal electromyography may be helpful in specific clinical scenarios, but controversy exists as to its routine use in all neurologic complaints.

Focal and systemic neurologic conditions may affect the laryngeal functions. The laryngeal findings of systemic neurologic conditions may even precede their presentation in other locations. The key in accurately diagnosing these conditions is a careful, detailed history combined with a clinical voice evaluation. Direct visualization of the larynx is important, and may be best done with flexible fiberoptic laryngoscopy. Stroboscopic assessments may also be helpful in assessing the vocal fold mucosal wave, and complements the neurolaryngeal exam. The most common neurolaryngeal disorder encountered is vocal fold motion impairment (see Chapter 4.3.3). Other common disorders include spasmodic dysphonia, tremor, Parkinson disease (PD), stroke, and vocal fold dysfunction (VFD).

◆ Clinical

Signs and Symptoms

The laryngeal complaints of patients may take the form of vocal, dysphagia, airway, or a combination. Dysphagia is covered in Chapter 4.4.2. Airway symptoms may occur with bilateral vocal fold immobility or significant paresis, or with paradoxical vocal fold adduction. The features of the vocal

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concern that should be defined are onset, situational context, perceived quality, pitch, pitch control, fluidity, and stamina.

Differential Diagnosis

Neurologic issues involving the larynx may be either focal or systemic. Therefore, other neurologic features in the remainder of the body should be sought. The systemic diagnosis may have been already determined, or the vocal issue may be the first presenting sign of a new diagnosis. In general, neurolaryngeal illnesses may involve a lack of neuromuscular strength or mobility of the vocal folds, or a discoordination of function. The former includes paresis, paralysis, atrophy, and incomplete glottic closure, as well as diminished vocal support. These type of conditions may be acute losses (e.g., stroke, surgical injury) or degenerative (e.g., PD, amyotrophic lateral sclerosis [ALS]). The discoordination differential includes tremors, myoclonus, and dystonia.

◆ Evaluation

History

As with many vocal disorders, patients may precisely self-define their vocal concern, but also may have only a vague description of their problem. A careful history to define their issues includes the timing and situational onset of the symptoms, exacerbating and ameliorating factors, associated symptoms, and vocal quality.

Voice Exam

Evaluation of the voice by the trained listener is essential. This begins even as the patient is relaying their history. Nonlaryngeal factors should be noted, such articulation issues or hypo- and hypernasality. Laryngeal issues include overall vocal quality (e.g., raspy, breathy, strained, spasmodic), severity of dysfunction, appropriateness of vocal pitch, and presence of voice or pitch breaks. Assistance in this assessment can be provided through the use of certain prepared readings. The most common of these is the "Rainbow Passage," which contains balanced consonants and vowels:

> "When the sunlight strikes raindrops in the air, they act like a prism and form a rainbow. The rainbow is a division of white light into many beautiful colors. These take the shape of a long round arch, with its path high above, and its two ends beyond the horizon. There is, according to legend, a boiling pot of gold at one end. People look, but no one ever finds it. When a man looks for something beyond his reach, his friends say he is looking for the pot of gold at the end of the rainbow."

Using predominantly voiced or voiceless phrases may also be of benefit, particularly in spasmodic dysphonia. Appropriate phrases for adductor spasmodic dysphonia include the following:

- Counting from 80 to 89
- "Eeee-eee-eee"

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- "We mow our lawn all year."
- "We eat eels every day."
- "We eat eggs every evening."
- "A dog dug a new bone."
- "Where were you one year ago?"
- "We rode along Rhode Island Avenue."

Appropriate phrases for abductor spasmodic dysphonia include:

- Counting from 60 to 69
- "See-see-see"
- "The puppy bit the tape."
- "Peter will keep at the peak."
- "When he comes home, we'll feed him."
- "Harry has a hard head."
- "Tap the tip of the cap, please."
- "Keep Tom at the party."

Some vocal disorders are particular to certain tasks, while others are universal; having a patient sing a well-known tune such as "Happy Birthday" can help determine this fact. The next component, the neurolaryngologic examination, is best done with a flexible endoscopy. This allows for fluent speech and avoids the distortion, and potential inhibitory effects of tongue retraction on direct or indirect laryngoscopy. The larynx should be observed at rest, during normal breathing, and during phonation. Appropriate adduction and abduction with phonation and respiration should be observed. The "/i/ - sniff" maneuver should elicit maximal abduction. Asymmetric or paradoxical vocal fold motion may indicate a paresis or dystonia. Rhythmic spontaneous or intention tremor of the larynx should be noted as well as nonrhythmic myoclonus. Compression of the false folds and supraglottis represents excess use of accessory muscles suggestive of muscle tension issues or dystonias. Quickly repetitive phonatory tasks (such as /i/ - sniff, alternating /i/ - /hi/, and /pa/ - /ta/- /ka/) may make subtle paresis or discoordination more evident. Glissando (sliding low to high pitch /i/) can be used to assess tensioning function.

Imaging

In vocal fold paresis and paralysis, CT and MRI can confirm that no lesions exist along the course of the vagus and recurrent laryngeal nerves, which could interfere with function. Central nervous system (CNS) imaging may be appropriate if it assists in confirmation of a systemic neurologic condition associated with the neurolaryngeal complaints.

Other Tests

Stroboscopy

Stroboscopic examination allows for the evaluation not only of gross laryngeal mobility and glottic closure assessment, but subtle mucosal wave

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abnormalities. Assessment of the mucosal wave function may yield additional information regarding the dysphonia; however, not all mucosal wave issues necessarily contribute to vocal complaints.

Laryngeal Electromyography

Laryngeal electromyography (LEMG) can be integrated into the laryngeal functional assessment. Controversy exists as to the extent it should be routinely used in dysphonia. Some agreement exists in its utility in certain circumstances:

1. Needle guidance during botulinum toxin injection to the larynx
2. Diagnosis of vocal fold movement disorders
3. Assessment and prediction of unilateral vocal fold paresis or paralysis after recurrent laryngeal nerve injury
4. Research tool

The validity and interpretation of LEMG in the diagnosis of other neurologic or myopathic disorders is not well defined at this time. Interpretation of LEMG often involves a partnership between the otolaryngologist and a skilled electromyographer, often a neurologist or physical medicine and rehabilitation physician. The factors of fibrillation potentials, fasciculations, recruitment, and action potential characteristics should be noted and can assist in supporting history and examination-generated diagnosis.

◆ Treatment Options

Medical

Medical therapy for neurolaryngeal complaints focuses on attempts to maintain a patent airway, protect the airway from aspiration, and restore normal laryngeal aerodynamics and phonation. Swallowing assessments (e.g., bedside swallow evaluation, fiberoptic endoscopic evaluation of swallowing [FEES], modified barium swallow [MBS]) may be necessary for diagnosis and to determine therapy in some conditions affecting the pharyngeal mobility or protective reflexes of the larynx. After a thorough swallowing evaluation, a speech and language pathology consultation may be beneficial for rehabilitation exercises.

The most common neurolaryngeal disorder, vocal fold paralysis and paresis, is discussed in detail in Chapter 4.3.3. Five other common neurolaryngologic issues will be addressed here as examples of treatment: cerebrovascular accident, Parkinson disease (PD), laryngeal tremor, spasmodic dysphonia, and VFD.

Cerebrovascular accident (stroke) may have multiple effects on laryngeal function. It may alter the ability to protect the airway, swallow, respire, and phonate. Some stroke patients will have language issues (aphasias), as opposed to laryngeal phonatory issues, and these must be distinguished to properly design treatment. Deficits in stroke may include poor breath support, incomplete glottic closure (even vocal fold paralysis), loss of laryngeal sensation, and discoordination of the laryngoesophageal complex. Specific medication is not recommended, but may be instituted for global deficits. Attention should primarily be paid to speech and language pathology therapy to maximize function and minimize aspiration.

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PD is caused by progressive degeneration of particular brainstem structures, including the substantia nigra. Seventy to 89% of patients experience vocal symptoms, even as the first presenting symptom. Patients with PD can have voices characterized by soft, breathy, monotone speech, occasionally tremor, as well as nonlaryngeal issues such as poor articulation. Specific speech therapy has been designed for PD patients, Lee Silverman Voice Treatment, an intensive therapy involving phonatory effort and glottal closure. Pharmacologic management of PD is beyond the scope of this chapter but primarily involves dopaminergic replacement.

Vocal tremor is rarely restricted to the laryngeal musculature, but often involves many muscles involved in phonation. Treatment of vocal tremor may be initiated with medications indicated in essential tremor but has been less well investigated specifically for the larynx. Propranolol (β-adrenergic blocker–reduces tremor amplitude), primidone (neuroleptic–mechanism unknown), and methazolamide (carbonic anhydrase inhibitor) have all been utilized in vocal tremors, but limited studies show efficacy, and dosing is not well defined. Botulinum toxin injections can be performed, typically into one of both thyroarytenoid muscles, to reduce tremor amplitude, but it does not eliminate tremor entirely.

Spasmodic dysphonia may be either adductor or abductor, and rarely mixed. Speech therapy has a little role in treating these patients. Botox injection into one or both thyroarytenoid muscles (adductor spasmodic dysphonia) or posterior cricoarytenoid muscle (abductor spasmodic dysphonia) remains the mainstay of therapy. Unilateral or bilateral injections, as well as dosing, must be titrated to the individual patient through trial and effect. A reasonable starting dose for injection is 1 to 2 U. Some clinicians prefer unilateral injections, as they seem to have the best voice outcome for effect/side effect ratio.

Vocal fold dysfunction (VFD) is likely a spectrum of disorders wherein inappropriate vocal fold adduction occurs during inspiration. Rare organic causes of this occurrence, such as brainstem compression (e.g., Arnold-Chiari malformation) or upper/lower motor neuron injury should be considered and confirmed if suspected. In classic VFD, this inspiratory adduction creates the characteristic anterior fold adduction with posterior glottic chink. This may occur with laryngeal irritants and psychological stress. The process is often mistaken initially for asthma, and can rarely occur synchronously. Therapy directed at vocal fold relaxation and breathing is paramount. Psychological stressors should also be addressed to reduce initiating factors. Any concomitant psychiatric illnesses and symptoms should be managed pharmacologically, if necessary. Acute exacerbations may be managed with the assistance of benzodiazepines and topical laryngeal lidocaine. Some clinicians believe reflux accompanies many patients with VFD and should be treated concurrently.

Surgical

Surgical therapy for laryngeal issues of stroke is limited to palliative measures. Patients with severe impairment of swallowing and airway protection may benefit from tracheotomy for pulmonary hygiene, and possible gastrostomy tube placement for enteral nutrition.

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PD may involve vocal fold atrophy, and has been treated with vocal fold augmentation. Although this therapy aids glottic closure to a degree, issues remain. Overall, PD may be treated surgically through deep brain stimulation, which improves the extremity symptoms, but less so the voice and articulation problems.

Essential tremor has been successfully treated with thalamic deep brain stimulation, but isolated vocal tremor is not an indication for this neurosurgical intervention. Some patients treated with bilateral brain stimulation developed dysarthria raising concerns over this procedure in vocal tremor.

Spasmodic dysphonia patients do have some surgical options, but currently most patients continue botulinum toxin injections. Recurrent laryngeal nerve section was described by Dedo in 1976. Although initially beneficial in a half to two thirds of patients, many had recurrent symptoms years later. The procedure is also complicated by glottic incompetence, strain, and high-pitch. Resection of a section of the nerve as described by a group at Vanderbilt University Medical Center (Nashville, TN) improves the rate of recurrence, but 33% needed medialization procedures. Isshiki describes an expansion laryngoplasty for spasmodic dysphonia, but data and follow-up is limited. Berke has modified the recurrent laryngeal nerve section to include only the adductor branch and introduced reinnervation by the ansa cervicalis. Selective nerve section and reinnervation has reported good subjective voice improvements with 90% having, mild or no voice breaks and mild or no dysphonia.

Vocal fold dysfunction has no surgical treatments. Tracheotomy has been used to bypass the larynx, but is rarely necessary with nonorganic causes of VFD.

◆ ICD-9 Codes

332	Parkinson disease
333.9	Essential tremor
335.20	Amotrophic lateral sclerosis
478.3	Paralysis of vocal folds
478.75	Laryngeal spasm
784.49	Hoarseness

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4.3 Voice Disorders

4.3.1 Papillomatosis

◆ Key Features

- Papillomatosis is the most common benign neoplasm of the larynx.
- It is a human papillomavirus (HPV) induced lesion.
- There is a low chance of malignant conversion.
- The mainstay of therapy is surgical resection, although adjuvant therapies may be helpful in severe cases.

Papillomatosis may affect any mucosal surface of the head and neck, but does have a predilection for junctions between ciliated respiratory and squamous mucosa. The most common sites are the nasal vestibule, oropharynx, nasopharyngeal surface of the soft palate, upper and lower limits of the laryngeal ventricle, and undersurface of the vocal folds. Human papillomavirus is the etiologic agent, with some subtypes (HPV-6, HPV-11) predominating. Recurrent respiratory papillomatosis (RRP) typically presents with dysphonia, although some children, and even fewer adults, may present with airway compromise. Treatment is surgical resection, with cold steel dissection, microdébrider, or laser. Adjuvant chemotherapy agents may have some role in recalcitrant cases.

◆ Epidemiology

Laryngeal papilloma can be categorized into two subgroups: juvenile and adult onset. Juvenile usually occurs in children less than 5 years, with 25% presenting in infancy. Incidence of juvenile RRP is 4.3 per 100,000 in the United States. Children are frequently (75%) the firstborn, vaginally delivered offspring of teenage mothers. Males and females are equally affected. Transmission is maternal–neonatal and of higher risk in those with active HPV genital warts (condylomata), although other factors play

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a role. Adult-onset RRP presents at ages 20 to 40 years, with a 4:1 male/female ratio. Incidence is 1.8 per 100,000. In adults, the disease is likely to be sexually transmitted.

◆ Clinical

Signs and Symptoms

Children may present with airway obstructive symptoms, particularly if they are presenting very young. Children may also have a husky cry or dysphonia. Secondary to the rare nature of the disease and the pediatrician's inability to visualize the larynx, children are frequently misdiagnosed as having other airway problems such as asthma, bronchitis, or croup. Symptoms are present on average 1 year before diagnosis. In some cases, children will present with emergent airway obstruction. Adults typically present with dysphonia, and rarely with acute airway compromise. Symptoms are present on average for 6 months before diagnosis.

Differential Diagnosis

Hoarseness in children may be caused by vocal nodules, reflux disease, vocal fold immobility, laryngotracheobronchitis, laryngeal cysts, congenital laryngeal abnormality, or neurologic conditions. Upper airway compromise causes may include congenital laryngeal lesions, laryngeal cysts, vocal fold immobility, subglottic stenosis, a foreign body, and infectious processes, such as epiglottitis or laryngotracheobronchitis.

Hoarseness in adults may be caused by vocal fold nodules, reflux laryngitis, vocal fold cysts or polyps, leukoplakia, vocal fold neoplasms, sulcus vocalis, inflammatory laryngitis (e.g., tobacco abuse, steroid inhalers), vocal fold immobility, hypothyroidism, systemic illnesses such as sarcoidosis or amyloidosis.

◆ Evaluation

Physical Exam

The physical exam should include a full head and neck examination. Attention should be made to the respiratory status of the patient, to assess whether acute interventions will be necessary to preserve the airway. Vocal quality should be noted. Indirect laryngoscopy can be done on adult patients. Flexible laryngoscopy may be done in adults and nondistressed children to assess location, extent, and functional limitations of the papilloma disease. Videostroboscopy can be useful when available to assess the impact of the papilloma on mucosal wave dynamics.

Imaging

Imaging has limited use, except in assessing for other issues causing airway compromise in children or assessing distal pulmonary papillomatosis. High-kilovoltage plain films or airway fluoroscopy may be helpful in this regard but do not specifically assist in the diagnosis of RRP.

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Labs

No specific labs are helpful in RRP. Some otolaryngologists recommend HPV-typing at the time of resection. This does not alter treatment but can offer some prognostic information, as HPV-11 patients tend to have more aggressive disease, more recurrences, more surgical procedures, and more use of adjuvant therapies.

Pathology

Papillomas contain a pedunculated, vascular, fibrous core with overlying nonkeratinized squamous epithelium. Multiple projections emanate off the central core, giving a frond- or wart-like configuration. Cellular atypia may occur in the epithelium, and can be concerning for premalignant changes.

◆ Treatment Options

Medical

Although there are no primary medical therapies for RRP, adjuvant therapy to surgical resections may be necessary. Criteria for adjuvant methods include more than four procedures per year, rapid recurrence with airway compromise, and distant spread of disease. Approximately 10% of juvenile RRP patients require adjuvants.

The most commonly used therapy is recombinant α-interferon. This protein complex is a host defense to viral infection and immunomodulates the host into an antiviral condition. This has been shown to reduce the frequency of operative interventions. It is administered daily for 1 month, then tapered to three times weekly for at least 6 months. Side effects include flu-like symptoms, alopecia, leukopenia, coagulopathy, and neurologic side effects.

Indole-3-carbinol is an herbal supplement derived from cruciferous vegetables. The mechanism is unclear, but is believed to be related to alterations in estrogen metabolism. Studies show a majority of patients receive partial to complete response. Dosages for children less than 25 kg are 100 to 200 mg daily and adults 200 mg twice daily. Side effects include headache and dizziness.

Cidofovir is a cytosine nucleotide analog antiviral agent, designed for herpetic viruses and cytomegalovirus. Intralesional injections have shown good response in some patients. Concern for promoting progression to squamous cell carcinoma has been raised, but not proven.

Acyclovir has been used systemically, but the benefits are not well defined.

Photodynamic therapy utilizes the uptake of hematoporphyrins by papilloma to sensitize the tissue to red laser light. Disease progression is improved, but remission is not achieved.

Quadrivalent HPV vaccines have recently been released for HPV subtypes 6, 11, 16, and 18, with the indication for treating young girls before sexual activity to reduce the rate of cervical cancers. The hope exists that this use will influence the rate of laryngeal papilloma in future generations, and may even be applied to males in the future.

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Surgical

Surgical resection via microlaryngoscopy remains the mainstay of therapy. Recurrences are typical and multiple procedures are the norm. Juvenile RRP tends to be more aggressive and require more surgeries, likely related to increase growth rate of the RRP and the smaller dimensions of the juvenile larynx prompting earlier intervention for recurrence. Techniques for removal include several modalities, and are influenced heavily by surgeon preference. Cold steel dissection of the papilloma may be useful for small isolated lesions, but not diffuse lesions. The CO_2 laser has been the workhorse of RRP for many years. Vaporization of the lesions is a viable treatment option. The laryngeal microdébrider can also be used; it is favored by some surgeons over laser as it may have less "peripheral damage" given its lack of thermal injury, and does not require special intraoperative laser precautions. Several new lasers have been utilized in RRP, even in the office setting via flexible scopes. These include flexible CO_2 and pulse-dye lasers.

◆ Outcome and Follow-Up

Recommendations regarding postoperative care vary by surgeon (e.g., voice rest). Reflux medications are recommended by many to reduce postoperative scarring exacerbated by any acid exposure, as well as to reduce the potential cofactor of acid exposure in RRP regrowth.

By its very name, RRP is a recurrent problem for patients. The course is variable, with some patients experiencing lifelong recurrences and others manifesting spontaneous remissions. Juvenile- onset RRP does seem to have a higher rate of remission as the children enter adolescence.

Some patients have the concern of spread of RRP to family members or sexual partners. There are no well-documented cases of patient-to-patient transmission of laryngeal RRP. Some theoretical concern exits for caregivers, however, with reported viable virus in laser smoke plumes.

◆ ICD-9 Code

212.1 Benign laryngeal neoplasm

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4.3.2 Vocal Fold Cysts, Nodules, and Polyps

◆ Key Features

- Nonneoplastic disorders of the larynx include nodules, cysts, and polyps.
- Dysphonia is a common presenting complaint.
- Large polyps may rarely present with airway obstruction.

Nonneoplastic changes affecting the vocal folds are common causes of chronic hoarseness. An office exam and videostroboscopy can generally lead to an accurate diagnosis. Treatment may involve voice therapy, microlaryngeal surgery, and behavioral modifications. With accurate diagnosis, appropriate management, and patient compliance, treatment should be highly effective.

◆ Epidemiology

Benign and reactive laryngeal lesions are common disorders; true incidence is difficult to determine.

◆ Clinical

Signs and Symptoms

Patients with various nonneoplastic vocal fold disorders generally complain of hoarseness of variable duration. A history of voice abuse or violent coughing is common. Vocal nodules are almost always seen in young women or young children with a history of voice abuse. Vocal fold polyps are more frequent in men. Stridor is occasionally associated with large vocal polyps. Vocal fold cysts present also with substantial voice changes and usually affect adults. Cysts are thought to be congenital or acquired and seem to arise in the setting of chronic irritation and inflammation after hemorrhage. Reinke's edema, or polypoid chorditis, is a distinct entity from vocal polyps or nodules and usually presents with a pitch disturbance (e.g., females being mistaken for males on the telephone). It is caused by tobacco abuse.

Differential Diagnosis

The major consideration is the possibility of laryngeal squamous cell carcinoma, especially in the setting of tobacco abuse. History and physical examination alone may be insufficient to differentiate early-stage cancer from certain benign conditions. Differential diagnosis includes nodules, cysts, polyps, Reinke's edema, granuloma, papillomatosis, reflux laryngitis, other chronic laryngitis forms, glottic sulcus vocalis, and squamous cell carcinoma.

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◆ Evaluation

History

A specific history of severe coughing, anticoagulant use, voice abuse, vocal fatigue, singing, tobacco abuse, or reflux symptoms should be ascertained. Prior intubations, laryngeal trauma, or laryngeal surgeries must not be overlooked. It is important to elicit timing of onset.

Physical Exam

A complete head and neck exam is performed. Office endoscopy and, if available, videostroboscopy often enable a diagnosis. Vocal nodules are seen as a thickened bilaterally symmetric lesion involving the midmembranous vocal fold contact site, generally found in a young female patient. Vocal fold cysts are usually solitary and visible as a bulge in the affected fold, but when small may be confused with other subepithelial lesions. Characteristic changes in vibration are appreciable on stroboscopic exam. Vocal fold polyps can be variable in size and may be single or multiple. Generally, polyps arise near the vibratory margin of the vocal fold. A feeding vessel or evidence of hemorrhage may be obvious. Large pedunculated polyps are occasionally seen arising from the upper or lower surface of the vocal fold. Reinke's edema appears as symmetric pale thickening of the folds, often with dehydration and thick mucus. Glottic sulcus vocalis may be suggested by stroboscopic findings revealing a segment with reduced vibration, but microlaryngoscopy is often required for definitive diagnosis.

Imaging

Radiologic examination is not indicated in most cases.

◆ Treatment Options

Vocal nodules usually respond to voice therapy alone. Vocal fold cysts may respond to voice therapy; if not, they are removed by microsurgery via cordotomy and microflap. Vocal fold polyps may respond to voice therapy if small but are usually treated with surgery followed by voice therapy and antireflux therapy. Surgery involves excision with microinstruments and the use of short-pulsed CO_2 laser coagulation of a feeding vessel, if present. Reinke's edema may be treated conservatively with voice rest and smoking cessation, or treated surgically with submucosal evacuation via a cordotomy, generally placing the incision on the superolateral surface. In all cases, behavioral modification to address tobacco abuse, reflux, voice abuse, and vocal hygiene is important.

◆ Outcome and Follow-Up

In most cases, prognosis should be very good. Prevention of recurrence should involve follow-up monitoring of smoking and other behavioral factors discussed above.

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◆ ICD-9 Codes

478.4 Vocal fold polyp
478.5 Other disease of the vocal fold
478.6 Edema of the vocal fold

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4.3.3 Vocal Fold Motion Impairment

◆ Key Features

- "Immobility" and "motion impairment" are better terms for this condition than the routine use of "paralysis" given that other etiologies exist for the motion defect.
- Immobility may be due to central or peripheral neural pathology.
- Immobility may also involve fixation of the joint or subluxation.
- Unilateral vocal fold immobility creates primarily dysphonia, whereas bilateral vocal fold immobility may create vocal and airway problems.

Vocal fold motion impairment may occur at any age. The impairment may relate to a vocal fold paresis, a vocal fold paralysis, and cricoarytenoid joint fixation or dislocation. A unilateral vocal fold paralysis is more often on the left secondary to the recurrent laryngeal nerve's further descent into the chest. Most patients present with dysphonia, although a bilateral issue may cause airway distress acutely or in a delayed fashion. An evaluation is performed to attempt to rule out etiologies that may require further therapy. Rehabilitation of the voice may be accomplished via medialization of the affected vocal fold to improve glottic closure. Airway impairment secondary to bilateral lesions may be palliated by bypassing the obstruction (tracheotomy) or by increasing the glottic airway area.

For bilateral vocal fold paralysis in children, see Chapter 6.3.

◆ Clinical

Signs and Symptoms

In pediatric patients, unilateral vocal fold motion problems may be overlooked, particularly if the child has other congenital anomalies or medical

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issues. A weak cry, with possible feeding or choking problems, may be noted. In some cases aspiration and cyanotic episodes may occur. Rarely, stridor and airway symptoms exist. Bilateral vocal fold motion impairment will typically involve stridor and respiratory insufficiency. A normal cry may be preserved.

In adults, unilateral vocal fold motion impairment presents with dysphonia. The degree of hoarseness varies widely from essentially a normal voice to severe breathy dysphonia. Diplophonia may also be present. If the sensory portion of laryngeal innervation is also involved, then secretion pooling or silent aspiration may occur. This will typically be worse with thin liquids than with thicker liquids or solids. Similar to children, a bilateral vocal fold may have a relatively preserved voice secondary to the more media position of the vocal folds. However, varying degrees of airway obstruction and distress may be noted. Patients may have severe obstruction requiring urgent, airway interventions (endotracheal intubation, tracheotomy), or may exist as a more chronic dyspnea with steady or variable symptoms.

Differential Diagnosis

Vocal fold motion impairment may be neurologic (central or peripheral) or mechanical. Vocal fold motion problems may arise from:

- Cervical surgery (thyroidectomy, carotid endarterectomy, anterior cervical fusion)
- Thoracic surgery (pulmonary lobectomy, cardiac bypass or valve surgery, aortic aneurysm repair, esophagectomy)
- Neurologic disease (Guillain-Barré syndrome, Charcot-Marie-Tooth disease, Arnold-Chiari malformation, hydrocephalus, cerebrovascular accident, myasthenia gravis, ALS, Lyme disease
- Posterior glottic stenosis
- Endotracheal intubation trauma to cricoarytenoid joint or pressure injury to recurrent laryngeal nerve
- Neck trauma
- Inflammatory diseases (rheumatoid arthritis, Wegener's granulomatosis, sarcoidosis, syphilis, pemphigoid)
- Neoplasms along the course of the recurrent laryngeal nerve (intracranial, cervical, thoracic)
- Laryngeal neoplasm
- Viral neuritis
- Idiopathic

◆ Evaluation

History

The etiology may be evident via the history and timing of the onset of symptoms, or may require further evaluation. A careful history for aerodigestive tract symptoms, and a history of prior cervical or thoracic surgery, cervical

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masses, neoplastic disorders, endotracheal intubation, thyroid disease, generalized neurologic symptoms, and viral prodromes should be elicited.

Physical Exam

The physical exam should include a complete head and neck examination, flexible fiberoptic laryngoscopy, and/or videostroboscopy.

Imaging

Imaging to involve the course of the recurrent laryngeal nerve is recommended to rule out compressive or infiltrative process. A CT scan from the skull base through the aortic arch should be ordered in any patient without an obvious etiology. Some clinicians will use MRI of the brain to evaluate for CNS lesions or posterior fossa malformations, but this is not routine in the evaluation of vocal fold motion problems.

Labs

Laboratory tests are appropriate if a system illness is suspected as an etiology, but no tests are done routinely. Tests may include rheumatoid factor, antineutrophil cytoplasmic antibody, antinuclear antibody, VDRL, or Lyme titer. LEMG may be utilized in the immobile fold to assess for denervation and recovery. This may be helpful in differentiating paralysis (fibrillation potentials on LEMG) versus fixation (normal action potentials on LEMG). The state of denervation and recovery may be prognosticated by the presence of fibrillation potential (denervation), electrical silence (prolonged denervation with muscle atrophy), or polyphasic action potentials (recovery of some innervation).

◆ Treatment Options

The overall treatment may be guided by the etiology of the vocal fold motion impairment. Treatment of the underlying illness may yield motion recovery in some cases (e.g., immunomodulators for rheumatoid arthritis), or may yield no motion recovery. Rehabilitation of the voice and possibly dysphagia improvement are the goals for most unilateral problems, whereas airway maintenance predominates in bilateral cases.

Medical

No evidence exists for the use of oral steroids in acute-onset vocal fold paralysis. Speech therapy consultation may be beneficial for maximizing vocal function, given the limitations of a nonfunctional fold. Additionally, as some patients may experience dysphagia or aspiration symptoms, the therapist may be able to clinically assess the swallow function and make recommendations about therapeutic maneuvers to improve symptoms.

Surgical

Medialization of the impaired vocal fold is performed to provide better glottic closure and thus more normal phonatory dynamics. Techniques may provide temporary or permanent solutions. Injection augmentation may utilize absorbable or permanent materials. This may be performed

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transcutaneously, under endoscopic guidance in the office, or during operative microlaryngoscopy. Open approaches (medialization laryngoplasty) involve making a thyroid ala window and placing preformed or surgeon-carved silastic implants, or layered polytetrafluoroethylene Gortex (W.L. Gore and Associates, Flagstaff, AZ). Arytenoid adduction may be beneficial in some patients as an adjuvant to reduce posterior glottic gap. Reinnervation of the thyroarytenoid muscle may assist in vocal fold tone and improved glottic closure. The main purpose of reinnervation is to prevent laryngeal muscular atrophy.

◆ Outcome and Follow-Up

In the case of vocal fold paralysis, clinical recovery may require as long as 12 months. Therefore, serial evaluations may be necessary to document the recovery and to reevaluate for vocal issues benefiting from intervention. A functional voice initially may worsen over time as vocal fold atrophy and final fold position manifest. LEMG may provide some prognostic information regarding recovery.

◆ ICD-9 Code

478.3 Paralysis of the vocal folds or larynx

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4.3.4 Voice Rehabilitation

◆ Key Features

- Voice therapy encompasses techniques employed in the management schema for patients with voice disorders.

Vocal rehabilitation often involves a multidisciplinary approach combining the services of both otolaryngologists and speech-language pathologists. Speech-language pathologists offer several therapeutic approaches

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with consideration of the patient's variables: the etiology of the voice disorder, the vocal demands of the patient, and patient cooperation/compliance. Vocal rehabilitation methods include resonant voice therapy, vocal function exercises, confidential voice therapy, manual circumlaryngeal reposturing, and Lee Silverman Voice Therapy. Comprehensive vocal rehabilitation often includes a combination of one or more therapeutic approaches.

◆ Epidemiology

Vocal disorders amenable for voice therapy include several possible etiologies, including benign mid-membranous vocal fold lesions, functional voice disorders, vocal fold paralysis or paresis, laryngeal movement disorders, and progressive neurogenic disorders such as PD and ALS.

◆ Clinical

Signs

Signs of vocal disorders include perceptual, acoustic, and physiological variables. These variables are often defined by the patient as one or more of nine common symptoms of voice problems.

Symptoms

The nine most common symptoms of voice problems include hoarseness, vocal fatigue, breathiness of voice, reduced phonatory range, aphonia or total loss of voice, pitch breaks or inappropriate pitch level, strain, tremor, and pain.

Differential Diagnosis

Differential diagnosis of voice disorders must include evaluation of all perceptual, acoustic, and physiological variables to determine the most appropriate voice therapy approach.

◆ Evaluation

Evaluation of voice disorders should include evaluation of perceptual, acoustic, and physiological components of vocal function.

Perceptual Evaluation

Perceptual evaluation includes evaluation of pitch, loudness, quality, and other related features. Perceptual evaluation is often subjective based on the assessment of the voice by both the speech-language pathologist and the patient. The Voice Handicap Index is a quality of life instrument used by a patient to rank his or her voice in areas of functional, physical, and emotional impact of the voice (**Table 4.3**).

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Table 4.3 Voice Handicap Index

Pitch	Evaluation of monopitch, inappropriate pitch, pitch breaks, reduced pitch range
Loudness	Evaluation of monoloudness, loudness variation, reduced loudness range
Quality	Hoarse or rough, breathy, tension, tremor, strain/struggle, sudden voice interruptions, diplophonia
Other behaviors	Stridor, throat clearing, habitual cough

Acoustic Evaluation

Acoustic evaluation includes evaluation of the variables related to movement of the vocal folds and sound production from the vocal tract. This assessment can be completed with the use of voice analysis software including a Visi-Pitch or Computerized Speech Laboratory (both from Kay Elemetrics Corp., Lincoln Park, NJ). Assessment includes measurement of:

- Fundamental frequency
- Amplitude
- Signal-to-noise ratio
- Vocal rise and fall time
- Vocal tremor
- Maximum phonation time
- Frequency breaks
- Habitual pitch

Physiological Evaluation

Physiological evaluation relates to the aerodynamics, vibratory behaviors, and muscle activity of the vocal folds. This evaluation can be completed with videostroboscopic evaluation of the larynx, electroglottography, electromyography, and laryngeal endoscopy. The parameters of videostroboscopy include:

- Evaluation of vocal fold edge and texture
- Degree of glottic closure
- Phase closure
- Vertical level
- Amplitude of vibration
- Mucosal wave
- Vibratory behavior
- Phase symmetry
- Periodicity

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◆ Treatment Options

Several voice rehabilitation programs exist including general vocal hygiene, resonant voice therapy, vocal function exercises, confidential voice therapy, manual circumlaryngeal reposturing, and Lee Silverman Voice Therapy. Often, comprehensive voice therapy includes a combination of elements from each of these therapy approaches and close interaction with an otolaryngologist's treatment program.

Vocal Hygiene

Vocal hygiene is a vital component of all voice rehabilitation. This includes focus on adequate hydration, reduction and elimination of vocal abusive and misuse behaviors, proper warm up and cool down of the voice, adequate voice rest, modification of contributing environmental factors, and voice amplification.

Resonant Voice Therapy

Resonant voice therapy focuses on establishing adequate oral and nasal resonance of the voice. This includes tasks focusing through a hierarchy including lip and tongue trills, nasal humming (/mmmm/), nasal words, rote and structured tasks, and conversation.

Vocal Function Exercises

Vocal function exercises are a series of structured vocal exercises that are used to maintain or establish muscle balance within the larynx, strength, and ease of phonation. These exercises promote complete closure of the true vocal folds and are thought to encourage equal open and closed phases of the vocal folds. Four exercises are used.

1. Sustain vowel /ee/.
2. Produce word "knoll" with a pitch glide starting with a high pitch.
3. Produce word "knoll" with pitch glide starting with low pitch.
4. Sustain vowel /oo/ on designated notes C, D, E, F, and G.

It is important to focus on these exercises as speaking tasks and not singing tasks. These exercises are not used primarily for speech tasks but are best when paired with another therapy approach such as resonant voice therapy.

Confidential Voice Therapy

Confidential voice therapy is used to reduce increased glottal closure by producing a glottal gap during phonation and creating a low-intensity, breathy vocal quality. Although focusing on creating a breathy vocal quality, this approach also accomplishes reduction of loudness, rate, and hyperfunction.

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Manual Circumlaryngeal Reposturing

Manual circumlaryngeal reposturing is a hands-on method of vocal rehabilitation focused on relaxation of extraneous muscle tension. This includes direct physical manipulation and massage of the laryngeal area. This method usually works quickly to break muscle patterns that interfere with vocal production. Areas of focus include the base of tongue, cornu of the hyoid bone, thyrohyoid space, and the posterior borders of the thyroid cartilage. Voice production is then trained for similar use of the larynx during phonation without physical manipulation.

Lee Silverman Voice Therapy

Lee Silverman Voice Treatment is a voice therapy approach designed specifically for patients with voice disorders associated with PD. This approach includes specific exercises to promote increased vocal volume. The speech-language pathologist is required to undergo specialized training prior to instruction. The therapy course is based on an intensive program of four sessions per week for 1 month.

◆ Outcome and Follow-Up

Follow-up with repeat laryngeal visualization and follow-up with an otolaryngologist is recommended following a completed course of voice therapy, especially in cases involving vocal pathology such as vocal fold nodules, polyps, paresis, or paralysis. This ensures resolution of the pathology and determination of whether continued medical treatment is necessary.

◆ ICD-9 Codes

784.4 Voice disturbance
300.11 Conversion disorder

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4.4 Swallowing Disorders

4.4.1 Zenker Diverticulum

◆ Key Features

- Zenker diverticulum is a posterior pharyngeal pulsion diverticulum.
- It occurs in an area of potential weakness (Killian-Jamieson, Laimer-Hackermann area, Killian's dehiscence) in the inferior posterior part of the pharynx.
- It occurs in men twice as often as in women.
- It is found more often on the left.

Zenker diverticulum is a pouch that develops in the pharynx just above the upper esophageal sphincter. Typically, this causes dysphagia, regurgitation, halitosis, and generalized irritation. It typically manifests in a posterolateral fashion, with 90% appearing on the left side.

◆ Epidemiology

Zenker diverticulum is 2.5 times more common in men than women and typically occurs in older patients (eighth or ninth decade). It occurs more frequently in European countries or in patients of European heritage. Persons of African descent are rarely affected.

◆ Clinical

Signs and Symptoms

Patients typically complain of dysphagia, regurgitation of undigested food, a feeling of food sticking in the throat, a globus sensation, and a persistent cough (especially after eating). Signs or symptoms may include aspiration, unintentional weight loss, and halitosis. Occasionally, a soft swelling may be palpable in the neck, typically in the left side.

Differential Diagnosis

The differential diagnosis includes pharyngeal malignancy, esophageal malignancy, and dysphagia due to CNS etiology.

◆ Evaluation

History

A history is often suggestive of Zenker diverticulum. Questions should be asked pertaining to weight loss, regurgitation, halitosis, and signs or

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symptoms associated with aspiration, such as frequent choking and coughing. There is typically no pain except in the presence of carcinoma.

Physical Exam

A full head and neck exam should be performed. When performing a laryngoscopy, signs of laryngitis and pooling of saliva in the hypopharynx secondary to underlying cricopharyngeal hypertrophy may be seen. Less commonly, undigested food particles may be seen.

Imaging

Video fluoroscopy with barium typically demonstrates the pouch especially near the end of the second stage of swallowing. This test is usually diagnostic and no further imaging exams are typically necessary. Of note, ensuring the unilaterality of the pouch during this exam is important.

Pathology

Zenker diverticulum is a herniation or false diverticulum of the esophageal mucosa posteriorly between the cricopharyngeus muscle and the inferior pharyngeal constrictor muscles (**Fig. 4.4**). Although rare, it is important to recognize that a small percentage of patients with Zenker diverticulum may have a squamous carcinoma in the pouch (0.5 to 1%).

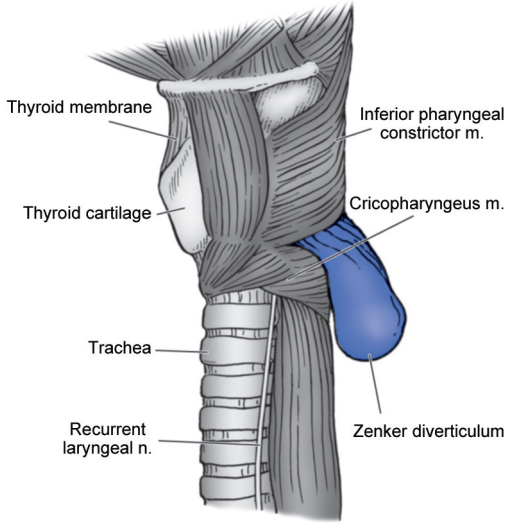

Fig. 4.4 Drawing of laryngopharyngeal anatomy including a Zenker diverticulum. (From Stewart MG, Selesnick SH. Differential Diagnosis in Otolaryngology–Head and Neck Surgery. Stuttgart/New York: Thieme; 2011: 289.)

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◆ Treatment Options

Medical

In a medically infirm patient, Botox injections to the oropharyngeus muscle may be effective.

Surgical

Treatment is typically surgical and reserved for symptomatic patients or patients with aspiration and pneumonia. Surgical management of Zenker diverticulum entails division of the cricopharyngeus muscle to eliminate the potentially elevated pressure zone and elimination of the diverticular pouch as a reservoir of food and secretions. Surgical treatment may be endoscopic or open. Operative intervention is usually undertaken when the diverticulum is at least 3 cm in length.

Endoscopic treatment includes endoscopic identification of the pouch and stapler transection of the cricopharyngeal bar, or common wall between the pouch and the cricopharyngeal introitus are divided to make a common lumen.

Open surgical techniques include open diverticulectomy, inversion, cricopharyngeal myotomy, or diverticulopexy in which the diverticulum is inverted and sutured to the prevertebral fascia. Open techniques are typically performed through a left neck incision.

◆ Outcome and Follow-Up

Long-term follow-up care is not routinely required.

◆ ICD-9 Codes

750.27 Diverticulum of pharynx
Pharyngeal pouch

Further Reading

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4.4.2 Dysphagia

◆ Key Features

- Dysphagia may reflect dysfunction at any level of the swallowing reflex from the oral cavity to the distal esophagus.
- Evaluation requires a detailed history, full head and neck examination, and often endoscopic evaluations and imaging studies.
- Swallowing therapy may be beneficial to those with upper aerodigestive tract dysfunction, who have no medically or surgically correctable problem.

Dysphagia is a term encompassing a large area of clinical symptoms. Patients complain of "trouble swallowing." A careful, detailed history may be necessary to elicit the exact nature of the patient's problems with swallowing. Dysfunction may be secondary to physical obstructive phenomena, neuromuscular weakness or discoordination.

◆ Epidemiology

Dysphagia affects all age groups, dependent on the etiology of the symptom. The elderly are disproportionately affected, however. Some estimate that 50% of nursing home patients suffer with swallowing problems. The percentage of stroke patients who relate dysphagia is 50 to 75%.

◆ Clinical

Signs and Symptoms

Patients may relate very specific aspects of the swallowing reflex that are problematic for them or may simply have a generic complaint of trouble swallowing. Dysphagia is associated with symptoms, including choking, gagging, globus, odynophagia, difficulty initiating swallow, drooling, aspiration, coughing, nasal reflux, and regurgitation. A history of pneumonia is concerning for chronic aspiration. Weight loss may be a sign of dysphagia significant enough to reduce caloric intake, or potentially a neoplastic process. Children with dysphagia may have similar symptoms but also prolonged feeding times, repeated swallow efforts, or unusual posturing during feeding.

Dysphagia may involve one or more of the phases of the swallowing reflex including oral preparatory, pharyngeal, or esophageal phases. Specific symptoms may direct the examiner's attention to one of these sites.

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Differential Diagnosis

- Neurologic disorder (e.g., myasthenia gravis, PD, ALS, multiple sclerosis, cerebral palsy, mental retardation or developmental delay, muscular dystrophy, postpolio syndrome, cranial neuropathies)
- Neurologic injury (e.g., cerebrovascular accident, traumatic brain injury, neurosurgical or skull base procedures with cranial neuropathies)
- Obstruction (e.g., upper aerodigestive tract neoplasms, esophageal web, strictures, achalasia, foreign body, cricopharyngeal dysfunction, cervical spine osteophytes)
- Other (e.g., xerostomia, gastroesophageal reflux disease [GERD], presbyphagia or presbyesophagus, neck irradiation, cleft lip/palate, hypopharyngeal diverticulum, tracheotomy)

◆ Evaluation

Physical Exam

The physical exam should include all components of the upper aerodigestive tract. The oral cavity should be examined and note made of labial competence; salivary function; tongue mobility, symmetry, and strength; palatal motion; mucosal lesions or masses; and gag reflex. The pharyngeal examination should include mirror examination and/or flexible nasopharyngoscopy. In addition to examining for symmetry, mass effect, and mucosal lesion, the presence of pooled secretions is a key finding. Saliva and liquid or food residue in the vallecula, pyriform sinuses, and the postcricoid area indicate either an obstruction of material passage somewhere between the cricopharyngeus and stomach, or a sensory defect of the hypopharynx and larynx.

Laryngeal examination is a component of the pharyngeal evaluation. A "wet voice" may indicate retained secretions in the hypopharynx and laryngeal introitus. The sensation, general mobility, and ability to completely close the vocal folds should be assessed. Neck examination should note symmetry, contour, presence of masses, and presence of laryngeal elevation with swallow. Normal presence of laryngeal crepitus (easy mobility and click encountered on moving the larynx over the cervical spine) should be elicited. Cranial nerves should be assessed, particularly those associated with the swallow mechanism: CNs V, VII, IX, X, and XII.

Imaging

Plain film radiography has a limited role in the assessment of dysphagia. Cervical films may demonstrate foreign bodies, cervical osteophytes, and air/fluid levels in the cervical esophagus or diverticulum. Chest radiographs may show pneumonia or evidence of chronic aspiration, as well as esophageal air–fluid levels. The mainstay of evaluation is the modified barium swallow, or rehabilitation swallow. This test is typically done in conjunction with a speech pathologist. Cinefluoroscopic examination involves having the patient swallow contrast containing a coated material of different consistencies. This allows for an anatomic evaluation of the upper aerodigestive tract but also a

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functional assessment of bolus preparation, transfer, and transit. Patients may vary in their ability to manage various consistencies of food; the MBS allows for identification of this fact, as well as information about therapeutic consistency changes. Retained material may be noted. Additionally, laryngeal penetration and aspiration may be identified. Rehabilitation maneuvers may be tried under fluoroscopy by the speech pathologist and feedback as to their effectiveness can be immediately ascertained. CT and MRI may be necessary to investigate asymmetry for the pharynx and esophagus, or when tumor is suspected.

Other Tests

Fiberoptic endoscopic evaluation of swallowing (with or without sensory testing), or FEES, is another technique available to assess the swallow mechanism. This can be an alternative to a modified barium swallow. A flexible endoscopy is passed through the nose and positioned to visualize the oropharynx and hypopharynx and larynx during swallowing trials. Trailed substances are colored with food dye and the examination is recorded to ease assessment of rapid events and subtle findings. Sensory testing is accomplished via an air pulse delivered through the flexible laryngoscope and identification of the presence and strength of the laryngeal adductor reflex. Formal esophagoscopy is useful if esophageal pathologies are suspected. This technique also allows for biopsy and sometime interventions. Some otolaryngologists have adopted in-office, nonsedated transnasal esophagoscopy for assessing the esophagus. Esophageal manometry and pH probe testing may be adjuvants to assess for esophageal dysmotility and reflux issues.

◆ Treatment Options

Medical

Dysphagia secondary to nonobstructive phenomena is typically managed with therapy techniques. Most of these techniques require voluntary action on the part of the patient, although some require little patient cognition. Techniques may involve head and neck positioning during the swallow, food-consistency changes, and sensory enhancement therapy. A speech pathologist with an interest in swallow rehabilitation should be consulted. Many patients perform better with various consistencies of food: dietary modification and thickening agents may help a patient maintain oral intake. Nutritional supplements may be useful in the nutritionally compromised patient. If reflux is a component of the dysphagia or is thought to be the underlying etiology, medical therapy may be trialed.

Surgical

Surgery may be directed to obstructive phenomena such as tumors, strictures, webs, cricopharyngeal hypertonicity, or hypopharyngeal diverticulum. Vocal fold immobility that contributes to aspiration issues may benefit from medialization thyroplasty. Procedures for chronic aspiration exist (see Chapter 4.4.3). If oral intake is deemed unsafe due to aspiration, feeding gastrostomy may be necessary.

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◆ Outcomes and Follow-Up

The etiology largely determines the treatment outcome and follow-up.

◆ ICD-9 Codes

787.2 Dysphagia
787.20 Dysphagia, unspecified [difficulty in swallowing NOS (not otherwise specified)]
787.21 Dysphagia, oral phase
787.22 Dysphagia, oropharyngeal phase
787.23 Dysphagia, pharyngeal phase
787.24 Dysphagia, pharyngoesophageal phase
787.29 Other dysphagia (cervical dysphagia, neurogenic dysphagia)

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4.4.3 Aspiration

◆ Key Features

- Aspiration involves the passage of secretions or material into the lower airways.
- Aspiration is an important source of morbidity in the neuromuscular impaired and debilitated.
- The most common cause of aspiration is neuromuscular dysfunction.
- Chronic aspiration may have severe medical consequences and medical, sometimes surgical, interventions need to be enacted.

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In general, the lower airway should not be exposed to upper aerodigestive tract secretions or materials. Aspiration of small amounts of material during sleep (reported in 50% normal patients) may be tolerated if the tracheobronchopulmonary system clearing mechanisms are functional. Large episodes of aspiration or chronic aspiration may yield complications, the severity of which are determined by the nature and volume of the material aspirated. Aspiration may be primary (swallowed dietary materials or secretions) or secondary (regurgitated diverticulum or gastric contents).

◆ Clinical

Signs and Symptoms

The presence of chronic aspiration may be evident to the patient and healthcare providers or may be "silent," with no cough generated. Dysphagia is intimately associated with aspiration, as most patients with noted aspiration will also complain of "trouble swallowing" (see Chapter 4.4.2). Symptoms of chronic aspiration include choking or coughing when swallowing (may be with specific types of dietary intake, all food, or even the patient's own secretions). Some patients may also complain of chronic cough independent of swallowing, related to the bronchopulmonary complications of the chronic aspiration. Productive cough, fevers, and dyspnea in a patient with dysphagia is concerning for this issue. Also, recurrent lower respiratory infections in a patient with predisposing comorbidities should make the clinician suspicious of this issue.

Differential Diagnosis

Some conditions may share respiratory symptoms with the complications of chronic aspiration. The key differential diagnosis is for those diseases that may predispose to chronic aspiration:

- Neuromuscular
  - Degenerative diseases
    - Alzheimer disease
    - ALS
    - Multiple sclerosis
    - Progressive supranuclear palsy
    - PD
    - Huntington disease
  - Disorders
    - Myasthenia gravis
    - Myopathy (dermatomyositis, inclusion body myositis)
    - Muscular dystrophy
    - Poliomyelitis and postpolio syndrome
    - Guillain-Barré syndrome
    - Cranial neuropathies

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- CNS injury
  - Cerebrovascular accident
  - Head trauma
  - Anoxic brain injury
  - Postsurgical
  - Encephalitis/meningitis
  - Cerebral palsy
- Upper aerodigestive tract disorders
  - Vocal fold immobility
  - Stricture (web, reflux, caustic ingestion, post surgical)
  - Zenker diverticulum
  - Cricopharyngeal dysfunction
  - Postirradiation/chemoradiation
  - Postsurgical dysfunction
  - Neoplasms
  - Achalasia
  - Reflux
- Other
  - CNS/skull base neoplasm
  - Severe deconditioning or systemic illness
  - Intoxication
  - Mental retardation or developmental delay
  - Presence of a tracheotomy tube

◆ Evaluation

After a detailed history and physical examination (including nasopharyngoscopy), several options exist for evaluation. A bedside swallow evaluation by a speech pathologist is reasonable for those with mild dysphagia, but secondary to the morbidity that may accompany aspiration, if aspiration is suspected, an objective evaluation is warranted.

Imaging

Modified barium swallow has been the traditional imaging study for evaluation of dysphagia and aspiration. This test may demonstrate the passage of contrast material into the trachea and bronchi. Laryngeal penetration or retained contrast within the pyriform sinuses or vallecula are concerning because of their aspiration potential. Therapeutic maneuvers may be performed under cinefluoroscopy and their impact determined immediately, thus aiding in care planning.

Scintigraphy has been proposed to demonstrate and quantify aspiration. Overall, it offers few advantages beyond MBS and fiberoptic endoscopic evaluation of swallowing (FEES) (see below). Chest radiography (plain film or CT radiography) may be helpful in demonstrating pneumonia and chronic pulmonary disease stemming from aspiration (e.g., bronchiectasis).

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Other Tests

FEES involves the transnasal endoscopic monitoring of dyed food and liquid ingestion. Observations are made regarding premature leakage, retained materials, laryngeal penetration, or aspiration. This study is typically video recorded, so that slow motion review may be performed to assess for subtle evidence of swallowing dysfunction.

◆ Treatment Options

Initial management in a patient who has concerns for chronic aspiration is to ensure NPO status, to minimize the volume of potential aspirant until confirmation can be made. Good oral care and speech therapy assessment are appropriate in all patients. Alternative nutrition routes such as nasogastric tubes or percutaneous feeding conduits should be considered, depending on clinical circumstances, for those with prolonged inadequate nutrition.

Medical

A variety of speech therapy techniques are available to assist in safe swallowing. These include consistency changes (e.g., thickening agents, alternating consistencies), as well as positioning maneuvers (e.g., chin tuck, head turn) and swallowing maneuvers (e.g., double swallow, swallow–cough, swallow–cough–swallow, effortful swallow). If a patient is deemed to be inappropriate for any oral intake, alternative feeding conduits should be utilized. If aspiration of secretions is an issue, surgical procedures may be necessary.

Medical therapy should also involve treatment for the complications of the aspiration. These may include intubation, ventilator support, bronchoscopy, antibiotics, and pulmonary toilet.

Surgical

If medical management fails to correct the aspiration problem or is deemed inadequate, several surgical techniques are available. Placement of a tracheotomy tube facilitates pulmonary toilet, and with a cuffed tube inflated may decrease aspiration events. An inflated cuffed tracheotomy tube, however, will not prevent aspiration. The presence of a tracheotomy tube may even cause or worsen aspiration: the laryngeal elevation accompanying a swallow is limited by tracheal tethering by the tube, and laryngeal sensation gradually decreases when airflow through the glottis ceases. A speaking valve may aid in swallowing by improving subglottic pressures necessary to clear secretions, and maintenance of laryngeal sensation. Directed surgical procedures may be helpful in reducing aspiration (e.g., chemical or surgical cricopharyngeal myotomy for cricopharyngeal dysfunction, vocal fold medialization for vocal fold immobility).

Laryngeal surgical management of chronic aspiration may be classified as reversible and irreversible (**Table 4.4**). Each of these techniques has advantages and disadvantages that may influence their use. Reversible procedures all have the potential advantage of being reversible if the underlying aspiration condition improves sufficiently.

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Table 4.4 Surgical Management of Chronic Aspiration

Procedure	Description	Advantages	Disadvantages
Irreversible			
Narrow-field laryngectomy	Removal of larynx, preserving hyoid and strap muscles	Definitive separation of respiratory and digestive tracts	Definitively irreversible Negative psychosocial features for patients Transcervical approach Loss of phonation
Glottic closure	Median thyrotomy with suture closure of true and false vocal folds	High success rate Theoretical, but difficult reversal	Transcervical approach Loss of phonation Tracheotomy
Subperichondrial cricoidectomy	Subperichondrial resection of cricoid with closure subglottic mucosa and strap muscle reinforcement	High success rate Possible under local anesthesia	Transcervical approach Tracheotomy Loss of phonation Fistula potential
Reversible			
Laryngotracheal separation	Transection of the trachea with creation of tracheostoma and closure of subglottis	Among the most dependable of reversible procedures	Transcervical approach Tracheotomy Loss of phonation Fistula potential
Laryngotracheal diversion	Transection of the trachea with creation of tracheostoma and anastomosis of proximal trachea to esophagus	Among the most dependable of reversible procedures "Aspirated" secretions into proximal trachea diverted to esophagus	Transcervical approach Tracheotomy Loss of phonation Fistula potential
Epiglottic flap laryngeal closure	Larynx is closed by denuding edges of epiglottis and aryepiglottic folds and suturing epiglottis over the glottis.	Potential speech preservation if posterior glottis is kept open	Transcervical approach Tracheotomy Possible dehiscence 50% prevention of aspiration Supraglottic stenosis after reversal

continued

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Table 4.4 Surgical Management of Chronic Aspiration *(Continued)*

Procedure	Description	Advantages	Disadvantages
Double-barrel tracheotomy	Transection of trachea with externalization of both ends of the trachea	Decreased fistulization	Transcervical approach Skin contamination with secretions
Partial cricoidectomy	Submucosal resection of posterior cricoid with cricopharyngeal myotomy and tracheotomy, serves to narrow laryngeal inlet and widen esophageal inlet	Preserves the voice	Transcervical approach Tracheotomy Persistent aspiration
Vertical laryngoplasty	Denuding of lower edge of the epiglottis, aryepiglottic folds, arytenoids with vertical tubing of the epiglottis leaving superior opening	Preservation of voice Described for total glossectomy patients to reduce need for laryngectomy	Transcervical approach Persistent aspiration
Endolaryngeal stenting	Endolaryngeal stent placed to occlude the larynx	Simple to place	Leakage around the stent and aspiration Endolaryngeal injury Airway compromise if tracheotomy tube displaced Patient discomfort

◆ Complications

Complications may include chronic lung disease and pulmonary fibrosis.

◆ Outcome and Follow-Up

Outcome depends on the etiology of aspiration.

◆ ICD-9 Codes

507.0 Pneumonitis due to inhalation of food or vomitus

Aspiration pneumonia (due to): NOS, food (regurgitated), gastric secretions, milk, saliva, vomitus

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4.5 Acid-Reflux Disorders

◆ Key Features

- Laryngopharyngeal reflux is distinct from classic gastroesophageal reflux disease (GERD).
- The workup may involve an endoscopy, barium studies, and a pH probe.
- Extraesophageal manifestations relate to the pharynx, larynx, and lungs.

◆ Epidemiology

Twenty-five to forty percent of healthy adult Americans experience symptomatic gastroesophageal reflux disease (GERD) manifested as heartburn. Laryngopharyngeal reflux (LPR) typically does not cause heartburn or esophagitis. Certain foods, medications, hormones, and physical states (e.g., obesity) can decrease the pressure of the lower esophageal sphincter, thus worsening LPR or GERD symptoms.

◆ Clinical

Signs

GERD: esophagitis, Barrett's esophagus

LPR: coughing, throat clearing, laryngeal erythema

Symptoms

GERD symptoms typically include postprandial heartburn and regurgitation. LPR symptoms may be more subtle and include hoarseness, globus sensation, dysphagia, chronic throat clearing, halitosis, sensation of postnasal drainage, chronic cough, laryngospasm, and otalgia.

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◆ Evaluation

A suggestive history and exam findings usually prompts a "therapeutic trial" of behavioral changes and twice-daily use of a proton pump inhibitor. Ancillary testing should be employed if this regimen fails or if symptoms are atypical or worsening, due to risk of malignancy.

Physical Exam

Nasolaryngopharyngoscopy may reveal laryngeal erythema and edema of arytenoids and intraarytenoid space, laryngeal pachydermia, and vocal fold granulomas.

Imaging

A barium swallow study is useful for initial screening and to identify structural abnormalities.

Labs

Laboratory tests are seldom useful.

Other Tests

A dual-channel 24-hour pH monitoring probe is the gold standard. Esophagogastroduodenoscopy identifies the presence and severity of esophagitis and the possible presence of Barrett's esophagus. The role of screening endoscopy is evolving. With the advent of the transnasal esophagoscope (TNE), the routine screening of reflux patients for occult esophagitis or Barrett's esophagus has became more commonplace.

Pathology

Transient lower esophageal sphincter relaxation is a manometric finding. The Reflux Symptom Severity Index, a patient-completed survey, is useful for scoring or grading.

◆ Treatment Options

Medical

Behavioral modification: weight loss, smoking cessation, avoid eating before sleep, avoid caffeine, alcohol, peppermint, chocolate, spicy and acid foods.

H2 receptor antagonists: ranitidine 150 mg orally twice daily. These agents block histamine at the H2 receptors, particularly those in the gastric parietal cells to inhibit acid secretion. Tachyphylaxis may develop to H2 blockers after several weeks of use.

Proton pump inhibitors: esomeprazole 20–40 mg orally twice daily. These agents inhibit gastric acid secretion by inhibition of the H^+/K^+-ATPase system in the gastric parietal cells.

Prokinetics: metoclopramide 10 mg orally four times daily. These agents increase lower esophageal sphincter pressure to help reduce reflux and also accelerate gastric emptying.

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Surgical

About 20% of patients have a progressive form of reflux disease and may develop severe complications. For these patients, surgical treatment should be considered. The indications for this include Barrett's esophagus; strictures; respiratory manifestations (e.g., cough, wheezing, aspiration); ear, nose, and throat manifestations (e.g., hoarseness, sore throat, otitis media); and dental manifestations (e.g., enamel erosion).

With laparoscopic fundoplication, symptoms resolve in ~92% of patients.

◆ Complications

Complications of untreated or resistant GERD include esophagitis, pneumonia, asthma, and interstitial lung fibrosis. Barrett's esophagus is one of the most serious complications of GERD because it may progress to cancer.

◆ Outcome and Follow-Up

Patients should understand that there is a need for long-term maintenance therapy. Many physicians advocate mandatory esophagoscopy, if patients continue to experience symptoms, to rule out Barrett's esophagus changes and cancer. Others advocate screen endoscopy in patients with a positive response to PP1 treatment.

◆ ICD-9 Codes

530.81 Esophageal reflux
530.11 Reflux esophagitis

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4.6 Laryngeal Manifestations of Systemic Diseases

◆ Key Features

- Systemic diseases may generate vocal and airway consequences.
- Management of the underlying systemic illness may or may not allow for reversal of the laryngeal issues.

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Systemic diseases may manifest as laryngeal symptoms and findings. A careful clinical history and appropriate testing should yield a diagnosis. Frequently, consultation with colleagues from other services more familiar with the overall systemic illness may be needed.

◆ Rheumatoid Arthritis

Rheumatoid arthritis is a chronic, inflammatory condition affecting synovial joints with progressive arthritis and deformity. All synovial joints are vulnerable with hands and feet the most commonly affected. Women are three times more likely to develop the disease, usually between the third and seventh decade. A juvenile form of the disease also exists. The cricoarytenoid joint may be involved in this process in 25 to 50% of those with long-standing disease. Symptoms may include hoarseness, globus, and airway compromise. Examination may show inflammatory changes of the arytenoid region, diffuse laryngeal myositis, and rheumatoid nodules within the vocal folds or unilateral or bilateral vocal fold motion impairment. Treatment may include antiinflammatory nonsteroidal medications or corticosteroids. Local injections of steroids into the cricoarytenoid joint region have shown success in improving joint mobility.

◆ Relapsing Polychondritis

Relapsing polychondritis is a chronic and recurrent autoimmune inflammatory condition affecting all cartilage subtypes. The laryngotracheal complex may be involved in 40 to 50% of patients. Disease may affect the airways focally or diffusely. Symptoms may include hoarseness, dyspnea, stridor, laryngotracheal tenderness, or dysphagia. Airway symptoms may be secondary to acute inflammatory and swelling, laryngotracheal collapse secondary to replacement of cartilage by fibrosis, or subglottic stenosis. Treatment is as necessary for any airway obstruction, but in general, therapy is corticosteroids and other immunosuppressive agents.

◆ Wegener Granulomatosis

Wegener granulomatosis is an autoimmune disorder characterized by necrotizing granulomas and vasculitis. The upper and lower respiratory tract and renal systems are primarily affected, although vasculitis may occur anywhere. The larynx is involved primarily in the subglottic and upper tracheal region, and pathology is seen in the early phase of ~10 to 15% of patients. Biopsy may demonstrate the characteristic granulomas. Antineutrophilic cytoplasmic antibodies are elevated in 90% of cases. Airway management of the subglottic stenosis may be necessary and involve dilations, laser resection, or tracheotomy. Formal laryngotracheoplasty or tracheal resection should be reserved for chronic, nonactive disease.

◆ Sarcoidosis

Sarcoidosis is a systemic granulomatous disease of unknown etiology, with a U.S. predilection for African-Americans and those of Scandinavian descent.

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Women are more often affected than men. Pulmonary and lymphatic systems are most commonly affected. Laryngeal involvement may occur in 1 to 5% of affected individuals. The supraglottic structures are typically most involved, with sparing of the true vocal folds. Airway compromise may occur from enlargement of the supraglottic structures from the noncaseating, granulomatous infiltration. Treatment may include systemic steroids, but descriptions of local steroid injection into the involved larynx itself have been described. Serial, conservative lasering of excessive tissue may also be needed.

◆ Amyloidosis

Multiple variants of amyloidosis exist, including primary and secondary systemic amyloidosis, amyloidosis associated with multiple myeloma, as well as a localized form. In the aerodigestive tract, submucosal and intramuscular deposits of fibrillar protein–polysaccharides complex occur. The larynx is seldom involved. Presentation varies from mild hoarseness symptoms secondary to small submucosal vocal fold deposits causing mucosal stiffness, to airway compromise secondary to large supraglottic, glottic, or subglottic deposits. Biopsy is used for diagnosis, with the classic apple-green birefringence under polarized light microscopy with Congo red stain. Treatment with local resection may be necessary if symptoms warrant.

◆ Epidermolysis Bullosa

This is a rare, hereditary connective tissue disorder based on a defect in the dermal basement membrane. Blisters form related to separation of the epidermis and dermis, often after minimal trauma. Milder forms may heal without event unless secondary infections occur, with more severe forms creating mucosal scarring. When the larynx is involved with the process, webbing, stenosis, and synechiae may form. Intubation should be avoided to avoid any trauma that may precipitate the bulla formation and subsequent scarring.

◆ Pemphigoid

Pemphigoid is an autoimmune disease involving antibodies against components of the basement membrane. Blisters form on both cutaneous and mucosal surfaces. Bullous pemphigoid typically heals without scarring, unless secondary scarring occurs. Cicatricial pemphigoid occurs in older patients and may result in significant mucosal scarring. This scarring may occur in the larynx or esophagus, resulting in vocal, airway, and swallowing symptoms. Clinical features and biopsy can make the diagnosis. Laser resection of symptomatic scarring may be necessary. Systemic immunosuppression may be used in significant cases.

◆ Angioneurotic Edema

Angioedema involves plasma extravasation into tissues, resulting in swelling. Multiple forms exist, but idiopathic, medication-related (angiotensin-converting enzyme [ACE] inhibitors), and hereditary forms are most

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commonly encountered by the otolaryngologist. Airway edema may form within the lips, tongue, pharynx, and larynx. Hereditary angioneurotic edema typically presents early in life and is characterized by multiple edema events. Hereditary forms are associated with a defect in C1 esterase inhibitor. Levels of this enzyme may be performed to help make the diagnosis. Treatment acutely involves airway management as necessary. Edema may progress so airway monitoring should be considered, even if acute intervention is not necessary. The hereditary form may be treated acutely with C1 inhibitor concentrate, with androgens being helpful in chronic prevention. ACE inhibitor–related events should be treated acutely with medication cessation, and consideration for steroids. Angioneurotic edema may occur anytime after initiation of the drug, hours to years later.

◆ Neuromuscular Disease

Numerous systemic neuromuscular disorders may cause laryngeal symptoms. These findings may range from vocal fold paresis to paralysis and discoordination of vocal fold motion during speech, respiration, and swallowing. Diseases include myasthenia gravis, ALS, PD, Guillain-Barré syndrome, multiple sclerosis, Charcot-Marie-Tooth disease, and postpolio syndrome.

◆ Other

Many other systemic conditions may cause laryngeal symptoms. Hypothyroidism (myxedema) and allergic disease may both cause vocal fold edema and hoarseness.

◆ ICD-9 Codes

135	Sarcoidosis
277.3	Amyloidosis
277.6	Hereditary angioedema
332	Parkinson disease
335.20	Amyotrophic lateral sclerosis
446.4	Wegener granulomatosis
714.0	Rheumatoid arthritis
784.49	Hoarseness

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5 Head and Neck

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5.1 Anatomy of the Neck

Surgically, it is useful to consider the neck anatomy in terms of compartments or levels and to consider fascial layers. The neck is covered by a layer of superficial fascia, and the deep cervical fascia divides into superficial, middle, and deep layers (**Fig. 5.1**). This is relevant in terms of spread of infection and lymphatic spread of cancer. The visceral structures of the neck are included in the central compartment and include the laryngotracheal complex, the thyroid and parathyroids, and the cervical esophagus. Posteriorly, of course, are the cervical spine and paraspinal muscles. Anterolaterally, the neck contents is considered in terms of nodal levels I though VI (**Fig. 5.2**). Each level contains a compartment of fibrofatty lymph node-bearing tissue that is addressed for removal during neck dissection procedures for cancer. Level I is the submental region and the submandibular area containing the submandibular gland. Level II is the upper jugular chain lymph node region, level III is the middle jugular chain, and level IV is the inferior jugular chain region. Level V is the posterior triangle from the anterior border of the trapezius to the posterior border of the sternocleidomastoid muscle. Level VI is the node-bearing tissue of the central compartment.

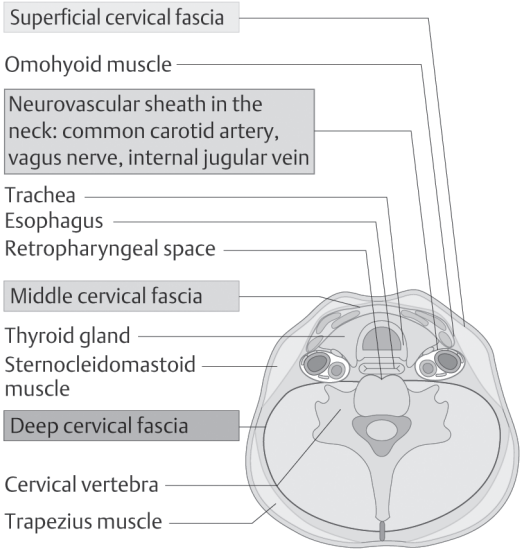

Fig. 5.1 Cervical fascial planes. (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:313.)

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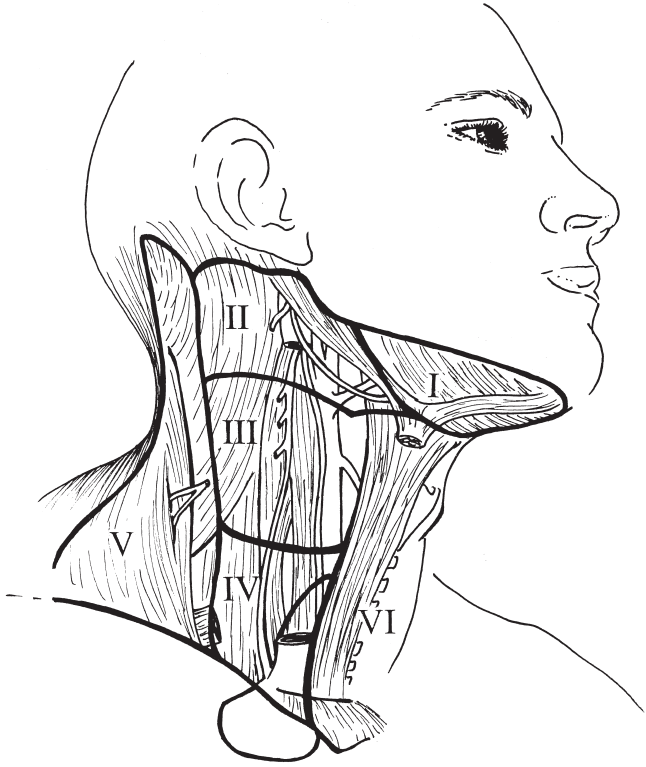

Fig. 5.2 Nodal levels I through VI. (From Van de Water TR, Staecker H. Otolaryngology: Basic Science and Clinical Review. Stuttgart/New York: Thieme; 2006:606.)

◆ Blood Supply

The common carotid artery ascends into the neck and bifurcates into the internal and external carotid artery. The internal carotid has no branches in the neck and delivers important blood supply to the brain, along with the vertebral arteries. The external carotid supplies the head and neck structures via multiple branches (in order, starting inferiorly): the superior thyroid, ascending pharyngeal, lingual, facial, occipital, posterior auricular, superficial temporal, and maxillary arteries (**Fig. 5.3**). Venous drainage tends to parallel major arteries, with variable anterior and external jugular branches superficially, and large common facial veins joining into the internal jugular vein, which feeds the subclavians. The carotid sheath, formed by all three layers of the deep cervical fascia, contains the carotid artery, jugular vein, and the vagus nerve. Important delicate lymphatics from the

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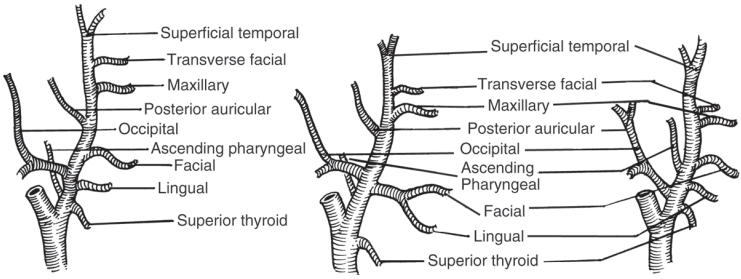

Fig. 5.3 The branching of the arteries originating from the carotid artery can be quite variable. (From Van de Water TR, Staecker H. Otolaryngology: Basic Science and Clinical Review. Stuttgart/New York: Thieme, 2006:603.)

thoracic duct carry chyle into the jugular vein near its junction with the subclavian in the left neck; however, accessory thoracic ducts commonly occur on the right.

◆ Innervation

The 12 cranial nerves (CNs) supply the head and neck region with motor, sensory, and special sensory fibers. They are, in order, the olfactory, optic, oculomotor, trochlear, trigeminal, abducens, facial, vestibulocochlear, glossopharyngeal, vagus, spinal accessory, and hypoglossal. In the neck there are surgically important aspects of CN anatomy. The hypoglossal emerges from the skull base at the hypoglossal canal and courses beneath the digastric and submandibular contents as motor supply to the tongue. The pars nervosa of the jugular foramen contains the glossopharyngeal, vagus, and accessory nerves. The vagus runs within the carotid sheath and the recurrent laryngeal branches course in the tracheoesophageal groove to supply the vocal folds; the superior laryngeal nerves arise from the vagus in the upper neck. The accessory nerve courses posteroinferiorly through the neck entering the anterior border of the sternocleidomastoid, emerging from the posterior border near Erb's point, and continuing into the trapezius. The facial nerve exits the skull base at the stylomastoid foramen and its main motor branches course within the parotid gland to innervate the muscles of facial expression, while the lingual nerve supplies parasympathetics to the submandibular gland and sensation to the anterior tongue and is encountered deep to the submandibular gland. Important cervical nerves are encountered in the neck as well. The great auricular nerve (it is not named gre*at*er, since there is no *lesser* auricular nerve) transmits sensory fibers of C2–3 to the auricular area (**Fig. 5.4**). This nerve is encountered along the sternocleidomastoid coursing superiorly from Erb's point. The phrenic nerve is deep to the floor fascia along the anterior scalene muscle, supplying motor fibers to the diaphragm. Also, the brachial plexus is in the same plane deep to the fascia on the middle scalene. The sympathetic trunk runs deep to the carotid. CN anatomy is reviewed in Appendix B of this book.

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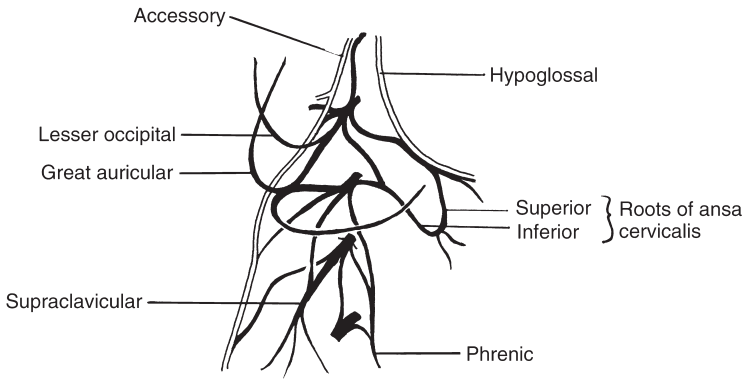

Fig. 5.4 Cervical plexus. (From Van de Water TR, Staecker H. Otolaryngology: Basic Science and Clinical Review. Stuttgart/New York: Thieme; 2006:603.)

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5.1.1 Neck Emergencies

5.1.1.1 Necrotizing Soft Tissue Infections of the Head and Neck

◆ Key Features

- Necrotizing fasciitis is a soft tissue infection that causes necrosis of fascia and subcutaneous tissue, but initially spares skin and muscle.
- It is rare in the head and neck.
- Half of patients develop systemic bacteremia.
- Necrotizing soft tissue infections require aggressive treatment to combat the associated high morbidity and mortality.

Necrotizing soft tissue infections rarely may involve the face and neck, scalp, and eyelids.

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◆ Epidemiology

In the head and neck, dental infections are the most common etiology, followed by trauma, peritonsillar and pharyngeal abscesses, and osteoradionecrosis. Immunocompromised patients are most susceptible. Predisposing conditions include diabetes mellitus, obesity, arteriosclerosis, alcoholism, chronic renal failure, hypothyroidism, malignancy, and poor nutrition. Gas-producing wound infections are usually produced by bacteria of the Clostridia group. Onset of symptoms is usually 2 to 4 days after the insult.

◆ Clinical

Signs

The signs of a necrotizing soft tissue infection include a low-grade fever, and the skin becomes smooth, warm, tense and shiny with no sharp demarcation. The infected skin also develops a dusky discoloration with poorly defined borders. Soft tissue crepitus is common from gas formation. Later in the disease process, bullae may develop. Systemic symptoms get more severe with typical signs of septicemia. The last stage of the disease is characterized by cyanotic skin discoloration typical of necrosis.

Symptoms

There is sudden severe pain and swelling. There may be anesthesia of the involved skin.

Differential Diagnosis

The differential diagnosis includes other deep neck space infections, pyoderma gangrenosum, radiation necrosis, cellulitis, or erysipelas.

◆ Evaluation

Physical Exam

When clinical hard signs are present (i.e., crepitus, skin necrosis, bullae, hypotension), a physical exam may be helpful. Unfortunately, the signs and symptoms are often very subtle on presentation.

Imaging

Plain soft tissue films of the neck looking for gas in soft tissue may be obtained. Computed tomography (CT) is the most useful study to detect gas in areas inaccessible to palpation and to identify areas where the infection has spread. In addition, it can detect vascular thrombosis, erosion of vessels, or mediastinitis.

Labs

Routine blood work is needed to look for metabolic abnormalities such as hyponatremia and hypoproteinemia due to fluid sequestration and hypocalcemia as a result of subcutaneous fat saponification.

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Microbiology

Group A hemolytic streptococci and *Staphylococcus aureus*, alone or in synergism, are frequently the initiating infecting bacteria. However, other aerobic and anaerobic pathogens may be present, including *Bacteroides, Clostridium, Peptostreptococcus, Enterobacteriaceae*, coliforms, *Proteus, Pseudomonas,* and *Klebsiella*.

Pathology

The pathology shows a localized necrosis of skin that is secondary to thrombosis of nutrient vessels as they pass through involved fascia. Tissue hypoxia resulting from small vessel vasculitis and thrombi and impaired host defenses help facilitate anaerobic bacterial growth.

◆ Treatment Options

Prompt measures offer hope for survival of the patient.

Medical

High-dose intravenous (IV) broad spectrum antibiotics are recommended. Hyperbaric oxygen therapy is sometimes used in addition to surgical and antimicrobial treatment.

Surgical

Aggressive surgical débridement until there are bleeding viable borders is necessary, and sometimes repeated excisions to remove necrotic tissue. If the necrotizing process involves the neck, avoid tracheotomy through the infected area.

◆ Outcome and Follow-Up

The overall mortality rate is close to 70%.

◆ ICD-9 Codes

728.8.6 Necrotizing fasciitis

Use additional code to identify:

041.00–041.89 Infectious organism
785.4 Gangrene, if applicable

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5.1.1.2 Ludwig Angina

◆ Key Features

- Ludwig angina is a rapidly expanding, diffuse inflammation of the submandibular and sublingual spaces.
- It is most often caused by dental infections.
- The condition is often found in immunocompromised patients such as those with diabetes or human immunodeficiency virus/acquired immunodeficiency syndrome (HIV/AIDS), or drug abusers.

Ludwig angina is a rapidly spreading bilateral cellulitis of the sublingual and submaxillary spaces. Before the advent of antibiotics, the mortality associated with Ludwig angina approached 50%. Today, mortality rates are in the range of 8 to 10%. The most common cause of death is respiratory compromise.

◆ Epidemiology

Ludwig angina represents up to 13% of all deep neck infections. Typically, young adults present with Ludwig angina. It is unusual in children. The infection generally spreads from a dental or periodontal infection. Other causes include upper respiratory infections, floor-of-mouth trauma, mandibular fractures, and sialadenitis, IV drug abuse, trauma and tonsillitis, and immunocompromised states such as diabetes or HIV/AIDS. Forty percent of Ludwig angina cases involve anaerobes.

◆ Clinical

Signs

The signs of the infection include an inability to close the mouth, trismus, drooling, sitting upright, inability to swallow and dysphonia, dyspnea, stridor, fever, chills, and tachycardia. Note that strider, dyspnea, decreased air movement, or cyanosis suggests impending respiratory compromise.

Symptoms

The symptoms of Ludwig angina are severe neck pain and tenderness, submandibular and submental swelling, fever, malaise, and dysphagia.

Differential Diagnosis

The differential diagnosis involves other deep neck space infections, an infected cyst, tumor, and cellulitis.

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◆ Evaluation

Physical Exam

On physical examination, the patient will often present with carious molar teeth, neck rigidity, and drooling. There is a "woody" or "brawny" induration of involved spaces with little to no fluctuance. The patient's mouth floor is swollen, and the tongue is swollen or elevated.

Imaging

A CT scan is most useful.

Labs

Blood cultures are usually negative; if the swelling is aspirated or drained, samples should be sent for Gram stain, culture, and sensitivity.

◆ Treatment Options

Airway control is the first priority of treatment, followed by IV antibiotics and timely surgical drainage. Blind oral or nasotracheal intubation or attempts with neuromuscular paralysis are contraindicated in Ludwig angina as they may precipitate an airway crisis.

Medical

Aggressive empiric high-dose, IV antibiotics are recommended—cefuroxime plus metronidazole. If the patient is allergic to penicillin, prescribe clindamycin plus a quinolone. Once culture and sensitivity results have been obtained, antibiotic therapy may be changed accordingly.

Surgical

To establish airway control, a tracheotomy may be indicated. Surgical drainage was once universally required but now may be reserved for cases in which antibiotic treatment fails. On external incision and drainage, straw-colored material and not frank pus is often found.

◆ Complications

A spontaneous rupture may lead to asphyxia, aspiration, or pneumonia. The infection may spread to other deep neck compartments.

◆ Outcome and Follow-Up

The offending tooth should also be removed if the infection's origin is odontogenic.

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◆ ICD-9 Code

528.3 Cellulitis and abscess
Cellulitis of mouth (floor)
Ludwig angina
Oral fistula

Further Reading

Har-El G, Aroesty JH, Shaha A, Lucente FE. Changing trends in deep neck abscess: a retrospective study of 110 patients. Oral Surg Oral Med Oral Pathol 1994;77(5):446–450

5.1.1.3 Deep Neck Infections

◆ Key Features

- Deep neck infections are most commonly caused by tonsillar, peritonsillar, or odontogenic infections.
- They may involve surrounding nerves, vessels, bones, and other soft tissue.
- Microbiology typically reveals mixed bacterial flora, including anaerobic species.
- Deep neck spaces have avenues of communication with each other: infection in one space can spread to adjacent spaces.

Deep neck infections hold the potential for severe complications. Complex head and neck anatomy often makes early recognition of deep neck infections challenging, and a high index of suspicion is necessary to avoid delay in treatment. Aggressive monitoring and management of the airway are the most urgent aspects of care, followed by appropriate antibiotic coverage and surgical drainage, as needed. Risk factors for deep neck infection include diabetes mellitus, HIV, steroid therapy, chemotherapy, and other sources of immune compromise.

◆ Clinical

Signs and Symptoms

Pain and swelling of the neck are the most prevalent symptoms. Fever, malaise, and dysphagia may occur. Other common symptoms are deep space specific and include dysphagia, trismus, dysphonia, otalgia, and dyspnea.

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In the pediatric population, fever, neck mass, and stiffness are most prevalent, followed by sore throat, poor oral intake, drooling, and lymphadenopathy. Stridor, dyspnea, decreased air movement, or cyanosis suggest impending respiratory compromise.

Differential Diagnosis

The differential diagnosis includes infected congenital cysts, lymphangitis, tumor, cellulitis, and necrotizing fasciitis.

◆ Evaluation

History, physical examination, laboratory work, and diagnostic imaging each provide important clues when assessing a patient for a deep neck infection.

Physical Exam

Initial evaluation of the airway is always the first priority and any signs of respiratory distress or impending airway compromise should be immediately and aggressively managed.

Imaging

A contrast-enhanced CT scan is most useful. Ultrasound may be more accurate than CT in differentiating a drainable abscess from cellulitis. Magnetic resonance imaging (MRI) provides better soft tissue definition than CT. MRI also avoids exposure to radiation and interference from dental fillings.

Labs

Labs should include a complete blood count (CBC) with differential, serum glucose, and electrolytes; coagulation studies; HIV screening in adults; blood cultures; and appropriate cultures of aspirates obtained before antibiotics are instituted, if possible.

Microbiology

Commonly polymicrobial and reflect the oropharyngeal flora. Frequently isolated aerobes include *Streptococcus viridans*, *Klebsiella pneumoniae*, and *Staphylococcus aureus*, and less frequently, *Streptococcus pneumoniae*, *Streptococcus pyogenes*, *Neisseria* species, and *Haemophilus influenzae*. Common anaerobic isolates include *Peptostreptococcus*, *Bacteroides fragilis*, pigmented *Prevotella* and *Porphyromonas* spp, *Fusobacterium* spp, and *Eikenella corrodens*.

◆ Treatment Options

Airway control is the first priority of treatment, followed by IV antibiotics and timely surgical drainage. Airway management, if necessary, should be undertaken under controlled conditions, if possible in the operating room,

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with either awake fiberoptic intubation or awake tracheotomy. Blind oral or nasotracheal intubation or attempts with neuromuscular paralysis may precipitate an airway crisis.

Medical

Every patient who has a deep neck infection should be given empiric antibiotic therapy until culture and sensitivity results are available. Empiric therapy should be effective against the aerobic and anaerobic bacteria that are commonly involved.

Either penicillin in combination with a β-lactamase inhibitor (e.g., amoxicillin with clavulanic acid) or a β-lactamase–resistant antibiotic (e.g., cefoxitin, cefuroxime, imipenem, or meropenem) in combination with a drug that is highly effective against most anaerobes (e.g., clindamycin or metronidazole) is recommended for optimal empiric coverage. Once available, the results of the culture and sensitivity tests can allow for tailoring of adequate antibiotic therapy.

In select cases, an uncomplicated deep neck abscess or cellulitis can be effectively treated with antibiotics and careful monitoring, without surgical drainage. Simultaneous medical treatment for associated comorbidities such as diabetes mellitus can improve the overall immune status of a patient.

Surgical

Indications for surgery include airway compromise, critical condition, septicemia, complications, descending infection, diabetes mellitus, or no clinical improvement within 48 hours of the initiation of parenteral antibiotics. In addition, abscesses >3 cm in diameter that involve the prevertebral, anterior visceral, or carotid spaces, or that involve more than two neck spaces, should be surgically drained.

Surgical drainage (**Table 5.1**) can be performed in several ways, including simple intraoral or extraoral incision and drainage for superficial abscesses, a more extensive external cervical approach with drain placement for more complicated infections, and minimally invasive techniques such as image-guided needle aspiration and indwelling catheter placement.

◆ Complications

Complications include mediastinitis, aspiration pneumonia, lung abscess, empyema, Lemierre syndrome (suppurative thrombophlebitis of the internal jugular vein), carotid artery aneurysm or rupture, osteomyelitis involving the mandible or cervical vertebral bodies, meningitis, intracranial abscess, and disseminated intravascular coagulation.

◆ Outcome and Follow-Up

The initiating etiology, if recognized (an infected tooth, a tonsillar abscess), and predisposing systemic conditions (diabetes mellitus) should be addressed.

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Table 5.1 Surgical Approaches for Drainage of Deep Neck Infections

Infection Site	Surgical Approaches for Drainage
Peritonsillar	Intraoral needle aspiration or incision and drainage
Submandibular space	Supramylohyoid – intraoral drainage Inframylohyoid – extraoral surgical drainage
Parapharyngeal space	External cervical approach along the anterior border of the sternocleidomastoid muscle CT-guided transoral drainage
Masticator space	External incision along inferior border of mandible Intraoral approach via retromolar trigone
Parotid space	External parotidectomy incision
Retropharyngeal space	Protected airway Transoral
Prevertebral space	External cervical approach
Carotid space	External cervical approach

◆ ICD-9 Codes

478.22 Parapharyngeal abscess
478.24 Retropharyngeal abscess
475 Peritonsillar abscess
Abscess of tonsil
Peritonsillar cellulitis
Quinsy

Further Reading

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5.1.1.4 Neck Trauma

◆ Key Features

- Neck trauma can be either penetrating or blunt.
- There is potential injury to the larynx, trachea, esophagus, major vessels, and nerves.
- Airway management is always the priority.
- Cervical spine injury must be excluded.

Factors in the mechanism of neck trauma determine the location of injury, the injury characteristics, the tissues and organs involved, and the extent of damage to the tissues and organs.

◆ Epidemiology

Neck trauma accounts for 5 to 10% of all serious traumatic injuries. Blunt trauma to the neck typically results from motor vehicle crashes, but it also occurs with sports-related injuries, clothesline injury, strangulation, or blows from the fists or feet. Blunt trauma has become much less common since routine seatbelt use has been established.

For penetrating trauma, >95% of wounds result from guns and knives, with the remainder resulting from motor vehicle accidents, household injuries, industrial accidents, and sporting events. The male to female ratio of penetrating neck trauma is 5:1.

◆ Clinical

Critical organs and structures are at risk from neck trauma; clinical manifestations may vary greatly. The presence or absence of signs and symptoms can be misleading, serving as a poor predictor of underlying damage.

Signs

Signs of airway injury:

- Subcutaneous emphysema – tracheal, esophageal, or pulmonary injury
- Air bubbling through the wound
- Stridor or respiratory distress – laryngeal and/or esophageal injury
- Cyanosis

Signs of vascular injury:

- Hematoma (expanding) – vascular injury
- Active external hemorrhage from the wound site – arterial vascular injury

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- Bruit/thrill – arteriovenous fistula
- Pulselessness/pulse deficit
- Distal ischemia (neurologic deficit in this case)

Signs of pharyngoesophageal injury:

- Hematemesis, inability to tolerate secretions
- Neck crepitus
- Development of mediastinitis

Symptoms

- Clinical manifestations may vary greatly depending on involved organs and systems.
- Dysphagia – tracheal and/or esophageal injury
- Hoarseness – tracheal and/or esophageal injury
- Oronasopharyngeal bleeding – vascular, tracheal, or esophageal injury
- Neurologic deficit – vascular and/or spinal cord injury
- Hypotension – nonspecific; may be related to the neck injury or may indicate trauma elsewhere

Differential Diagnosis

Considerations with neck trauma include cervical spine injury, laryngotracheal injury, vascular injury, and pharyngoesophageal injury.

◆ Evaluation

History

History, if available, can provide important details regarding the mechanism of injury.

Physical Exam

Advanced Trauma Life Support (ATLS) protocols are followed. Exam begins with ABCs (airway, breathing, circulation), followed by secondary assessment once a patient has a safe airway and is hemodynamically stable. All patients with neck trauma should be assumed to have a cervical spine injury until this has been ruled out.

With blunt trauma, injury to the larynx or trachea is the most common serious finding and often presents with subcutaneous air, hoarseness, or odynophagia. In a stable patient, flexible fiberoptic laryngoscopy can reveal evidence of injury such as blood, motion impairment, or edema.

With penetrating trauma, determine which vertical zones of the neck are involved (**Table 5.2**). Zone I extends from the clavicle to the cricoid cartilage; zone II extends from the cricoid to the mandibular angle; zone III extends from the mandibular angle to the skull base.

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Table 5.2 Zones of the Neck and Management of Penetrating Trauma

Zone	Anatomy	Contents	Management
I	Clavicle to cricoid	Common carotid, vertebral, and subclavian arteries and the trachea, esophagus, thoracic duct, and thymus	Angiography, esophagram
II	Cricoid to mandibular angle	Internal and external carotid arteries, jugular veins, pharynx, larynx, esophagus, recurrent laryngeal nerve, spinal cord, trachea, thyroid, and parathyroids	Surgical exploration if symptomatic
III	Mandibular angle to skull base	Distal extracranial carotid and vertebral arteries and the uppermost segments of the jugular veins	Angiography

Imaging

Controversy exists regarding aspects of trauma management, with trends away from surgical exploration in stable patients and expanding use of imaging with observation. There is an inherent delay with any imaging study; transport to the operating room should not be delayed by an imaging study when the patient's condition warrants emergent surgery.

Penetrating zone I and III injuries should undergo angiographic evaluation of the carotids. Zone I injuries should also undergo esophageal studies (i.e., Gastrografin swallow study).

Cervical spine radiography is routine. Review for emphysema, fractures, displacement of the trachea, and the presence of a foreign body.

CT scans prove most useful when bony or soft tissue damage is a consideration. MRI or MR angiography is used for evaluation of the patient exhibiting neurologic impairment with minimal or absent abnormalities of the cervical spine on plain radiograph.

CT angiography offers advantages over angiography. It is readily accessible, can be rapidly performed, and causes fewer complications than angiography. Additionally, some experts assert that subtle disruptions of the vessel wall may be detected on CT angiography. Artifacts secondary to metal can obscure vascular detail and can limit CT angiography.

Labs

As necessary, a trauma panel, CBC, electrolytes, other warranted blood chemistry levels, and blood type and cross-matching should be obtained.

◆ Treatment Options

Establishment of the airway is the first priority. If there is airway compromise, a surgical airway, rather than endotracheal intubation, is usually

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preferred. Either a cricothyroidotomy or tracheotomy is performed if there is respiratory distress. Bleeding is initially managed with direct pressure and establishment of large-bore IV access to permit fluid resuscitation.

- Endoscopy
  - Laryngoscopy, bronchoscopy, pharyngoscopy, and esophagoscopy may be useful in the assessment of the aerodigestive tract. Rigid endoscopes are superior to flexible scopes.
- Angiography
  - Angiography routinely is used to evaluate stable patients sustaining penetrating wounds to zones I and III that pierce the platysma.
  - A four-vessel study is a prerequisite.
  - Drawbacks include cost and the inherent danger of any vascular, particularly arterial, invasive procedure.

The unstable patient (hemodynamic instability, severe hemorrhage, expanding hematoma) is taken to the operating room. The stable patient is categorized as symptomatic or asymptomatic. Signs or symptoms of injury to the airway, esophagus, vessels or nerves (i.e., hemoptysis, hoarseness, dysphagia, crepitus) dictate further workup/interventions. If the penetrating injury is in zone II, neck exploration is generally done; if the injury is zone I, angiography and esophageal studies are performed. If the injury is in zone III, angiography should be performed. Asymptomatic patients are also typically imaged as above if the injury is zone I or III and observed if the injury is zone II.

In general, vascular injuries are managed either with embolization or surgical control. Surgery involves exploration and management of injuries of the carotid sheath, esophagus, and laryngotracheal complex (**Table 5.3**).

There is no role for probing or local exploration of the neck in the trauma bay or emergency room because this may dislodge a clot and initiate uncontrollable hemorrhage.

Table 5.3 Specific Injuries Sought and Treated During Neck Exploration

Carotid artery injuries
Vertebral artery injuries
Jugular vein injury
Laryngotracheal injuries
Esophageal injuries
Nerve injuries
Thoracic duct injuries
Thyroid injuries

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◆ Outcome and Follow-Up

Standard postoperative management for neck surgery is followed. Overall mortality is 1 to 2%.

◆ ICD-9 Codes

959.09	Injury of face and neck
900.9	Injury to unspecified blood vessel of head and neck
957.0	Injury to superficial nerves of head and neck

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5.1.2 Approach to Neck Masses

◆ Key Features

- A neck mass may be inflammatory, congenital, or neoplastic.
- They may be anterior (midline), lateral, posterolateral, or supraclavicular.
- The patient's history should include age, duration, progression, pain, infection, smoking, prior cancer, exposure to tuberculosis, and animal exposure.

Neck masses are relatively common and may present at any age group. The differential diagnosis is broad, and both benign and malignant processes should be considered. A systematic approach is crucial to developing a rapid diagnosis and treatment plan.

Age is an important factor in evaluating a neck mass. The age groups include pediatric, young adult, and older adult (>40 years). Each age group exhibits a certain relative frequency of disease occurrences, which can guide the diagnostician to further differential considerations. In general, neck masses in children are more commonly inflammatory or infectious. This is also true in young adults, although lymphoma is a consideration. In older adults, a neck mass should be considered neoplastic until proven otherwise.

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The location of congenital neck masses is important, as such lesions are often characterized by their location. The location of malignant neck masses particularly if metastatic may help identify the primary tumor (**Fig. 5.5**).

◆ Clinical

Signs and Symptoms

Depending on the cause, the neck mass may be painless (early neoplasm or congenital mass) or painful (infection or trauma). Depending on the etiology, associated symptoms may be those of an upper respiratory infection, toothache (infectious or inflammatory mass) or dysphagia, odynophagia, hoarseness, otalgia, hemoptysis, weight loss, night sweats, and fever (neoplasm).

Differential Diagnosis

See **Table 5.4**.

◆ Evaluation

History

A thorough review of the developmental time course of the mass, associated symptoms, personal habits prior to the trauma or infection, irradiation, or surgery is important. Ask about smoking, tobacco chewing, alcohol use, fever, pain, weight loss, night sweats, exposure to tuberculosis, animals, pets, and occupational/sexual history.

Physical Exam

All mucosal surfaces of the nasopharynx, oropharynx, larynx, and nasal cavity should be visualized by direct examination or by indirect mirror or fiberoptic visualization. State of dentition should be noted. The oropharyngeal surfaces should be palpated. Regarding the neck mass, emphasis on location, tenderness, mobility, and consistency of the neck mass can often place the mass within a general etiologic grouping.

Imaging

CT is important and can better elucidate the nature of a neck mass and its association with surrounding structures. Criteria such as heterogeneity of the center of the mass, blurred borders, and a round shape are suggestive of malignancy. CT may also be able to identify additional neck masses not clinically palpable and/or the primary tumor. Contrast should be used *except* in the suspected thyroid lesion as it may interfere with radioactive-iodine imaging studies or therapy.

MRI is comparable to CT, but it is more expensive and has longer imaging time. With contrast, it is especially good for vascular and neural delineation.

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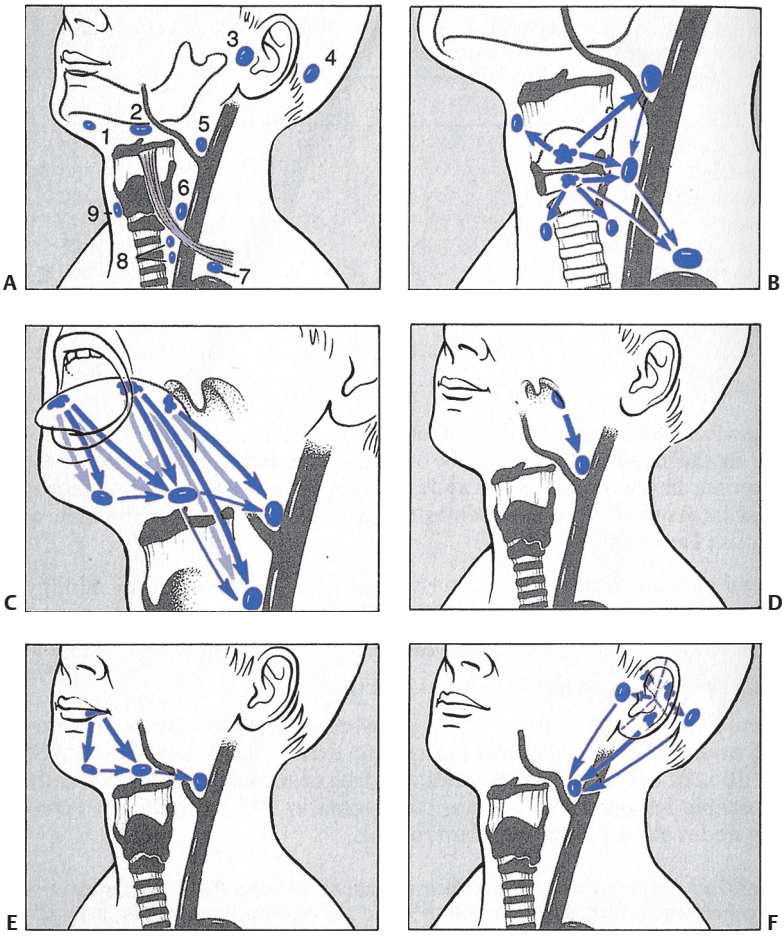

Fig. 5.5 Head and neck masses. **(A)** Typical sites of regional lymph node metastases: 1. submental lymph nodes; 2. submandibular lymph nodes; 3. parotid and preauricular lymph nodes; 4. retroauricular lymph nodes; 5. lymph nodes of the jugulofacial venous angle; 6. deep cervical lymph nodes; 7. lymph nodes in the jugulоclavicular venous angle: lower deep cervical lymph nodes and supraclavicular lymph nodes; 8. pre- and peritracheal lymph nodes; 9. prelaryngeal lymph nodes. **(B)** Laryngeal carcinoma. **(C)** Carcinoma of different parts of the tongue. Note the tendency to contralateral metastases. **(D)** Tonsillar carcinoma. **(E)** Lower lip carcinoma. **(F)** Carcinoma of the external ear. Note the segmental lymphatic efferent from the auricle. *(continued on p. 344)*

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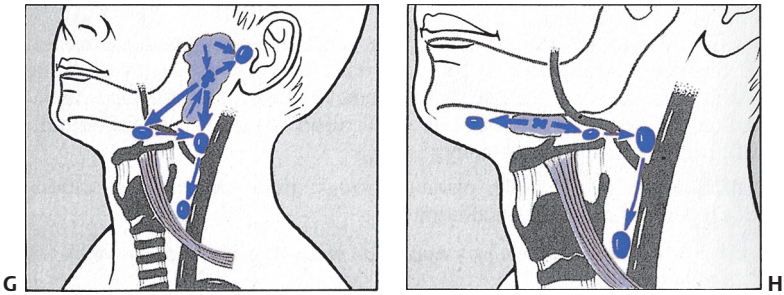

Fig. 5.5 *(continued)* **(G)** Parotid carcinoma. Note the intraglandular lymph node metastases. **(H)** Submandibular gland carcinoma. (From Becker W, Naumann HH, Pfaltz CR. Ear, Nose and Throat Diseases: A Pocket Reference. 2nd ed. Stuttgart/New York: Thieme; 1994:516.)

Angiography is recommended if primary vascular neck mass is suspected and synchronous embolization is being considered.

Ultrasound is helpful in differentiating solid from cystic masses and congenital cysts from solid lymph nodes and glandular tumors.

Labs

A routine complete blood count test may reveal an infectious process. More specialized laboratory tests may become necessary as the investigation proceeds.

Table 5.4 Differential Diagnosis of Neck Masses

Congenital	Neoplastic Malignant	Neoplastic Benign	Systemic, Inflammatory and Infectious Disease
Branchial cleft cyst	Head and neck metastasis	Hemangioma	Lymphadenitis – bacterial, viral
Dermoid cyst	Thyroid	Thyroid: neoplasm or goiter	Deep neck abscess
Thyroglossal duct cyst	Lymphoma	Lymphangioma	Cat scratch disease
Laryngocele	Salivary gland tumor	Salivary gland tumor	Tuberculosis or atypical mycobacterium
Ectopic thyroid tissue			Sarcoidosis, Kikuchi-Fujimoto disease, Kimura disease, Castleman disease
		Lipoma Carotid body tumor or paraganglioma	Sialadenitis

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Other Tests

- Fine-needle aspiration biopsy (FNAB): Currently, FNAB is the standard of diagnosis for neck masses and is indicated in any neck mass that is not an abscess and persists despite appropriate antibiotic therapy. FNAB separates inflammatory and reactive processes that usually do not require surgery from neoplastic lesions, either benign or malignant.
- Panendoscopy: If careful examination in the office does not identify the etiology of the neck mass and a tumor is suspected, the upper aerodigestive tract should be examined under anesthesia. Biopsies should be performed on any suspicious mucosal lesions.

◆ Treatment Options

Medical

A tender, mobile mass or one highly suggestive of inflammatory or infectious etiology may warrant a short clinical trial of antibiotics and observation with close follow-up. Use steroids judiciously; steroids may shrink a neck mass caused by lymphoma lulling the physician and patient into a false sense that the condition is improving.

Surgical

- Open excisional biopsies should be avoided in cases in which a nonlymphoma malignancy (epidermoid, melanoma) is suspected. If FNAB results are negative or equivocal but suspicion for malignancy persists, an open excisional biopsy of the cervical lymph node may be performed. The patient and surgeon should be prepared to proceed immediately with a complete neck dissection depending on the results of frozen sections.
- Open excisional biopsies may be performed for lymphomas or granulomatous disease.
- Inflamed congenital masses are typically treated with antibiotics and then surgically removed after inflammation has subsided.
- Infectious processes causing neck masses are treated medically. Surgery in the form of incision and drainage is used in cases that do not respond to appropriate medical therapy.

◆ ICD-9 Code

784.2 Swelling mass or lump in head and neck

Further Reading

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5.2 Head and Neck Cancer

◆ Key Features

- Head and neck cancer is a heterogeneous group of tumors, consisting predominantly of squamous cell carcinoma (SCC) of the upper aerodigestive tract.
- This type of cancer is linked to tobacco smoking and alcohol.
- There is a propensity for second primary tumors (between 4 and 7% per year), especially if still smoking.
- Multimodality treatment includes surgery, radiation, and chemotherapy.
- Adult with persistent neck mass is SCC until proved otherwise.

Head and neck cancer, predominantly SCC, can affect the oral cavity, pharynx, larynx, hypopharynx, cervical esophagus, nose, and paranasal sinuses. The goal of treatment is cure or palliation with preservation of function. Specific sites and subsites of head and neck cancer are discussed in subsequent chapters.

◆ Epidemiology

Head and neck SCC accounts for ~5% of cancers in the United States. This corresponds to an estimated 17 per 100,000 Americans with newly diagnosed SCC of the head and neck per year. These cancers are more common in men, and typically occur in patients over age 50. The etiology includes tobacco use (smoking and smokeless) and alcohol consumption. Eighty-five percent of head and neck SCC are linked to tobacco use. The synergistic effect of alcohol and smoking increases the risk of disease many more times than the simple additive risk of either risk factor alone. In SCC, mutations in the p53 gene correlate with drinking and smoking habits. Some 15% of patients have a viral etiology. Epstein-Barr virus (EBV) has been implicated in the development of nasopharyngeal carcinoma. Human papillomavirus (HPV) infection is another factor implicated in the carcinogenesis of upper aerodigestive tract tumors. In particular, HPV-16 can be isolated in up to 72% of oropharyngeal cancers. The recent increase in cancers of the tongue and tonsils in developed countries, particularly in younger patients, has been linked to HPV infection.

During the past 20 years, the overall incidence of head and neck SCC has been declining in the United States, a decline that is attributed to a decrease in the prevalence of smoking. In other parts of the world, head and neck SCC is attributed to habitual and cultural habits such as chewing of paan (betel leaf with areca nut), smoking khat (*Catha edulis*), and drinking yerba maté.

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◆ Clinical

Signs

Signs may include hoarseness, muffled speech, trismus, and recurrent epistaxis. Many patients present with a neck mass as chief complaint, representing metastatic nodal disease from an occult primary tumor in the upper aerodigestive tract (**Table 5.5**).

Symptoms

Symptoms of head and neck SCC are variable and depend on the site and stage of the primary tumor (see Staging of Head and Neck Cancer, below). Early symptoms may be vague and mimic benign disease and are therefore only discovered at advanced stages of disease. Symptoms may include dysphagia, odynophagia, a globus sensation, changes in voice (this includes both hoarseness and velopharyngeal insufficiency), referred otalgia, CN hypesthesia, nasal obstruction, epiphora, and hyposmia.

Differential Diagnosis

- Upper respiratory infections such as pharyngitis, laryngitis, deep neck infections or abscesses
- Congenital masses and cysts
- Upper airway manifestations of rheumatologic and autoimmune diseases
- Hematologic malignancies (lymphoma)
- Tuberculosis
- Fungal infections

Table 5.5 Incidence of Cervical Lymph-Node Metastases Associated with Carcinomas of the Upper Aerodigestive Tract

Tumor Location	Incidence of Cervical Lymph-Node Metastases at Diagnosis (%)
Oral cavity	30–65
Oropharynx	39–83
Nasopharynx	60–90
Hypopharynx	52–72
Supraglottis	35–54
Glottis	7–9
Nasal cavity and paranasal sinuses	10–20
Salivary glands	25–50
Thyroid gland	18–84*

Source: Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2009:333.
*Depends on age and histologic subtype.

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◆ Evaluation

History

History should include questions about risk factors, breathy voice or prolonged hoarseness, dysphagia, hemoptysis, otalgia, and unintentional weight loss.

Physical Exam

Physical exam should include careful inspection of the oral and oropharyngeal mucosa for lesions, and palpation of the tonsillar region and tongue base for firm nodules or masses.

An indirect mirror or flexible fiberoptic laryngoscopy should be performed. During this examination, the patient should be asked to perform several maneuvers such as tongue protrusion, puffing out the cheeks, lightly coughing and speaking to better visualize and access the larynx and the hypopharynx. It is important that laryngeal motility be assessed, as this is critical in tumor staging. The neck should be examined in a systematic fashion. Any palpable lymph nodes should be assessed with regard to size, location, and mobility.

Imaging

A contrast-enhanced CT scan or MRI scan of head and neck should be obtained to assess local and regional extent of disease and involvement of adjacent structures, such as the great vessels of the neck and the prevertebral fascia. Specific characteristics of regional lymphadenopathy, if present, should be noted, such as extracapsular spread, central necrosis, and size of involved lymph nodes.

A metastatic workup may consist of either a chest radiograph with liver function tests, CT scan of the chest and abdomen, or alternatively, a fluorodeoxyglucose-positron emission tomography (FDG-PET) scan.

Labs

Blood count, electrolyte, and liver function tests should be performed to assess nutritional status.

Other Tests

FNAB is a highly accurate technique for the investigation of cervical lymph node metastases in head and neck SCC and is the first-line test in a patient with lymphadenopathy. Sensitivity of this test is improved when performed with ultrasound guidance. It is best to avoid open biopsy of a neck mass, as tumor spillage and violation of fascial planes is problematic.

Patients in whom a suspicion of head and neck SCC exists should undergo biopsy of suspicious primary site lesions. Because of the propensity for second primary tumors that accompany head and neck SCC, these patients should undergo a panendoscopy (i.e., triple endoscopy),

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laryngoscopy, esophagoscopy, and bronchoscopy together, to search for synchronous lesions. This may also lend insight as to the extent of the primary lesion, particularly important in the smoking patient. This is a point of controversy, and PET scanning may play a greater role in the future. Mention should be made that in the current literature, the sensitivity of PET scanning is not adequate to replace panendoscopy: for instance, the larynx has baseline activity on PET due to patient breathing and/or talking during the scan.

Pathology

Ninety percent of head and neck cancers are SCCs.

◆ Treatment Options

Treatment of head and neck SCC consists of surgery, radiotherapy, chemotherapy, or a combination of these. Surgical resection remains the gold standard for treatment of head and neck cancer. Surgery may address the primary tumor as well as cervical metastasis.

Radiation therapy for SCC of the head and neck involves the delivery of high-energy ionizing radiation to targeted tissues. Radiation doses can be delivered by different methods, including fractionation, hyperfractionation, accelerated fractionation, and intensity-modulated radiotherapy (IMRT).

Chemotherapy for SCC of the head and neck involves the systemic administration of cytotoxic drugs that target rapidly dividing cells. Individual chemotherapeutic agents effective in the therapy of head and neck cancer include cisplatin, methotrexate, 5-fluorouracil, taxanes, ifosfamide, and bleomycin.

Of the molecularly targeted agents, cetuximab is an IgG1 chimeric antibody directed against the epidermal growth factor receptor (EGFR).

Other therapy methods for head and neck cancer include photosensitizers and interstitial laser therapy, photodynamic therapy, immune therapy, gene therapy, and targeted therapy against EGFR and HPV.

◆ Cancer of Unknown Primary

Patients with head and neck cancer typically present with a painless neck mass. In 2 to 8% of these patients, the tumor origin is not known. This is known as cancer of unknown primary (CUP), carcinoma of unknown primary origin, or occult primary malignancy. A primary tumor is considered unknown only *after* a thorough investigation (including physical exam, imaging, and biopsies) has been completed.

Evaluation of possible CUP begins with a thorough history and physical examination, including flexible fiberoptic laryngoscopy. FNAB of the neck mass provides a histologic diagnosis, which may assist with finding the primary tumor, but 90% are attributable to SCC.

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Once a diagnosis of cancer has been established, imaging studies can assist with localization of the primary site. CT is faster and more cost-effective, but MRI has a higher sensitivity for small tumors given its better soft tissue delineation. PET-CT may be considered and may help rule out primary sights below the diaphragm. It has recently been recommended that imaging evaluation begin with a CT and chest x-ray followed by MRI or PET-CT if the primary site is still not found.

The next step is to perform panendoscopy with biopsies whether or not the primary site was located on imaging. As noted earlier, panendoscopy typically included bronchoscopy, esophagoscopy, and direct laryngoscopy. If no obvious tumor is visualized, tonsillectomy and guided biopsies are performed. Some advocate a unilateral tonsillectomy limited to the side of the neck mass, but others advocate bilateral tonsillectomies in this circumstance. The most common sites of CUP include the palatine tonsil and base of tongue, followed by the nasopharynx and pyriform sinus. Each of these sites should at least be inspected with consideration of guided biopsies.

Treatment of CUP is controversial. Surgical excision in the form of a neck dissection followed by radiation therapy allows for a lower total dose of radiation. Primary radiation therapy provides treatment to both the upper aerodigestive tract and its locoregional metastasis but forces the radiation oncologist to treat a wider field as the primary site is unknown.

◆ Outcome and Follow-Up

The treatment of head and neck SCC, whether surgical or chemoradiation, often leaves the patient with significant speech and swallowing deficits. Therefore, is it paramount for a multidisciplinary team to treat head and neck SCC. This should include the surgical oncologist, medical and radiation oncologist, oral surgeon, prosthodontist, speech language and swallowing pathologist, nurse, and social worker.

Patients with regional neck disease prior to treatment should have a CT scan or an integrated FDG-PET/CT. There is controversy as to the need for a planned neck dissection following radiotherapy in patients with high-risk disease. Most recurrences of SCC occur within 3 years of the initial treatment. Patients should be seen at 1- to 2-month intervals for 2 years after treatment. Thereafter, they should be seen at 3-month intervals for the next year. There is no evidence that routine follow-up beyond 3 years improves prognosis, although many clinicians support yearly follow-up.

Patients should be told of the risk of a second primary tumor and encouraged to report any new symptoms. The risk of a second primary carcinoma is highest in those who continue to smoke. Patients should be counseled for tobacco cessation.

◆ ICD-9 Code

199.1 Other malignant neoplasm of unspecified site

[See subsequent chapters for appropriate codes for cancers of specific sites.]

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◆ Staging of Head and Neck Cancer

- The regional lymph node metastases and their effect on stage grouping are fairly consistent throughout all anatomic sites of head and neck cancer.
  - NX – Regional lymph nodes cannot be assessed.
  - N0 – No evidence of regional lymph node metastasis
  - N1 – Metastasis to a single ipsilateral lymph node measuring ≤3 cm in greatest diameter
  - N2 – Further divided into three categories:
    - N2a – Single ipsilateral lymph node between 3 and 6 cm
    - N2b – Multiple ipsilateral lymph nodes <6 cm
    - N2c – Bilateral or contralateral lymph nodes <6 cm in greatest dimension
  - N3 – Lymph node >6 cm
- Distant metastatic disease is divided into two categories:
  - M0 – Absence of distant disease
  - M1 – Presence of distant metastatic disease
- The T stage of a tumor indicates the extent of the primary tumor and varies by anatomic subsite. This can be measured by size, as in the oral cavity, oropharynx, and salivary glands; by involvement of varying subsites, as in the nasopharynx, hypopharynx, and larynx; or by extent of invasion and destruction, as in the maxillary sinus. Across all anatomic sites of the head and neck, the following classifications apply:
  - Stage I disease – Includes only T1 N0 M0 tumors
  - Stage II disease – Includes T2 N0 M0 tumors
  - Stage III disease – Includes T3 N0 M0 and T1–3 disease, which is N1 M0
  - Stage IV disease – Includes T4 tumors with or without nodal disease, as well as any tumor with N2 or N3 disease or evidence of distant metastatic disease

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5.2.1 Chemotherapy for Head and Neck Cancer

◆ Key Features

- Concurrent chemotherapy with definitive radiotherapy is a safe and effective means to treat locally advanced SCC of the head and neck.
- Concurrent chemotherapy with postoperative radiotherapy (i.e., chemoradiotherapy) improves survival in select high-risk patients.
- Palliative chemotherapy can reduce symptoms and modestly extend survival in an incurable setting.
- Newer biologic and cytotoxic agents continue to cause the treatment of head and neck cancer to evolve.

The role of chemotherapy in head and neck cancer is expanding and its utility varies with the stage of the disease. For patients with metastatic or incurable locoregional disease, chemotherapy is palliative. For patients with potentially curable locoregional head and neck cancer, chemotherapy is an integral component of the multimodality approach.

Chemotherapy in the definitive treatment of head and neck cancer is an adjuvant therapy. Strictly defined, an adjuvant therapy is an addition to the potentially curable modality (primary surgery or definitive radiation) that improves outcomes. Broadly speaking, adjuvant therapies can be preoperative (or preradiotherapy), concurrent with radiation, or postoperative (or postradiotherapy). Most early adjuvant chemotherapy trials in cancer were postoperative in nature, so "adjuvant therapy" has also been used to describe only postoperative (or postradiotherapy) chemotherapy. This has given rise to the term "neoadjuvant" chemotherapy to describe preoperative (or preradiotherapy) chemotherapy. Induction chemotherapy and neoadjuvant chemotherapy are synonymous.

◆ Neoadjuvant Chemotherapy

Advantages of neoadjuvant chemotherapy (i.e., induction chemotherapy) include an intact vascular bed for better drug delivery, reduced tumor bulk to improve the ease of resection, and early eradication of regional and distant micrometastases. Disadvantages include delaying surgery in potentially curable patients with chemoresistant disease, relying on clinical staging to make treatment decisions, the morbidity of "overtherapy," and patient nonadherence after chemotherapy. Neoadjuvant cisplatin and 5-fluorouracil (5-FU) followed by radiotherapy in responders was an organ preservation strategy described in the frequently cited VA Laryngeal Cancer Study Group. Subsequent results have shown this approach to be inferior to concurrent cisplatin with radiation, but newer induction regimens including docetaxel have reintroduced neoadjuvant chemotherapy followed by radiotherapy as a viable option. Neoadjuvant chemotherapy before surgery has not been found to be helpful in randomized trials.

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◆ Concomitant Chemoradiotherapy

The simultaneous use of chemotherapy and radiation continues to be the standard for locally advanced SCC (stages III to IVb). The primary benefit has been in decreasing locoregional failure, which has translated into roughly a 10% overall survival benefit. The effect on decreasing metastatic disease has been inconsistent. It is believed that chemotherapy may have some benefit against radioresistant hypoxic tumor cells. However, the simultaneous use of chemotherapy and radiotherapy has significantly increased grade 3 and 4 toxicity, which can be potentially lethal or cause treatment breaks that decrease radiation's efficacy. In patients who are receiving surgery and are found to have high-risk features (positive margins, N2 disease, nodal extracapsular extension), postoperative cisplatin with radiation has proven superior to radiation alone. Cisplatin, 5-FU, taxanes, and mitomycin C all act as radiosensitizing agents.

◆ Adjuvant Therapy

The use of postoperative or postradiation chemotherapy has not been found to be helpful in randomized trials, although it is commonly done for three cycles in nasopharyngeal cancer based on the Intergroup Trial showing concurrent cisplatin with radiation followed by three cycles of cisplatin plus 5-FU improved survival over radiation alone. It is controversial whether the cycles given after radiation add any independent benefit.

◆ Types of Chemotherapeutic Agents Used for Head and Neck Cancer

Alkylating Agents

The cytotoxic effects of alkylating agents (e.g., cisplatin) are based on their interaction with DNA. These agents cause substitution reactions, cross-linking reactions, or strand-breaking reactions. These agents alter the information coded in the DNA molecule resulting in inhibition of or inaccurate DNA replication with resultant mutation or cell death.

Antimetabolites

The cytotoxic effect of antimetabolites (e.g., methotrexate) is due to their structural similarity to naturally occurring metabolites involved in nucleic acid synthesis. They inhibit critical enzymes involved in nucleic acid synthesis and become incorporated into the nucleic acid and produce incorrect codes. Both of these mechanisms result in an inhibition of DNA synthesis and ultimate cell death.

Antitumor Antibiotics

Antitumor antibiotics (e.g., mitomycin, bleomycin) are antimicrobial compounds produced by *Streptomyces* species in culture. They are cytotoxic in that they affect the structure and function of nucleic acids by intercalation between DNA base pairs (doxorubicin), DNA strand fragmentation, or cross-linking of DNA.

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Alkaloids

Alkaloids (e.g., vincristine, vinblastine) bind to free tubulin dimers and disrupt the balance between microtubule polymerization and depolymerization, resulting in the net dissolution of microtubules, destruction of the mitotic spindle, and arrest of cells in metaphase.

Taxanes

Taxanes (e.g., paclitaxel, docetaxel) are compounds that disrupt equilibrium between free tubulin and microtubules, causing stabilization of ordinary cytoplasmic microtubules and the formation of abnormal bundles of microtubules.

EGFR Inhibitors

EGFR is a protein found on the surface of some cells and to which epidermal growth factor binds, causing the cells to divide. It is found at abnormally high levels on the surface of many types of cancer cells, so these cells may divide excessively in the presence of epidermal growth factor (EGFR, ErbB1, and HER1). Cetuximab (Erbitux, Bristol-Myers Squibb, New York, NY) specifically targets EGFR and binds to EGFR with higher affinity than its natural ligands. Binding results in the internalization of the antibody receptor complex without activation of the intrinsic tyrosine kinase. Consequently, signal transduction through this cell pathway is blocked, which inhibits tumor growth and leads to apoptosis.

During a recent multinational, randomized study to compare radiotherapy alone with radiotherapy plus cetuximab in patients with locoregionally advanced head and neck cancer, cetuximab was found to improve locoregional control and reduce mortality.

◆ Complications

Each drug or combination of chemotoxic drugs can cause specific side effects, and some can be permanent. These side effects may be so severe that chemotherapy must be ceased. In general, chemotherapy may cause the following side effects: fatigue, nausea, vomiting, hair loss, xerostomia, anorexia, immunocompromise, diarrhea, mucositis, and death.

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5.2.2 Radiotherapy for Head and Neck Cancer

◆ Key Features

- Definitive radiotherapy is a safe and effective means to treat various cancers of the head and neck, either in inoperable patients or as an alternative to surgery for organ preservation.
- Postoperative radiotherapy decreases local failure in select high-risk patients.
- Palliative radiotherapy can reduce local symptoms in an incurable setting.
- Radiation can be improved by sensitizing tumor cells preferentially or by decreasing radiation damage to normal tissues.

Ionizing radiation is a locoregional therapy whereby photons (gamma rays or x-rays), electrons, neutrons, protons, or heavier particles (mesons, α particles, carbon ions) cause cells to either undergo death during mitosis or apoptosis, primarily through the creation of DNA double-strand breaks. The therapeutic ratio of radiation depends on the difference in sublethal repair between normal tissues and tumor cells, the use of radioprotectors and/or radiosensitizers, and the use of advanced methods to limit the irradiation of normal tissues.

◆ Fundamental Concepts of Radiation

Radiation dose is defined as the amount of energy (joule) imparted per unit mass (kg). The SI metric unit of dose is the Gray (Gy), defined as 1 J/kg. Historically, the unit used was the rad, which is equivalent to 0.01 Gy, or 1 cGy.

Each radiation treatment is called a fraction because for most situations the total radiation dose is given over multiple sessions. A standard fraction is 1.8 to 2 Gy per fraction, and a standard course is five fractions per week with one fraction given per day. Fractionation is biologically advantageous because of the processes of tumor reoxygenation and reassortment into more radiosensitive parts of the cell cycle. Increasing the number of fractions preferentially spares normal tissues by giving them more time to repair sublethal damage. Increasing the number of fractions cannot be

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extended indefinitely because of tumor repopulation, which significantly reduces radiation's efficacy if the total treatment time exceeds 7 weeks.

Various alternative fractionation strategies have been used to try to enhance radiation's effectiveness. Accelerated radiation delivers treatment faster than standard fractionation (>10 Gy per week). Hyperfractionated radiation is the use of fraction sizes smaller than 1.8 Gy. Hypofractionated radiation is the use of fraction sizes larger than 2.0 Gy. These strategies can be combined, as in accelerated hyperfractionation.

◆ Methods of Radiation Delivery

Radiation is broadly divided into brachytherapy and teletherapy. Brachytherapy is the placement of radioisotopes near or inside the target. In SCC of the head and neck, this is most commonly done by placing catheters in a tumor or operative bed and using an afterloading device to push the source into the catheters for predetermined periods of time to deliver a prescribed dose to the entire target volume. The exposure time ranges from over 2 to 3 days in low-dose rate applications, most commonly with cesium-137, to 10 to 30 minutes in high-dose rate applications, most commonly with iridium-192. With differentiated thyroid cancer, orally administered iodine-131 (^{131}I) preferentially binds to tumor cells, with ablative doses of 100 to 150 mCi delivering 250 to 300 Gy.

Teletherapy, or external beam radiation, is the delivery of radiation by pointing an external source of radiation at the target. The most common source in modern radiotherapy is the linear accelerator, which can generate high energy (4 to 25 MeV) photons and electrons. Gamma knife radiosurgery units use cobalt-60 sources that emit 1.25-MeV photon beams. Intraoperative radiation can be focally delivered to internal structures with a linear accelerator or portable x-ray generator in the operating room. External beam radiation is further subdivided by the technology used.

Conventional radiation planning uses x-ray films to define the target volume. Plans are generally limited to a small number of angles and radiation beams are shaped by fabricating Cerrobend blocks. Three-dimensional (3D) conformal radiation uses CT-based treatment planning systems to improve target identification and evaluate dose distribution more accurately. This increases dose conformality to the target by making it easier to use more fields from virtually any beam angle. IMRT improves dose conformality further by delivering different doses to different sections within the same beam, and optimizes the choice and intensity of beams by using a software algorithm to simultaneously test more plans than a human could do within a reasonable period of time. Image-guided radiotherapy (IGRT), further improves dose conformality by using real-time imaging to confirm the patient is in the appropriate position on the couch before delivering radiation, thereby decreasing set-up error and allowing for tighter margins. Stereotactic radiosurgery, or SRS, is the use of a highly conformal large single fraction of external beam radiotherapy, either using a gamma knife or linear accelerator. A gamma knife uses cobalt-201 sources aimed at the same point in space to produce a small area with a high dose and sharp dose drop-off. A common

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trait of all modern systems is that increased dose conformality to the target requires a high level of patient set-up consistency, and this is achieved using custom masks or external frames that connect to the patient couch.

◆ Rationale for Definitive (Curative) Radiotherapy

Primary radiotherapy in the treatment of SCC of the nasopharynx, oropharynx, oral cavity, and glottis has long been considered an option even in resectable disease. The primary justification for this is not increased efficacy over surgery but organ and functional preservation without compromising long-term efficacy. This is an option for patients with both early (stage I or II) and advanced (stage III or IV) disease. For patients with advanced disease, definitive radiation with chemotherapy with or without planned neck dissection, with surgery to the primary reserved for salvage, had an equivalent survival compared with surgery followed by radiation in randomized trials of cancers of the larynx, hypopharynx, and other areas of the pharynx. For patients with early-stage lesions of the larynx, no randomized trials of laryngectomy versus other modalities exist, but a large series of mature data exists regarding the long-term efficacy of definitive radiation. The results of these trials cannot be extrapolated to all cases, and it is likely that surgery should be the primary modality in some patient subsets. Tumor control, functional outcome, and quality of life should be considered by a multimodality treatment team before choosing an individual patient's treatment plan.

Definitive radiation, with or without chemotherapy depending upon the histology, is also used in mucosal melanoma, skin cancer, salivary gland cancer, lymphoma, and plasmacytoma. In select cases, conformal radiation using IMRT, SRS, or brachytherapy can be used in previously irradiated sites to salvage locally recurrent cases.

◆ Rationale for Adjuvant Radiotherapy

Postoperative radiotherapy is used if there is residual disease or a significant risk of occult residual disease. Randomized evidence supports the use of postoperative radiation for SCC that is stage III or IV or that has close or positive margins. The addition of current chemotherapy to adjuvant radiation has proven to be better than radiation alone in large randomized trials. Randomized data for other tissue types does not exist, but postoperative radiation is commonly given in high-risk cases of Merkel cell carcinoma, salivary gland carcinoma, skin cancer, and thyroid cancer. Preoperative radiation is generally reserved for marginally unresectable disease, but is more standard in esthesioneuroblastomas to make the definitive surgery smaller and less morbid.

◆ Rationale for Palliative Radiotherapy

In the noncurative setting, radiotherapy is used to treat areas that are causing local symptoms or at a high risk to cause local symptoms. Common indications in head and neck cancer to treat the primary lesion include uncontrolled bleeding, pain, dysphagia, and a compromised airway. Metastatic disease to the bone, brain, and lung can also be palliated effectively using radiation.

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◆ Complications

Radiation side effects can be characterized as acute or late. Acute effects occur during or within the first few weeks after radiotherapy and tend to be transient. Late effects occur months to years after treatment and tend to be permanent. Common acute side effects include dermatitis, mucositis, taste changes, xerostomia, fatigue, facial hair loss, decreased sweating, anorexia, and weight loss. Less common acute effects include cough, hoarseness, nausea, and sialadenitis. Common late effects include xerostomia, trismus, hypothyroidism, soft tissue fibrosis, dysphagia, and taste changes. Less common late effects include soft tissue necrosis, osteoradionecrosis, laryngeal edema, spinal cord myelopathy, carotid stenosis, and second malignancy. Acute effects are generally managed supportively because of their transient nature. Aggressive dental support, stretching exercises, and proper skin care can minimize some late effects. Routine evaluation for hypothyroidism and xerostomia should also be performed, as pharmacologic interventions can improve these conditions.

◆ Improving the Therapeutic Ratio of Radiation

Radiation can be improved by sensitizing tumor cells preferentially or by decreasing radiation damage to normal tissues. Hyperfractionation and accelerated radiation regimes have improved outcomes in stage III or IV SCC compared with standard fractionation, and hypofractionation has improved local control in early-stage glottic lesions. Radiation sensitizers with proven efficacy in randomized trials include concurrent platinum agents, mitomycin C, and cetuximab. Normal tissues can be spared using IMRT, submandibular gland transfer, and amifostine. Future improvements are expected as imaging, radiation delivery, and new agents continue to be further developed.

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5.2.3 Sinonasal Cancer

◆ Key Features

- Sinonasal cancer initially may mimic benign sinus disease.
- Tumors of the paranasal sinuses often present with advanced disease.
- Cure rates are generally ≤50%.
- Most patients die of direct extension into vital areas.

Malignant tumors of the sinonasal tract are extremely rare, accounting for 0.2% of all invasive cancers and 3% of head and neck cancers. Cancers of the maxillary sinus are the most common. Tumors of the ethmoid sinuses are less common (20%), and cancers of the sphenoid and frontal sinuses are rare (<1%). Local extension often makes it difficult to access the sinus of origin.

◆ Epidemiology

Chemical carcinogens such as chromium, nickel, thorium dioxide, and tanning chemicals have been implicated in the development of carcinoma of the paranasal sinuses. Exposure to wood dust has been implicated specifically in adenocarcinoma of the ethmoid. Interestingly, tobacco use was previously thought not to play a role in sinonasal carcinogenesis. However, up to a fivefold increased risk of sinonasal carcinoma has been observed with heavy smoking. Rarely, sinonasal cancers may present as a second primary tumor in tobacco users with other head and neck cancers.

◆ Clinical

Signs and Symptoms

Clinical presentation of sinus malignancies is nonspecific and often mimics benign disease, thus diagnosis is often delayed for months. Key indicators of malignancy are cranial neuropathies, proptosis, and pain of maxillary dentition; trismus, palatal, and alveolar ridge fullness; or frank erosion into the oral cavity. Symptoms include nasal obstruction, discharge, stuffiness, congestion, epistaxis, unilateral tearing, diplopia, exophthalmos, infraorbital nerve hypesthesia, cheek swelling, facial asymmetry, hearing loss, and serous otitis media due to nasopharyngeal extension may occur.

Differential Diagnosis

The differential diagnosis includes benign sinus disease, benign sinus tumors, and metastatic tumors to the sinus.

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◆ Evaluation

History

The patient history should include known carcinogen exposure, tobacco usage, and prolonged benign sinus symptoms and signs.

Physical Exam

A complete head and neck examination, including nasal endoscopy, should be performed. The sinonasal, ocular, and neurologic systems should be studied in detail. Evidence of nerve hypesthesia, diplopia, proptosis, and loose dentition should be carefully evaluated. Suspicious lesions should be biopsied.

Imaging

Imaging should include either a contrast enhanced CT scan or MRI. There may be a role for integrated FDG-PET/CT.

Other Tests

A definitive diagnosis requires a biopsy. Special attention should be paid to CN function because malignant paranasal tumors are associated with a high incidence of cranial neuropathies compared with inflammatory or benign sinus disease.

Pathology

SCC is the most frequent type of malignant tumor in the paranasal sinuses (70–80%). Minor salivary gland tumors constitute 10 to 15% of these neoplasms. Some 5% of cases are lymphomas. Other tumors include sinonasal undifferentiated carcinoma (SNUC), chondrosarcoma, osteosarcoma and malignant melanoma, and esthesioneuroblastoma.

Inverted papilloma, a benign tumor with a tendency to recur (see Chapter 3.4), may transform into a malignant SCC of the paranasal sinuses in a small percentage of cases.

◆ Treatment Options

Most stage T1 or T2 maxillary sinus carcinomas are treated by surgery alone, provided adequate resection margins are obtained. This may be en bloc surgical resection or endoscopic sinus surgery, depending on the extent of disease and experience of the surgeon. The specific approach is determined by the location of disease and histology (**Fig. 5.6**).

T3 and T4 lesions are treated by combination therapy with surgery and radiation. The issue regarding whether radiation is more effective before or after surgery remains controversial. Chemotherapy alone is generally used as a palliative measure.

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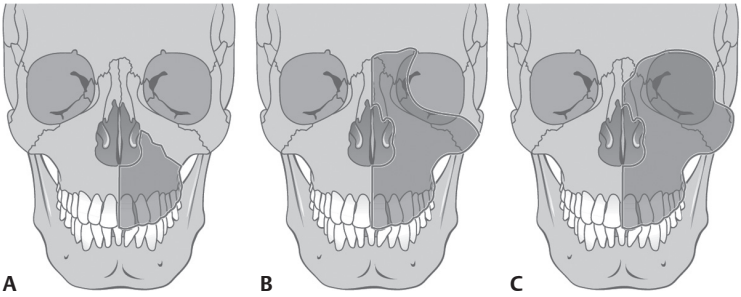

Fig. 5.6 Resection of the upper jaw. **(A)** Partial resection. **(B)** Total resection. **(C)** Total resection with exenteration of the orbit. (From Behrbohm H, Kaschke O, Nawka T, Swift A. Ear Nose and Throat Diseases: With Head and Neck Surgery. 3rd ed. Stuttgart/New York: Thieme; 2009:227.)

◆ Outcome and Follow-Up

Malignancy behind Öhngren's plane is regarded to carry a much poorer prognosis because of the rapid spread to the orbit and middle cranial fossae (**Fig. 5.7**). Despite improvements in surgical ablative and reconstructive techniques, radiation delivery modalities, and imaging technologies, disease-free survival at 5 years remains <50%, independent of stage. Five-year disease-free survival for patients with advanced stage cancer drops to 25%.

◆ ICD-9 Codes

160.2 Malignant neoplasm of maxillary sinus
160.3 Malignant neoplasm of ethmoidal sinus
160.4 Malignant neoplasm of frontal sinus
160.5 Malignant neoplasm of sphenoidal sinus

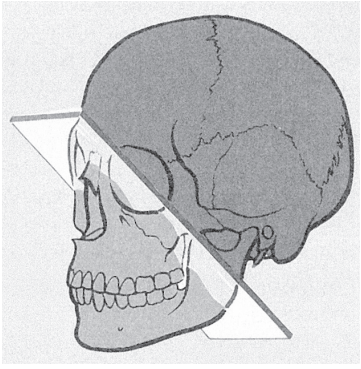

Fig. 5.7 Öhngren's plane passing through the medial canthus and the mandibular angle. It divides the maxillary sinus into a superoposterior part and an inferoanterior part. Cancer limited to the latter part typically carries a better prognosis. (From Becker W, Naumann HH, Pfaltz CR. Ear, Nose, and Throat Diseases: A Pocket Reference. 2nd ed. Stuttgart/New York: Thieme; 1994:293.)

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◆ Staging of Nose and Paranasal Sinus Cancer: For All Carcinomas Excluding Mucosal Malignant Melanoma

Primary Tumor: Maxillary Sinus

TX: Cannot be assessed

T0: No evidence of primary tumor

Tis: Carcinoma in situ

T1: Tumor limited to the maxillary sinus mucosa with no erosion or destruction of bone

T2: Tumor causing bone erosion or destruction including extension into the hard palate and/or middle nasal meatus, except extension to posterior wall of maxillary sinus and pterygoid plates

T3: Tumor invades any of the following: bone of the posterior wall of maxillary sinus, subcutaneous tissues, floor or medial wall of orbit, pterygoid fossa, or ethmoid sinuses

T4a: Tumor invades anterior orbital contents, skin of cheek, pterygoid plates, infratemporal fossa, cribriform plate, or sphenoid or frontal sinuses

T4b: Tumor invades any of the following: orbital apex, dura, brain, middle cranial fossa, cranial nerves other than maxillary division of trigeminal nerve (V2), nasopharynx, or clivus

Primary Tumor: Nasal Cavity and Ethmoid Sinus

TX: Cannot be assessed

T0: No evidence of primary tumor

Tis: Carcinoma in situ

T1: Tumor restricted to any one subsite, with or without bone invasion

T2: Tumor invading two subsites in a single region or extending to involve an adjacent region within the nasoethmoidal complex, with or without bone invasion

T3: Tumor extends to invade the medial wall or floor of the orbit, maxillary sinus, palate, or cribriform plate

T4a: Tumor invades any of the following: anterior orbital contents, skin of nose or cheek, minimal extension to anterior cranial fossa, pterygoid plates, or sphenoid or frontal sinuses

T4b: Tumor invades any of the following: orbital apex, dura, brain, middle cranial fossa, cranial nerves other than maxillary division of trigeminal nerve (V_2), nasopharynx, or clivus

Regional Lymph Nodes*

NX: Cannot be assessed

N0: No regional lymph node metastasis

N1: Metastasis in a single ipsilateral lymph node, ≤3 cm in greatest dimension

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N2: Metastasis in a single ipsilateral lymph node, >3 cm but not >6 cm in greatest dimension, or in multiple ipsilateral lymph nodes, none >6 cm in greatest dimension, or in bilateral or contralateral nodes, none >6 cm in greatest dimension

N2a: Metastasis in a single ipsilateral lymph node, >3 cm but not >6 cm in greatest dimension

N2b: Metastasis in multiple ipsilateral lymph nodes, none >6 cm in greatest dimension

N2c: Metastasis in bilateral or contralateral lymph nodes, none >6 cm in greatest dimension

N3: Metastasis in a lymph node >6 cm in greatest dimension

*Metastases at level VII are considered regional lymph node metastases. Midline nodes are considered ipsilateral nodes.

Distant Metastasis

M0: No distant metastasis

M1: Distant metastasis

Stage Groupings: For All Cancers Except Mucosal Malignant Melanoma

Stage 0

Tis N0 M0

Stage I

T1 N0 M0

Stage II

T2 N0 M0

Stage III

T1 N1 M0
T2 N1 M0
T3 N0,N1 M0

Stage IVA

T1,T2,T3 N2 M0
T4a N0,N1,N2 M0

Stage IVB

T4b Any N M0
Any T N3 M0

Stage IVC

Any T Any N M1

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5.2.4 Nasopharyngeal Cancer

◆ Key Features

- There is a high frequency of nasopharyngeal cancer (NPC) among patients of Chinese ethnicity and descent.
- It is associated with EBV exposure.
- The diagnosis must be excluded in patients with asymptomatic cervical lymphadenopathy and unilateral serous otitis media.

NPC is a distinct type of head and neck cancer that differs from other malignancies of the upper aerodigestive tract with respect to epidemiology, pathology, clinical presentation, and responses to treatment. NPC is an uncommon neoplasm in most parts of the world but is endemic in East Asia. Seventy percent of patients with newly diagnosed NPC present with locally advanced disease. Latent EBV infection seems to be crucial in the pathogenesis of NPC. Studies have established that NPC cells express two distinct EBV latent membrane proteins, LMP-1 and LMP-2. These proteins are attractive targets for adaptive immunotherapy.

◆ Anatomy

The nasopharynx is bounded superiorly by the basiocciput and basisphenoid, posteriorly by the C1 and C2 cervical bodies, anteriorly by the choanae, and inferiorly by the soft palate. The lateral walls are occupied primarily by the eustachian tube orifice. Immediately posterior to the eustachian tube orifice is Rosenmüller's fossa, where most nasopharyngeal carcinomas originate.

◆ Epidemiology

NPC occurs most often in China where it is the third most common malignancy among men, with an incidence rate of 15 to 50 per 100,000.

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NPC rates are also high in Vietnamese and Filipino men. There is an intermediate incidence in Inuit Eskimos and in the populations of the Mediterranean basin. Emigration from high- to low-incidence areas reduces the incidence of NPC in first-generation Chinese, but incidence still remains at seven times the rate seen in Caucasians. NPC occurs in younger patients, including children, but the peak incidence is seen in people aged 55 to 64 years. Males are affected three times more often than females.

◆ Clinical

Signs and Symptoms

Early signs and symptoms are subtle and variable, and are often initially ignored by both patient and physician. Cervical lymphadenopathy is the most common sign (50–90% of patients), followed by blood-stained nasal discharge or epistaxis, unilateral serous otitis media, and cranial neuropathies (most often CN VI, followed by CN V). Five to 7% of all patients have systemic metastases at presentation, most often to bone.

Symptoms include unilateral nasal obstruction, unilateral hearing loss and otalgia, diplopia, facial or neck pain, and paresthesia.

Differential Diagnosis

- Minor salivary gland tumors
- Juvenile nasopharyngeal angiofibroma
- Adenoid hypertrophy
- Tornwaldt cysts
- Fibromyxomatous polyps
- Choanal polyps, fibromas
- Papillomas
- Osseous/fibroosseous tumors
- Craniopharyngiomas
- Extracranial meningiomas
- Chordomas

◆ Evaluation

History

History should include questions about epistaxis, nasal obstruction and discharge, hearing loss or clogged ear, headache, diplopia, facial pain, and numbness.

Physical Exam

The physical exam should include a fiberoptic nasopharyngoscopy. The neck should be examined in a systematic fashion. Any lymph nodes should be assessed with regard to size, location, and mobility.

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Imaging

Imaging is required for staging and treatment planning for NPC. CT and MRI are recommended for the diagnostic process and evaluation of tumoral extent, bone erosion, and to delineate tumor extension into the parapharyngeal and retropharyngeal spaces, the oropharynx, the orbit, and the intracranial compartment.

A chest x-ray, liver ultrasound, and a bone scan are recommended for all patients with nodal disease. The role of PET in the staging of nasopharyngeal carcinoma has not been well established.

Labs

The quantitative analysis of cell-free EBV DNA in plasma of patients has been studied, and data suggest a possible value of this tool in screening and monitoring treatment. High IgA antibody levels to EBV capsid antigen and early antigen (EA) provide a valuable screening tool for early cases in high-incidence populations.

Other Tests

A dental examination is required before instituting radiotherapy to reduce the development of postradiotherapy complications. Patients should continue with meticulous dental care and fluoride prophylaxis.

Pathology

Classic nasopharyngeal carcinoma has been classified into three types by the World Health Organization (WHO):

Type 1: Keratinizing SCC
Type 2: Nonkeratinizing or poorly differentiated carcinoma
Type 3: Undifferentiated carcinoma (lymphepithelioma)

WHO types II and III exhibit between 82 to 100% positivity with respect to EBV antibody titers. Type 1 may have an association with cigarette and alcohol consumption and accounts for up to 30% of cases in nonendemic areas and $<5\%$ in endemic areas. Several genetic markers of the HLA system have been investigated in patients with NPC in China and other parts of Asia. HLA-A2 and HLA-BS-in-2 were associated with an increased incidence whereas HLA-A11 was associated with a decreased risk.

◆ Treatment Options

Medical

Radiotherapy is the cornerstone of the definitive treatment for NPC. This is because NPCs are particularly radiosensitive, and the tumor is in a relatively inaccessible location, making surgical excision difficult and highly morbid. External beam is most commonly delivered by opposed lateral fields to encompass the primary tumor and upper neck. Radiation doses of 70 to

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76 Gy in fractions of 1.8 to 2.0 Gy per day to the primary and anatomic structures at risk within the vicinity of the nasopharynx. Because there is a high incidence of subclinical neck disease, radiation doses between 50 and 60 Gy are used to electively treat the neck.

Recent data shows a clear role for concomitant chemoradiotherapy followed by adjuvant chemotherapy, which provides statistically significant improvement in overall survival and disease free survival.

EBV DNA titers seem to be an important index for prognostication. EBV DNA titers correlate with stage, treatment response, relapse, and survival.

Adoptive immunotherapy with EBV-specific CTL awaits further exploration.

Surgical

The role of surgery in NPC is largely confined to the treatment of residual or recurrent disease either in the nasopharynx or in the neck. Neck dissection for postradiation residual or recurrent nodal disease is the most common indication for surgery.

◆ Outcome and Follow-Up

Overall survival with the use of conventional radiotherapy alone in the treatment of NPC is in the range of 50 to 76%. Patients with stage I and II disease have a high rate of cure with radiotherapy alone. Seventy percent of patients with NPC present with locally advanced stage III or IV disease. For these patients, radiotherapy delivered in combination with chemotherapy has become the standard of care. The prognosis for those with distant metastatic spread remains poor.

A first complete evaluation should be performed 2 to 3 months after completion of treatment. The next evaluations should be scheduled for 6 months after this first posttherapeutic workup and on a yearly basis thereafter.

◆ ICD-9 Code

147 Malignant neoplasm of nasopharynx

◆ Staging of Nasopharyngeal Cancer

Primary Tumor: Nasopharynx

TX: Cannot be assessed

T0: No evidence of primary tumor

Tis: Carcinoma in situ

T1: Tumor confined to nasopharynx, or tumor extends to oropharynx and/or nasal cavity without parapharyngeal extension*

T2: Tumor with parapharyngeal extension*

T3: Tumor involves bony structures of skull base and/or paranasal sinuses

T4: Tumor with intracranial extension and/or involvement of CNs, hypopharynx, orbit, or with extension to the infratemporal fossa/masticator space

*Parapharyngeal extension denotes posterolateral infiltration of tumor.

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Regional Lymph Nodes (N): Nasopharynx†

NX: Cannot be assessed

N0: No regional lymph node metastasis

N1: Unilateral metastasis in lymph node(s), ≤6 cm in greatest dimension, above the supraclavicular fossa

N2: Bilateral metastasis in lymph node(s), ≤6 cm in greatest dimension, above the supraclavicular fossa

N3: Metastasis in a lymph node >6 cm and/or to supraclavicular fossa

N3a: >6 cm in dimension

N3b: Extension to the supraclavicular fossa

†Metastases at level VII are considered regional lymph node metastases; midline lymph nodes are considered ipsilateral nodes.

Distant Metastasis (M)

MX: Distant metastasis cannot be assessed

M0: No distant metastasis

M1: Distant metastasis

American Joint Committee on Cancer Stage Groupings for Nasopharynx Cancers Except Mucosal Malignant Melanoma

Stage 0

Tis N0 M0

Stage I

T1 N0 M0

Stage II

T2 N0 M0

Stage III

T1 N1 M0
T2 N1 M0
T3 N0 M0
T3 N1 M0

Stage IVA

T1 N2 M0
T2 N2 M0
T3 N2 M0
T4a N0 M0
T4a N1 M0
T4a N2 M0

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Stage IVB

Any T N3 M0
T4b any N M0

Stage IVC

Any T any N M1

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5.2.5 Oral Cavity Cancer

◆ Key Features

- A nonhealing or bleeding sore in the mouth or on the lip is the most common presentation.
- A persistent white or red patch on oral mucosa needs to be investigated.
- History of tobacco smoking or chewing occurs in most cases.
- Lip cancer may be caused by sun exposure.

The oral cavity extends from the skin–vermilion junctions of the anterior lips to the junction of the hard and soft palates superiorly and to the line of circumvallate papillae posteriorly. Oral cancer should be identified early and screening is useful. It is frequently preceded by an identifiable premalignant lesion. The progression from dysplasia may occur over a period of years.

◆ Epidemiology

Thirty thousand people are diagnosed yearly with oral cancer in the United States, and it will cause >8000 deaths. It constitutes <5% of US cancers, but in India, the incidence is far greater. For all stages combined,

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the 5-year relative survival rate is 59% and the 10-year survival rate is 44%. It typically occurs in those over the age of 45, and occurs in men twice as often as women. The number of new cases of this disease has been decreasing during the past 20 years. Tobacco smoking and alcohol are the primary risk factors. Smokeless tobacco in the Western world and paan (betel leaf with areca nut) in Asia are also risk factors for oral cancer. Other suspected risk factors include viral infection (HPV), a diet low in fruits and vegetables, vitamin A deficiency, genetic susceptibility, immunosuppressive drugs or immunocompromised conditions. Recently there has been a growing number of young patients with oral cancers, particularly involving the tongue.

◆ Clinical

Signs

Signs may include uncomfortable or poorly fitting dentures, loosening of the teeth, changes in articulation, a mass in the neck, weight loss, and persistent halitosis.

Symptoms

Symptoms depend on site and stage of the primary tumor and its effect on function of that area. They include a nonhealing white or red (leukoplakia, erythroplasia) patch or sore in the mouth (most common symptom), persistent pain in the mouth, and a thickening in the cheek or floor of mouth. More advanced disease may cause a sore throat, difficulty chewing, dysphagia, trismus or tongue tethering, numbness of the tongue or mouth, and pain around the teeth or jaw.

Differential Diagnosis

- Oral and pharyngeal infections such as pharyngitis or stomatitis
- Chancre
- Benign oral or odontogenic lesions
- Denture sores
- Aphthous ulcers or herpetic sores
- Lesion due to cheek biting
- Oral manifestations of systemic diseases
- Necrotizing sialometaplasia

◆ Evaluation

History

Evaluation begins with a detailed history inquiring about tobacco and alcohol usage, oral pain, referred otalgia, dysphagia, articulation changes, and weight loss.

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Physical Exam

A physical exam should include a complete head and neck exam. Specific attention should be directed at the site of the lesion. The lesion size should be noted as should its infiltration and spread to adjacent oral cavity or oropharyngeal subsites such as floor of mouth, alveolus, and tongue base. A bimanual examination of the lesion, the surrounding floor of mouth, and the submandibular triangle should be performed.

Careful palpation of the neck may reveal adenopathy. The main routes of lymph node drainage from the oral cavity are into the first echelon nodes (i.e., buccinator, jugulodigastric, submandibular, and submental). Tumor sites close to the midline commonly drain bilaterally.

Imaging

Contrast-enhanced CT of the head and neck is a necessary component of initial evaluation. Tumor size and spread may be evaluated as well as discrete nodal disease, bony destruction, and vascular involvement. MRI may be helpful in the evaluation of oral cancer because it provides a higher contrast between normal tissue and tumor on T2-weighted images, has no beam artifact from dental material, and provides multiplanar imaging.

The combination of PET and CT is a useful diagnostic and staging modality in the evaluation of the patient with head and neck cancer.

A chest CT may be used to rule out pulmonary metastasis. Periapical dental radiographs provide fine detail and may show minimal invasion. Panoramic dental radiographs may show gross bony destruction.

Labs

Routine preoperative laboratory studies are employed.

Other Tests

Patients with suspected oral cancer must undergo a biopsy for pathologic diagnosis. The first test for the evaluation of a neck mass presenting with an oral cavity lesion is FNAB.

The routine use of panendoscopy, which includes bronchoscopy, esophagoscopy, and laryngoscopy, is recommended. It allows for the complete evaluation of the upper aerodigestive tract and helps rule out the presence of a synchronous tumor. The mucous membranes of the upper aerodigestive tract are carefully evaluated, and biopsy samples of any abnormal-looking areas. This is particularly important in the patient who smokes.

Toluidine blue staining and photodynamic agents such as Foscan (Sigma-Aldrich, St. Louis, MO) and 5-aminolevulinic acid (ALA) may be used to enhance detection of oral lesions. Oral brush biopsy (OralCDx; Oral CDx Laboratories, Inc., Suffern, NY) can be used in the screening of oral precancer and cancer.

A dental evaluation should be performed, with attention to dental hygiene, dentition status, and integrity of the mandible.

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Pathology

Ninety percent of oral cancers are SCCs, and they may be preceded by various precancerous lesions. Non-SCC oral cancers may also represent minor salivary gland tumors. Other rarer cancers may be of odontogenic apparatus origin, lymphomas, soft tissue sarcomas, and melanomas.

There is no significant correlation between degree of squamous differentiation (i.e., grading of dysplasia) and the biologic behavior of oral cancer (**Table 5.6**). Vascular and perineural invasion thickness and depth of invasion are all negative prognostic factors.

Verrucous carcinoma (VC) refers to a clinicopathologic concept implying a locally aggressive, clinically exophytic, low-grade, well-differentiated SCC with minimal metastatic potential. It is also known as Ackerman tumor. An HPV infection is thought to facilitate or cause verrucous carcinoma. Associations with verrucous carcinoma have been found in patients who chewed tobacco and betel nut.

Fully developed lesions are white cauliflower-like papillomas with a pebbly surface that may extend and coalesce over large areas of the oral mucosa. Overall, patients with verrucous carcinoma have a favorable prognosis; the course of verrucous carcinoma lesions is characterized by slow, continuous local growth.

Proliferative verrucous leukoplakia (PVL) is a particularly aggressive form of oral leukoplakia that commences with a hyperkeratosis, spreads to become multifocal and verruciform in appearance, and later becomes malignant. It is significant because it has a high recurrence rate and the potential to develop into verrucous carcinoma or SCC in 60 to 70% of the affected patients. PVL is more commonly found in elderly females and is associated with tobacco use or alcohol abuse in 30 to 50% of patients. The etiology of PVL is unknown. An association with HPV infection, particularly strains 16 and 18, has been implicated in some cases. The most common locations are the gingiva or alveolar ridge tongue, and buccal mucosa sites. The gingiva is the most likely site for the malignant transformation of PVL. PVL often begins as a focal lesion spreading laterally over time and can be multifocal. Early in its course it is a flat hyperkeratotic lesion that becomes progressively verrucous and histologically often exhibits varying degrees of epithelial dysplasia.

Table 5.6 Tumor Grading Is Recommended Using the Broder Classification (Tumor Grade [G])

G1: Well differentiated
G2: Moderately well differentiated
G3: Poorly differentiated
G4: Undifferentiated

Note: No statistically significant correlation between degree of differentiation and the biologic behavior of the cancer exists; however, vascular invasion is a negative prognostic factor.

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◆ Treatment Options

Surgical resection and radiotherapy are the current treatments of choice.

Lip Cancer

Most lip SCCs present on the lower lip (88–95%), 2 to 7% present on the upper lip, and 1% on the oral commissure. Basal cell carcinoma is more common on the upper lip.

Treatment of Lip Cancer

- For T1 and T2 lesions, radiotherapy and surgery produce similar cure rates; the method of treatment is determined by functional and cosmetic factors.
- Advanced lesions of the lip generally require a combination of surgery and radiotherapy.
- Patients with upper lip and oral commissure SCC have a worse overall prognosis.
- The 5-year survival for stage I and II lesions is 90%.

Oral Tongue Cancer

Seventy-five percent of tongue cancers occur on the posterior lateral aspect, 20% on the anterior lateral aspect, and 3 to 5% on the lingual dorsum. At the time of diagnosis, 75% oral tongue cancers are T2 or smaller. Forty percent of patients with oral tongue cancer demonstrate clinical evidence of neck metastasis at presentation.

Treatment of Oral Tongue Lesions

- Early tongue cancer: Wide local excision is often used for T1 lesions that can be resected transorally.
- For larger T1 and T2 lesions, either surgery or radiotherapy are acceptable treatments.
- Deeply infiltrative lesions (>4 mm depth) can be treated with surgery with postoperative radiotherapy and a selective neck dissection.
- Selected patients with T4 tongue cancer can be treated with combined surgery (i.e., total glossectomy, sometimes requiring laryngectomy due to the high risk of postoperative aspiration) and postoperative radiotherapy.
- For T1 and T2 lesions, 20 to 30% of patients harbor metastatic disease in cervical lymph nodes. Thus, therapy (surgery or radiotherapy) aimed at the neck should be considered as part of definitive treatment.
- The 5-year survival is 75% for stage I and II oral tongue cancers and <40% for stage III and IV oral tongue cancers.

Buccal Mucosa Cancer

Carcinomas of the buccal mucosa represent 5 to 10% of oral cancers. The most common area is in the region of the third mandibular molar. Lesions

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<1 cm in diameter can be treated by surgery alone if the oral commissure is not involved. If involved, radiotherapy should be considered. Premalignant conditions include submucosal fibrosis and lichen planus. The latter has a reported transformation rate of 0.5 to 3%, whereas the former has a malignant transformation rate of 0.5%.

Treatment of Buccal Mucosa Cancers

- Lesions smaller than 1 cm in diameter can be managed by surgery alone if the commissure is not involved. If the commissure is involved, radiotherapy (including brachytherapy) should be considered.
- Advanced lesions of the buccal mucosa can be treated with surgical resection alone, radiotherapy alone, or surgical resection plus postoperative radiation.
- The 5-year survival for buccal mucosa cancer is 75% for stage I, 65% for stage II, 30 to 65% for stage III, and 20 to 50% for stage IV buccal cancer.

Floor of Mouth Cancer

Cancers of the floor of mouth represent 28 to 35% of oral cancers. Thirty-five percent of patients with floor of mouth cancer present with T3 or T4 disease. The most common presentation of cancer of the floor of the mouth is a painless inflamed superficial ulcer with poorly defined margins. Preexistent or coincident leukoplakia can be observed in adjacent tissues in ~20% of cases.

Treatment of Floor of Mouth Cancer

Note that a cancer involving the gingiva adjacent to recent dental extraction is at high risk for bony extension via the tooth socket.

- For T1 lesions either transoral surgery or radiotherapy is an acceptable treatment.
- For small T2 lesions (≤3 cm), surgery is often used if the lesion is attached to the periosteum, whereas radiotherapy is often used if the lesion encroaches on the tongue.
- For large T2 lesions (>3 cm), surgery and radiotherapy are alternative methods of treatment, the choice of which depends primarily on the expected extent of disability from surgery.
- External-beam radiotherapy with or without interstitial radiotherapy should be considered postoperatively for larger lesions.
- For more advanced lesions, surgery should incorporate rim resection plus neck dissection or partial mandibulectomy with neck dissection as appropriate.
- The 5-year survival for floor of mouth cancer is 90% for stage I, 80% for stage II, 65% for stage III, and 30% for stage IV.

Retromolar Trigone Cancer

Retromolar trigone cancers account for ~10% of all oral cancers. These cancers typically present with advanced disease, and 50% of patients have regional metastasis at the time of diagnosis.

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Treatment of Retromolar Trigone Cancer

For small lesions without detectable bone invasion, limited resection of the mandible may be performed. Radiotherapy may be used initially, with surgery reserved for radiation failure. Selective neck treatment should be performed—for advanced stages, multimodality therapy with surgery and postoperative radiation is most often used.

Hard Palate Cancer

Cancer of the hard palate accounts for 5% of all oral cavity malignancies. Ten to 25% of patients with head and neck SCC of the hard palate present with regional metastasis. (Only 53% of hard palate cancers are SCC; minor salivary gland malignancies make up the rest.)

Treatment of Cancer of the Hard Palate

For both early and advanced disease, surgery (inferior maxillectomy with surgical obturator) is used for primary therapy. Radiotherapy has a role depending on factors such as close or positive surgical margins, evidence of perineural involvement or the presence of lymph node metastases. The prosthodontist is important in the care of these patients for oral rehabilitation. The 5-year survival for hard palate cancer ranges from 40 to 60%.

Advanced Oral Cavity Cancer

Clinical trials for advanced oral tumors evaluating the use of chemotherapy preoperatively, before radiotherapy, as adjuvant therapy after surgery, or as part of combined modality therapy are appropriate.

◆ Outcome and Follow-Up

Patients should be seen at 2-month intervals for 2 years after treatment. Thereafter they should be seen at 3-month intervals for the next year and then every 6 months for another year. Patients with regional neck disease prior to treatment should have undergo CT scan or integrated FDG-PET/CT 12 weeks after the completion of radiotherapy to assess for residual disease that may necessitate postradiotherapy neck dissection. The risk of a second primary carcinoma is highest in those who continue to smoke, and patients should be strongly urged to quit.

◆ ICD-9 Codes

140–149	Malignant neoplasm of lip, oral cavity, and pharynx
140	Malignant neoplasm of lip
141	Malignant neoplasm of tongue
143	Malignant neoplasm of gum, includes alveolar (ridge) mucosa gingiva (alveolar) (marginal) interdental papillae

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144 Malignant neoplasm of floor of mouth
145 Malignant neoplasm of other and unspecified parts of mouth
149 Malignant neoplasm of other and ill-defined sites within the lip, oral cavity, and pharynx

◆ Staging of Oral Cavity Cancer

Primary Tumor

TX: Primary tumor cannot be assessed
T0: No evidence of primary tumor
Tis: Carcinoma in situ
T1: Tumor ≤2 cm in greatest dimension
T2: Tumor >2 cm but not >4 cm in greatest dimension
T3: Tumor >4 cm in greatest dimension
T4a: Moderately advanced local disease.

Lip: Tumor invades through the cortical bone, the inferior alveolar nerve, the floor of the mouth, or the skin of the face, i.e., the chin or nose.

Oral cavity: Tumor invades the adjacent structures (e.g., through the cortical bone [mandible, maxilla], into the deep [extrinsic] muscle of the tongue [genioglossus, hyoglossus, palatoglossus, and styloglossus], the maxillary sinus, or the skin of the face).

T4b: Very advanced local disease. Tumor invades the masticator space, pterygoid plates, or skull base, and/or encases the internal carotid artery.

Regional Lymph Nodes*

NX: Regional lymph nodes cannot be assessed
N0: No regional lymph node metastasis
N2a: Metastasis in a single ipsilateral lymph node, >3 cm but not >6 cm in greatest dimension
N2b: Metastasis in multiple ipsilateral lymph nodes, none >6 cm in greatest dimension
N2c: Metastasis in bilateral or contralateral lymph nodes, none >6 cm in greatest dimension
N3: Metastasis in a lymph node >6 cm in greatest dimension

*Superior mediastinal lymph nodes are considered regional lymph nodes (level VII). Midline nodes are considered ipsilateral nodes.

Distant Metastasis

M0: No distant metastasis
M1: Distant metastasis

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American Joint Committee on Cancer Stage Groupings for Oral Cavity Cancers

Stage 0

Tis N0 M0

Stage I

T1 N0 M0

Stage II

T2 N0 M0

Stage III

T1 N1 M0
T2 N1 M0
T3 N0 M0
T3 N1 M0

Stage IVA

T1 N2 M0
T2 N2 M0
T3 N2 M0
T4a N0 M0
T4a N1 M0
T4a N2 M0

Stage IVB

Any T N3 M0
T4b any N M0

Stage IVC

Any T any N M1

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5.2.6 Oropharyngeal Cancer

◆ Key Features

- Oropharyngeal cancer includes cancer of the tonsil, tongue base, soft palate, and oropharyngeal wall.
- Oropharyngeal cancer is primarily linked to tobacco and alcohol use.
- HPV is a risk factor for cancer of the tonsil.
- Neck metastasis may be cystic.

The oropharynx is located between the soft palate superiorly and the hyoid bone inferiorly; it communicates with the oral cavity anteriorly, the nasopharynx superiorly and the supraglottic larynx and hypopharynx inferiorly. Oropharynx cancers are typically detected at a more advanced stage than oral cancer. The oropharynx is an important component in swallowing; therefore, treating these tumors is challenging and often requires a multidisciplinary approach and posttreatment rehabilitation.

◆ Epidemiology

In the United States, an estimated 8300 new cases of pharyngeal cancer (including cancers of the oropharynx and hypopharynx) are diagnosed yearly, with an estimated mortality of 2000. It affects men three times more than women. Seventy-five percent of oropharynx cancers occur in the tonsil. Tobacco (including smokeless tobacco) and alcohol abuse represent the most significant risk factors for the development of oropharynx cancer. Viral infection with HPV is a risk factor for SCC of the tonsil and may be a positive prognostic factor.

Other risk factors include Yerba maté, a stimulant beverage commonly consumed in South America, and chewing of paan (betel leaf with areca nut), which is common in parts of Asia.

◆ Clinical

Signs

Signs include changes in articulation, muffled speech, a mass in the neck, unintentional weight loss, hemoptysis, and persistent halitosis.

Symptoms

Symptoms may include pain, dysphagia, globus sensation, referred otalgia, trismus, and fixation of the tongue. Cancers of the base of the tongue and tonsil are typically insidious.

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Differential Diagnosis

- Oropharyngeal infections such as pharyngitis or stomatitis
- Chancre
- Benign oropharyngeal or odontogenic lesions
- Aphthous ulcers or herpetic sores
- Oral manifestations of systemic diseases

◆ Evaluation

History

Evaluation begins with a detailed history inquiring about tobacco and alcohol usage, sexual history, oral pain, odynophagia, referred otalgia, dysphagia, hemoptysis, articulation or speech changes, and unintentional weight loss.

Physical Exam

The physical exam should include a complete head and neck exam, with specific attention directed at the site of the lesion. The lesion size should be noted as should its apparent infiltration and spread to adjacent pharyngeal or oral cavity subsites such as oral tongue, hypopharynx, nasopharynx, and vallecula. Palpation of the lesion should be performed if possible in an awake patient. In advanced cases, discerning the primary origin of the lesion, such as the tongue base or tonsil, is not always possible. Fiberoptic laryngoscopy should be performed. Inspection and palpation of the neck often reveals adenopathy.

Imaging

Clinical staging may understage oropharynx tumors, especially the tongue base extension. Both contrast-enhanced CT and MRI are able to assess oropharynx tumor extent as well as regional spread. Neck metastasis from oropharynx cancer may be cystic in morphology; this finding by itself should raise suspicion of a cancer in the tonsil or tongue base.

At minimum, a chest CT should be used to rule out pulmonary metastasis or second primary. Alternatively, an integrated FDG-PET/CT may be used in a combined staging and metastatic workup.

Labs

Standard preoperative laboratories, as indicated, should be obtained.

Other Tests

Patients with suspected cancer of oropharynx must undergo a biopsy and a sample of the lesion taken for pathologic examination. This may be done in an office setting in cases of tonsil cancer and soft palate cancer, but is not usually possible in cases of tongue base lesions. If neck metastases are evident, they should be sampled by FNAB.

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Because of the propensity for second primary tumors, panendoscopy (triple endoscopy)—laryngoscopy, esophagoscopy, and bronchoscopy together—has been advocated. This is particularly important in patients who smoke or in patients with large, bulky tumors to establish the true extent of these lesions.

Pathology

Histologically, 90% of oropharynx cancers are SCCs. Basaloid SCC is an uncommon but aggressive SCC variant. Other cancers of the oropharynx include minor salivary gland carcinomas, lymphomas, and "lymphoepithelial-like" carcinomas.

◆ Treatment Options

For stage I oropharynx cancer, surgery or radiotherapy may be used depending on the expected functional deficit. Radiation clinical trials evaluating hyperfractionation schedules should be considered.

For stage II oropharynx cancer, surgery or radiation therapies are equally successful in controlling disease. Radiotherapy may be the preferred modality where the functional deficit is expected to be great.

The management of stage III oropharynx cancer is complex and requires a multidisciplinary approach to establish the optimal treatment. A combination of surgery with postoperative radiotherapy and/or chemotherapy is most often used. An alternative is chemoradiation therapy alone based on the patient's initial nodal involvement.

In stage III tonsil cancer, hyperfractionated radiotherapy yields a higher control rate than standard fractionated radiotherapy.

In advanced unresectable oropharyngeal cancer, radiotherapy or chemoradiation is used.

Treatments currently under investigation include chemotherapy with radiation clinical trials as well as with radiosensitizers, radiation clinical trials evaluating hyperfractionation schedules and/or brachytherapy, particle-beam radiotherapy, and hyperthermia combined with radiotherapy.

Recently, transoral robotic-assisted resection (TORS) of select oropharyngeal tumors has gained momentum.

◆ Outcome and Follow-Up

The overall 5-year disease-specific survival for patients with all stages of disease is ~50%. Patients with cancer of the oropharynx should have a careful head and neck examination to examine for recurrence monthly for the first posttreatment year, every 2 months for the second year, every 3 months for the third year, and every 6 months to a year thereafter.

◆ ICD-9 Codes

141	Malignant neoplasia of base of tongue
145.3	Malignant neoplasia of soft palate
146	Malignant neoplasia of oropharynx/tonsil

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146.1 Malignant neoplasia of tonsil fossa
146.2 Malignant neoplasia of tonsillar pillars
146.3 Malignant neoplasia of vallecula epiglottica
146.6 Malignant neoplasia of lateral wall of oropharynx
146.7 Malignant neoplasia of posterior wall of oropharynx

◆ Staging of Oropharyngeal Cancer

Primary Tumor

TX: Primary tumor cannot be assessed
T0: No evidence of primary tumor
Tis: Carcinoma in situ
T1: Tumor ≤2 cm in greatest dimension
T2: Tumor >2 cm but not >4 cm in greatest dimension
T3: Tumor >4 cm in greatest dimension or extension to the lingual surface of epiglottis
T4a: Moderately advanced local disease. Tumor invades the larynx, the deep/extrinsic muscle of tongue, the medial pterygoid muscles, hard palate, or the mandible.*
T4b: Very advanced local disease. Tumor invades the lateral pterygoid muscle, pterygoid plates, the lateral nasopharynx or skull base, or encases the carotid artery.

*Note: The mucosal extension to the lingual surface of the epiglottis from primary tumors of the base of the tongue and vallecula does not constitute invasion of the larynx.

Regional Lymph Nodes

NX: Regional lymph nodes cannot be assessed
N0: No regional lymph node metastasis
N1: Metastasis in a single ipsilateral lymph node, ≤3 cm in greatest dimension
N2a: Metastasis in a single ipsilateral lymph node, >3 cm but not >6 cm in greatest dimension
N2b: Metastasis in multiple ipsilateral lymph nodes, none >6 cm in greatest dimension
N2c: Metastasis in bilateral or contralateral lymph nodes, none >6 cm in greatest dimension
N3: Metastasis in a lymph node >6 cm in greatest dimension

Distant Metastasis

MX: Distant metastasis cannot be assessed
M0: No distant metastasis
M1: Distant metastasis

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American Joint Committee on Cancer Stage Groupings of Oropharyngeal Cancer

Stage 0

Tis N0 M0

Stage I

T1 N0 M0

Stage II

T2 N0 M0

Stage III

T1 N1 M0
T2 N1 M0
T3 N0 M0
T3 N1 M0

Stage IVA

T1 N2 M0
T2 N2 M0
T3 N2 M0
T4a N0 M0
T4a N1 M0
T4a N2 M0

Stage IVB

Any T N3 M0
T4b any N M0

Stage IVC

Any T any N M1

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5.2.7 Hypopharyngeal Cancer

◆ Key Features

- Hypopharyngeal cancer occurs in the pyriform sinuses, the posterior hypopharyngeal, or postcricoid region.
- Patients present with dysphagia, globus sensation, hoarseness, and referred otalgia.
- It usually presents in advanced stages of disease.
- It has a high propensity for regional metastasis at presentation.

The hypopharynx is the part of the pharynx, which lies behind the larynx and connects the oropharynx and the esophagus. It is subdivided into three subsites: the paired pyriform sinuses, the posterior hypopharyngeal wall, and the postcricoid region. Sixty-five to 85% of cancers of the hypopharynx involve the pyriform sinuses, 10 to 20% involve the posterior pharyngeal wall, and 5 to 15% involve the postcricoid area.

◆ Epidemiology

Cancer of the hypopharynx is uncommon, with ~2500 new cases diagnosed in the United States each year. Cancer of the hypopharynx typically presents in advanced stages, and the incidence of regional metastases and distant metastases are also among the highest of all head and neck cancers. Cancer of the hypopharynx is typically seen in men over 55 years old with a history of tobacco product use and/or alcohol ingestion.

An exception is an increased incidence of postcricoid cancer in women aged 30 to 50 years with Plummer-Vinson syndrome. Asbestos may pose an independent risk for the development of cancer of the hypopharynx.

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◆ Clinical

Signs and Symptoms

Signs include muffled speech, hoarseness, a mass in the neck, hemoptysis, unintentional weight loss, airway obstruction, and persistent halitosis. The incidence of regional metastases is 50 to 70% at presentation. Symptoms include dysphagia, chronic sore throat, hoarseness, globus sensation, and referred otalgia.

Differential Diagnosis

Differential diagnosis may include pharyngeal infections such as pharyngitis or candidal infection, benign hypopharynx or upper esophageal lesions, and pharyngeal manifestations of systemic diseases.

◆ Evaluation

History

A detailed history should include questions about smoking and alcohol usage, prolonged hoarseness, dysphagia, odynophagia, hemoptysis, otalgia, and unintentional weight loss.

Physical Exam

The physical exam should include a complete head and neck exam. Specific attention should be directed at the site of the lesion. A fiberoptic laryngoscopy should be performed. The lesion size should be noted as should its infiltration and spread to adjacent laryngeal and hypopharynx subsites. During this examination, the patient should be taken through maneuvers such as protrusion of the tongue, puffing out the cheeks, lightly coughing, and speaking to better visualize and access the pharynx and larynx. It is important that laryngeal motility be assessed, as this is critical in tumor staging. Inspection and palpation of the neck often reveals adenopathy. On neck examination, loss of the grating sensation (laryngeal crepitus) of the laryngeal cartilages over the prevertebral tissues may indicate deep pharyngeal wall involvement. Impeding airway obstruction should be recognized and addressed.

Imaging

Chest imaging with a chest radiograph or CT scan should be performed to rule out pulmonary metastasis or a pulmonary second primary.

Superficial mucosal lesions in the pyriform sinus may be seen on barium swallow studies, although this is not the imaging modality of choice. Negative findings on swallow study despite progressive or continuous symptoms should not preclude an endoscopic examination. CT and MRI are used to image the primary tumor and regional lymph nodes prior to definitive treatment. They provide information about the location and extent of tumor involvement and demonstrate the interface of tumor with cartilage, muscles, soft tissues, and blood vessels. Alternatively, an integrated FDG-PET/CT may be used in a combined staging and metastatic workup.

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Labs

Blood count, electrolyte, and liver function tests should be performed to assess nutritional status.

Other Tests

Patients with suspected cancer of the hypopharynx must undergo a biopsy and a sample of the lesion taken for pathologic examination. This biopsy may be coupled with a triple endoscopy to evaluate the patient for the presence of synchronous second primary tumors. The direct laryngoscopic exam under anesthesia is a critical part of staging and treatment planning.

Pathology

Ninety-five percent of cancers of the hypopharynx are SCCs.

◆ Treatment Options

The treatment of cancer of the hypopharynx is controversial, in part because of its low incidence and the inherent difficulty in conducting adequately powered, prospective, randomized clinical studies. In general, both surgery and radiotherapy are the mainstays of most curative efforts aimed at this cancer.

Stage I Tumors

Laryngopharyngectomy and neck dissection has been the most frequently used therapy for surgical hypopharyngeal cancers. In very select cases, a partial laryngopharyngectomy may be successfully used. Radiotherapy may be used as a primary treatment modality and should include the neck.

Stage II Tumors

Laryngopharyngectomy and neck dissection has been the most frequently used therapy. Neoadjuvant chemotherapy has been used to reduce tumors and render them more definitively treatable with either surgery or radiation.

Stage III and IV Tumors

A combination of surgery and radiation most often is given postoperatively. Patients with stage III and IV cancer of the hypopharynx should be considered for treatment with combined postoperative adjuvant radiotherapy and chemotherapy.

◆ Outcome and Follow-Up

The prognosis of cancer of the hypopharynx is poor, with most series reporting a $<25\%$ 5-year survival rate. Presentation at a late stage, multisite involvement within the hypopharynx, unrestricted soft tissue tumor growth, an extensive regional lymphatic network allowing development of metastases, and restricted surgical options for complete resection contribute to an overall poor prognosis. Hypopharynx cancer patients should have

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a careful head and neck examination to look for recurrence monthly for the first posttreatment year, every 2 months for the second year. Patients with regional neck disease prior to treatment should have a CT scan or an integrated FDG-PET/CT scan 12 weeks after the completion of treatment. The risk of a second primary carcinoma is highest in those who continue to smoke and patients should be strongly urged to quit.

◆ ICD-9 Codes

148 Malignant neoplasm of hypopharynx
148.7 Malignant neoplasm of pyriform sinus
148.3 Malignant neoplasm of posterior hypopharyngeal well
148.0 Malignant neoplasm of postericoid region of hypopharynx

◆ Staging of Hypopharyngeal Cancer

Primary Tumor

TX: Primary tumor cannot be assessed

T0: No evidence of primary tumor

Tis: Carcinoma in situ

T1: Tumor limited to one subsite of the hypopharynx and ≤2 cm in greatest dimension

T2: Tumor invades more than one subsite of the hypopharynx or an adjacent site, or measures >2 cm but not >4 cm in greatest dimension without fixation of the hemilarynx

T3: Tumor measures >4 cm in greatest dimension or with fixation of the hemilarynx or extension to the esophagus

T4a: Moderately advanced local disease. Tumor invades the thyroid/cricoid cartilage, hyoid bone, the thyroid gland, the esophagus, or the central compartment soft tissue.*

T4b: Very advanced local disease. Tumor invades the prevertebral fascia, encases the carotid artery, or involves the mediastinal structures.

*Central compartment soft tissue includes the prelaryngeal strap muscles and subcutaneous fat.

Regional Lymph Nodes

NX: Regional lymph nodes cannot be assessed

N0: No regional lymph node metastasis

N1: Metastasis in a single ipsilateral lymph node, ≤3 cm in greatest dimension

N2a: Metastasis in a single ipsilateral lymph node, >3 cm but not >6 cm in greatest dimension

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N2b: Metastasis in multiple ipsilateral lymph nodes, none >6 cm in greatest dimension

N2c: Metastasis in bilateral or contralateral lymph nodes, none >6 cm in greatest dimension

N3: Metastasis in a lymph node >6 cm in greatest dimension

*Metastases at level VII are considered regional lymph node metastases; midline lymph nodes are considered ipsilateral nodes.

Distant Metastasis

MX: Distant metastasis cannot be assessed

M0: No distant metastasis

M1: Distant metastasis

American Joint Committee on Cancer Stage Groupings of Hypopharyngeal Cancer

Stage 0

Tis N0 M0

Stage I

T1 N0 M0

Stage II

T2 N0 M0

Stage III

T1 N1 M0
T2 N1 M0
T3 N0 M0
T3 N1 M0

Stage IVA

T1 N2 M0
T2 N2 M0
T3 N2 M0
T4a N0 M0
T4a N1 M0
T4a N2 M0

Stage IVB

Any T N3 M0
T4b any N M0

Stage IVC

Any T any N M1

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5.2.8 Laryngeal Cancer

◆ Key Features

- Laryngeal cancer is the second most common head and neck cancer.
- The most common site is the glottis.
- It may present early with hoarseness.
- Treatment options are based on site and extent of disease.

Cancer arising from the squamous epithelium of the larynx is a common head and neck cancer, with well-known risk factors. Proper staging is complex, but crucial for treatment formulation. The larynx plays a unique role in respiration, speech, and swallowing. The complex anatomy of the larynx explains the unique patterns of spread of laryngeal cancer:

- The preepiglottic fat is located in the anterior and lateral aspects of the larynx and is often invaded by advanced cancers.
- The recurrent laryngeal nerve innervates the intrinsic laryngeal muscles. Invasion of this nerve causes hoarseness clinically and fixation of the vocal folds.
- The anterior commissure invasion may be a conduit for tumor spread.

The larynx is divided into three anatomic regions: the supraglottic larynx, the glottis, and the subglottic region. The supraglottic larynx includes the

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epiglottis, the preepiglottic space, the laryngeal aspects of the aryepiglottic folds, the false vocal folds, the arytenoids, and the ventricles. The inferior boundary is a horizontal plane drawn through the apex of the ventricle. This corresponds to the area of transition from squamous to respiratory epithelium. The glottis consists of the true vocal folds extending to roughly 1 cm below the true folds, the paraglottic space, and the anterior and posterior commissures extending inferiorly ~1 cm. The subglottic larynx has its superior border at the inferior border of the glottis, ~1 cm below the true vocal folds and extending inferiorly to the trachea.

Laryngeal cancer can also be classified by anatomic location—signs, symptoms, and tumor behavior vary depending on the site and the extent of disease.

◆ Epidemiology

In 2008, 12,250 men and women were diagnosed with cancer of the larynx in the United States; of those, 3670 patients died. Risk factors include smoking and drinking alcohol, which act synergistically; laryngeal papillomatosis; radiation exposure; immunosuppression; and occupational exposure to metals, plastics, and asbestos. Laryngeal carcinoma is more common in blacks than in whites, with a ratio of 3.5:1.

◆ Clinical

Signs and Symptoms

The common symptoms of *supraglottic cancers* include mild odynophagia, mild dysphagia, and mass sensation. Later symptoms include severe dysphagia and aspiration and referred ear pain. The mechanism of the referred ear pain is through the activation of the internal branch of the superior laryngeal branch of CN X with referral to Arnold's nerve: 25 to 50% have nodal metastasis to the cervical lymph nodes. Supraglottic tumors often spread to nodes on both sides of the neck.

Glottic cancers account for over half of all laryngeal cancers and present typically with hoarseness. Voice change often happens early and can help diagnose early stage cancer, improving prognosis. Additionally, the vocal folds' sparse lymphatics accounts for a relatively low incidence of nodal metastasis. Advancing glottic cancer can spread posteriorly to the arytenoid complex causing vocal fold fixation, or anteriorly to the commissure, where it can invade the thyroid cartilage.

Subglottic cancer is uncommon. It may produce biphasic stridor or airway obstruction. Patients may be asymptomatic until advanced stages of disease and thus the prognosis is worse. Lymphatics drain through the cricothyroid and cricotracheal membranes to the pretracheal, paratracheal, and inferior jugular nodes, and occasionally to mediastinal nodes.

Differential Diagnosis

- Hyperkeratosis
- Papillomas

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- Polyps
- Fibromas
- Granulomas
- Laryngoceles
- Laryngeal manifestations of systemic, infectious, or autoimmune disease

◆ Evaluation

The most important adverse prognostic factors for laryngeal cancers include increasing T stage and N stage (see Staging of Laryngeal Cancer section, below). Other prognostic factors may include sex, age, performance status, and a variety of pathologic features of the tumor, including grade and depth of invasion.

History

History should focus on timing and duration of symptoms and assessment of risk factors. A full understanding of the patient's general medical health, nutritional and cardiopulmonary status, social support, and compliance are crucial for planning of therapy.

Physical Examination

A thorough physical examination of the head and neck should be performed, including inspection of the oral mucosa, laryngoscopy, bimanual palpation of floor of mouth and base of tongue, and a careful assessment of the cervical lymph nodes and thyroid cartilage contour.

Imaging

Contrast-enhanced CT scans obtained with appropriate section thickness aid in the evaluation of laryngeal cancer and neck masses. PET-CT can assist in identifying metastasis and set a baseline of future follow-up for recurrence. At the very least, a chest CT should be performed to rule out a secondary pulmonary malignancy or metastatic disease.

Other Tests

Suspension direct laryngoscopy provides an opportunity for examination under general anesthesia, palpation, and biopsy. Extent of the tumor and the overall condition of the airway mucosa can be evaluated. Panendoscopy (i.e., laryngoscopy, esophagoscopy, and bronchoscopy together) can be performed to rule out synchronous malignancies.

FNAB of a neck mass may yield a positive result when the certainty of an enlarged lymph node is in question.

Pathology

Ninety to ninety-five percent of laryngeal cancers are SCCs. Other less common types of laryngeal malignancies include adenoid cystic carcinoma, with

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a characteristic indolent course of growth and perineural invasion. Rarely, cancer can be of neuroendocrine, stromal, or mesenchymal origin.

◆ Treatment Options

Stage I Laryngeal Cancer

Supraglottis

Standard treatment options include:

- External-beam radiotherapy alone
- Supraglottic laryngectomy

Glottis

Standard treatment options include:

- Radiotherapy
- Cordectomy for very carefully selected patients with limited and superficial T1 lesions
- Partial or hemilaryngectomy or total laryngectomy, depending on anatomic considerations
- Laser excision

Subglottis

- Radiotherapy alone

Stage II Laryngeal Cancer

Supraglottis

Standard treatment options include:

- External-beam radiotherapy, supraglottic laryngectomy or total laryngectomy, depending on location of the lesion, clinical status of the patient, and expertise of the treatment team
- Postoperative radiotherapy is indicated for positive or close surgical margins.

Glottis

Standard treatment options include:

- Radiotherapy
- Partial or hemilaryngectomy or total laryngectomy, depending on anatomic considerations. Under certain circumstances, laser microsurgery may be appropriate.

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Subglottis

- Lesions can be treated successfully by radiotherapy alone with preservation of normal voice.

Stage III Laryngeal Cancer

Supraglottis

Standard treatment options include:

- Surgery with or without postoperative radiotherapy
- Definitive radiotherapy with surgery for salvage of radiation failures
- Chemotherapy administered concomitantly with radiotherapy can be considered for patients who would require total laryngectomy for control of disease.

Glottis

Standard treatment options include:

- Surgery with or without postoperative radiotherapy
- Definitive radiotherapy with surgery for the salvage of radiation failures
- Chemotherapy administered concomitantly with radiotherapy can be considered for patients who would require total laryngectomy for control of disease.

Subglottis

Standard treatment options include:

- Laryngectomy plus isolated thyroidectomy and tracheoesophageal node dissection usually followed by postoperative radiotherapy
- Treatment by radiotherapy alone is indicated for patients who are not candidates for surgery.

Stage IV Laryngeal Cancer

Supraglottis

Standard treatment options include:

- Total laryngectomy with postoperative radiotherapy
- Definitive radiotherapy with surgery for salvage of radiation failures
- Chemotherapy administered concomitantly with radiotherapy can be considered for patients who would require total laryngectomy for control of disease.

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Glottis

Standard treatment options include:

- Total laryngectomy with postoperative radiotherapy
- Definitive radiotherapy with surgery for salvage of radiation failures
- Chemotherapy administered concomitantly with radiotherapy can be considered for patients who would require total laryngectomy for control of disease.

Subglottis

Standard treatment options for:

- Laryngectomy plus total thyroidectomy and bilateral tracheoesophageal node dissection usually followed by postoperative radiotherapy.
- Treatment by radiotherapy alone is indicated for patients who are not candidates for surgery.

Treatment Types

Radiation

Radiation is sometimes preferred because of the good oncologic results, preservation of the voice, and the possibility of surgical salvage in patients whose disease recurs locally.

Surgery

Surgical treatment options should be reviewed carefully to ensure adequate pulmonary and swallowing function postoperatively.

● Transoral Laser Microsurgery. Transoral laser microsurgery is ideal for the treatment of early to intermediate glottic and supraglottic cancer. It may be performed under suspension microlaryngoscopy with a laser. The tumor may be transected and removed piecemeal. This treatment has the same indications and contraindications as open partial laryngectomies. A functional cricoarytenoid unit must be preserved. Survival and laryngeal preservation is comparable to other conventional treatments.

● TORS. Transoral robotic-assisted supraglottic laryngectomy may be appropriate for certain supraglottic cancers.

● Partial Laryngectomy

Vertical Hemilaryngectomy. When disease is small and involves only one vocal fold and arytenoid, a vertical hemilaryngectomy can be performed. This procedure removes, unilaterally, the true vocal fold from anterior to commissure to vocal process of arytenoid, as well as the false vocal fold, the ventricle paraglottic space, and the overlying thyroid cartilage. Resection can be extended to include the whole arytenoid. A frontolateral hemilaryngectomy additionally removes the anterior commissures, part of the contralateral vocal fold, and the overlying thyroid cartilage.

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Supraglottic Laryngectomy. Supraglottic laryngectomy can be performed in tumors limited to the supraglottic region with normal vocal fold mobility, with 2 mm margin at the anterior commissure. It is contraindicated if disease extends anteriorly into the thyroid cartilage or the anterior neck, posteriorly to postcricoid or intraarytenoid areas or prevertebral fascia, and in patients with poor pulmonary function. This procedure may remove the entire epiglottis and aryepiglottic folds, false vocal folds, preepiglottic space, and part of the thyroid cartilage and it can be extended to include the vallecula, pyriform sinus, and the base of the tongue up to the circumvallate papillae.

Supracricoid Laryngectomy. Supracricoid laryngectomy can be performed for removal of large, even certain T4 tumors of the glottis and supraglottis. However, it is contraindicated in subglottic extension, 10 mm anteriorly or 5 mm posteriorly, and with involvement of both arytenoid cartilages, the hyoid bone, the base of the tongue, or the cricoid cartilage. Supracricoid laryngectomy resects both true and both false vocal folds, the paraglottic space, the entire thyroid cartilage, as well as the epiglottis and one arytenoid if necessary. Reconstruction involves cricohyoidoepiglottopexy or cricohyoidopexy if epiglottis is removed.

● Total Laryngectomy. A total laryngectomy may be performed in the event of radiation failure with unclear extent of recurrence, chondroradionecrosis, or when other conservation techniques are not feasible. A total laryngectomy removes the entire larynx, including the thyroid and cricoid cartilages, part of the thyroid gland, the paratracheal lymph nodes, and may involve removal of the overlying strap muscles.

◆ Complications

- Psychosocial trauma from surgery and/or radiotherapy
- Vocal fold–powered voice loss in some procedures
- Aspiration pneumonia, in some procedures
- Osteoradionecrosis
- Chondroradionecrosis
- Chronic pain
- Breathing difficulties
- Stoma infections
- Potential stoma malignancies

◆ Outcome and Follow-Up

Consideration should be given to airway protection, nutritional support, and voice rehabilitation. Establishing a stable airway via tracheotomy should be considered preoperatively for tumors threatening the airway or obscuring intubation. Preemptive gastrostomy tube placement can ensure maximized preoperative and postoperative nutritional status. If voice preservation is not possible in the treatment plan, a noninvasive option for voice rehabilitation is an electrolarynx. Alternately, patients can learn techniques for producing

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esophageal voice, using expulsion of air through the esophagus to generate the base vibration for speech. With tracheoesophageal puncture, a fistula can be created between the cervical trachea and the esophagus to allow efficient swallowing of air for esophageal voicing.

Close oncologic follow-up care is necessary because second primary cancers, recurrences, and late metastases are all strong possibilities, especially if the patient continues to smoke.

◆ ICD-9 Codes

161	Malignant neoplasm of larynx
161.0	Malignant neoplasm of glottis
161.1	Malignant neoplasm of supraglottis
161.2	Malignant neoplasm of subglottis
161.3	Malignant neoplasm of laryngeal cartilages
161.8	Malignant neoplasm of other specified sites of larynx
161.9	Malignant neoplasm of larynx unspecified

◆ Staging of Laryngeal Cancer

Primary Tumor: Supraglottis

- TX: Primary tumor cannot be assessed
- T0: No evidence of primary tumor
- Tis: Carcinoma in situ
- T1: Tumor limited to one subsite of supraglottis *with normal vocal fold mobility*
- T2: Tumor invades mucosa of more than one adjacent subsite of supraglottis or glottis or region outside the supraglottis (e.g., the mucosa of base of tongue, vallecula, or medial wall of pyriform sinus) *without fixation of the larynx.*
- T3: Tumor limited to larynx *with vocal fold fixation* and/or invades any of the following: the postcricoid area, preepiglottic tissues, paraglottic space, and/or minor thyroid cartilage erosion (e.g., inner cortex).
- T4a: Moderately advanced local disease. Tumor invades through the thyroid cartilage and/or invades tissues beyond the larynx (e.g., the trachea, the soft tissues of the neck including the deep extrinsic muscle of tongue, the strap muscles, the thyroid, or the esophagus).
- T4b: Very advanced local disease. Tumor invades the prevertebral space, encases the carotid artery, or invades the mediastinal structures.

Primary Tumor: Glottis

- TX: Primary tumor cannot be assessed
- T0: No evidence of primary tumor
- Tis: Carcinoma in situ

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- T1: Tumor limited to the vocal fold(s) (may involve anterior or posterior commissure) *with normal mobility*
- T1a: Tumor limited to one vocal fold
- T1b: Tumor involves both vocal folds
- T2: Tumor extends to the supraglottis and/or the subglottis *and/or with impaired vocal fold mobility*
- T3: Tumor limited to the larynx *with vocal fold fixation* and/or invasion of the paraglottic space, and/or the inner cortex of thyroid cartilage
- T4a: Tumor invades through the outer cortex of the thyroid cartilage and/or invades the tissues beyond the larynx (e.g., the trachea, the soft tissues of the neck including the deep extrinsic muscle of the tongue, strap muscles, the thyroid, or the esophagus)
- T4b: Tumor invades the prevertebral space, encases the carotid artery, or invades the mediastinal structures

Primary Tumor: Subglottis

- TX: Primary tumor cannot be assessed
- T0: No evidence of primary tumor
- Tis: Carcinoma in situ
- T1: Tumor limited to the subglottis
- T2: Tumor extends to the vocal fold(s) *with normal or impaired mobility*
- T3: Tumor limited to the larynx *with vocal fold fixation*
- T4a: Tumor invades the cricoid or thyroid cartilage and/or invades the tissues beyond the larynx (e.g., the trachea, the soft tissues of neck including the deep extrinsic muscles of the tongue, the strap muscles, the thyroid, or the esophagus)
- T4b: Tumor invades the prevertebral space, encases the carotid artery, or invades the mediastinal structures

T Category Considerations

- Supraglottis: Normal vocal fold mobility (T1), fixation of the larynx (T2), and vocal fold fixation (T3) may only be determined clinically.
- Glottis: Normal vocal fold mobility (T1), impaired vocal fold mobility (T2), and vocal fold fixation (T3) may only be determined clinically.
- Subglottis: Normal or impaired vocal fold mobility (T2) and vocal fold fixation (T3) may only be determined clinically.

Primary Tumor: Regional Lymph Nodes*

- NX: Primary tumor cannot be assessed
- N0: No regional lymph node metastasis
- N1: Metastasis in a single ipsilateral lymph node, $\leq$3 cm in greatest dimension

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- N2: Metastasis in a single ipsilateral lymph node, >3 cm but not >6 cm in greatest dimension, or in multiple ipsilateral lymph nodes, none >6 cm in greatest dimension, or in bilateral or contralateral lymph nodes, none >6 cm in greatest dimension
- N2a: Metastasis in a single ipsilateral lymph node, >3 cm but not >6 cm in greatest dimension
- N2b: Metastasis in multiple ipsilateral lymph nodes, none >6 cm in greatest dimension
- N2c: Metastasis in bilateral or contralateral lymph nodes, none >6 cm in greatest dimension
- N3: Metastasis in a lymph node >6 cm in greatest dimension

*Superior mediastinal lymph nodes are considered regional lymph nodes (level VII). Midline nodes are considered ipsilateral nodes.

Distant Metastasis

- MX: Distant metastasis cannot be assessed
- M0: No distant metastasis
- M1: Distant metastasis

American Joint Committee on Cancer Stage Groupings of Supraglottis, Glottis, and Subglottis

Stage 0

Tis N0 M0

Stage I

T1 N0 M0

Stage II

T2 N0 M0

Stage III

T1 N1 M0
T2 N1 M0
T3 N0 M0
T3 N1 M0

Stage IVA

T1, N2, M0
T2, N2, M0
T3, N2, M0
T4a N0 M0
T4a N1 M0
T4a N2 M0

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Stage IVB

T4b any N M0
Any T N3 M0

Stage IVC

Any T any N M1

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5.2.9 Dysphonia and Aphonia After Laryngectomy

◆ Key Features

- Partial laryngectomy may make phonation difficult, resulting in dysphonia.
- Total laryngectomy, associated with removal of the larynx and modification of the respiratory tract, results in a total loss of phonatory ability (aphonia).
- Evaluation for appropriate posttotal laryngectomy communication is multifactorial.
- The three primary options for communication are the electrolarynx, tracheoesophageal puncture, and esophageal speech.

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Surgery to the larynx, whether in the form of total or partial laryngectomy, has the potential to greatly impact the vocal communication system. Partial laryngeal surgery often requires intensive vocal rehabilitation, and full functionality may never be regained. Total laryngectomy results in aphonia, and there are several communication options to replace this function.

◆ Epidemiology

The annual incidence of diagnosed head and neck cancer in the United States is ~45,660 cases. Of these, cancer of the larynx accounts for 25% of cases (see Chapter 5.2.8). Cancers diagnosed in the first or second stage are more likely to be treated with local surgical excision or chemoradiation therapy; cancers of the larynx in the third or fourth stage are more likely to result in a total removal of the larynx in combination with chemotherapy and radiotherapy. Of the three communication options postlaryngectomy, 55% of individuals use an electrolarynx as a primary communication method, 31% use a tracheoesophageal puncture prosthesis, and 6% use the esophageal speech method (8% remain nonvocal).

◆ Clinical

Signs and Symptoms

Following partial laryngeal surgery, patients often present with dysphonia characterized by a weak, strained, or breathy vocal quality. Patients who have had a total laryngectomy have a total inability to phonate postoperatively secondary to removal of the larynx, including the vocal folds.

Differential Diagnosis

In patients with partial laryngeal surgery, it is important to determine whether the current vocal qualities are a result of surgical treatment versus an advancement or recurrence of the carcinoma. Any change in previous alaryngeal communication abilities of individuals following a total laryngectomy can indicate recurrence of cancer and should be carefully evaluated.

◆ Evaluation

Evaluation for communication methods following total laryngectomy include an evaluation of physical changes from surgery and chemoradiation therapy to assess for the ability for electronic larynx placement either transcervically (neck-type) or intraorally (mouth-type), stoma size and placement for stomal occlusion with tracheoesophageal puncture voicing. Additionally, manual dexterity, motivation level, and financial/insurance resources should be considered.

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◆ Treatment Options

After Partial Laryngeal Surgery

Voice rehabilitation following partial laryngeal surgery should include treatment with a speech-language pathologist and should focus on appropriate vocal hygiene, adequate breath support, decreased muscle tension, and oral resonance (see Chapter 4.3.4).

After Total Laryngectomy

Electrolarynx

A battery-powered electronic device called an electrolarynx is used. Depending on anatomic changes following surgery, an electrolarynx can be placed either transcervically (neck-type) or intraorally (mouth-type). The electrolarynx produces a vibration that is transmitted intraorally through a straw attached to the device or through the tissues of the neck or cheek.

The electrolarynx offers a communication option immediately after surgery, is relatively easy to use, and has a lower one-time cost (when compared with the tracheoesophageal puncture voice prosthesis). Disadvantages include a mechanical sound quality, requirement for one free hand during communication, and unfamiliarity of the sound by most listeners.

Tracheoesophageal Puncture Voice Prosthesis

For the tracheoesophageal puncture voice prosthesis, a small fistula is surgically placed in the tracheoesophageal wall, ~1 cm below the upper lip of the stoma. This fistula is kept patent by the insertion of a one-way valved prosthesis. Voicing is then achieved by passing air from the trachea to the esophagus via stomal occlusion with either manual finger occlusion or a hands-free stomal attachment.

The voice prosthesis allows for an esophageal sound production, which is then shaped by the oral cavity for speech production. Individuals with a laryngectomy often feel this method allows for speech to be most comparable to preoperative speech in terms of quality, fluency, and ease of production. Disadvantages include anatomic variations and mechanical problems. Anatomic variations include hypertonicity or flaccidity of the pharyngoesophageal muscle segment, stomal stenosis, or stoma irregularity. Mechanical problems include size, fit, and prosthesis breakdown secondary to *Candida* infection or gastroesophageal reflux disease, or dislodgement. Other disadvantages include the cost of the prosthesis (which must be replaced every few months), accessibility to a speech-language pathologist or otolaryngologist trained to change and maintain indwelling valves, and manual dexterity for cleaning and management.

Esophageal Speech

Speech is produced from a learned method of vibrating the pharyngoesophageal muscle segment. Air is introduced into the esophagus through the oral cavity and is then passed back out of the esophagus past the

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pharyngoesophageal segment. This can be done using either a glossopharyngeal press method or an inhalation method.

Esophageal speech allows for communication without the use of mechanical or prosthetic devices and allows for more natural sound production. Disadvantages include an increased time period for learning this method (estimated 4 to 6 months of regular speech therapy and daily practice), limited success rates, and decreased ability to control volume.

◆ Outcome and Follow-Up

Any patient with laryngeal carcinoma must follow up with an otolaryngologist for at least 5 years postoperatively. A patient with an indwelling tracheoesophageal puncture voice prosthesis must follow up with a speech-language pathologist or otolaryngologist trained in prosthesis management for all changes, approximately every 3 months. A patients with a nonindwelling tracheoesophageal puncture voice prosthesis (i.e., one that is removed, cleaned, and reinserted by the patient on a frequent basis) should follow up with an speech-language pathologist or otolaryngologist yearly for tract evaluation and fitting. Additionally, follow-up should occur immediately for prosthesis dislodgement or significant change in sound quality, as this can indicate a more significant problem, recurrence, aspiration, or may allow tract stenosis or closure.

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5.2.10 Referred Otalgia in Head and Neck Disease

◆ Key Features

- Referred otalgia is the sensation of ear pain originating from a source outside the ear.
- If no otic source for otalgia can be identified malignancy must be investigated and excluded.

By definition, referred otalgia is the sensation of ear pain originating from a source outside the ear. Many remote anatomic sites share innervations with the ear, and noxious stimuli to these areas may be perceived as otalgia.

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In adult patients, especially those at risk, if no otic source for otalgia can be identified malignancy must be investigated and excluded, especially for adult patients with known risk factors.

The sensory innervation of the ear is provided by the auriculotemporal branch of the fifth CN (mandibular nerve [CN V3]), contributions from C2 and C3 through the cervical plexus via the greater auricular nerve, Jacobson's nerve [branch of CN IX], Arnold's nerve [branch of CN X], and the sensory branch of CN VII [Ramsay Hunt branch], which innervates a portion of the external auditory canal and surrounding concha.

The explanation for complex sensory innervation of the ear ultimately lies in the ear's embryologic development. The otic vesicle comes to rest central to branchial arches 1, 2, 3, and 4. The sensory and motor nerves of these arches are CN V, CN VII, CN IX, and CN X, respectively.

◆ Etiology

See **Table 5.7**. Lesions of the anterior two thirds of the tongue and inferior oral cavity tend to refer pain to the ear via CN V3. Lesions of the lateral base of tongue, tonsillar region, and inferior two thirds of the nasopharynx cause deep, intense otalgia via sensory impulses through CN IX. Lesions of the supraglottic larynx communicate sensory afferents via the internal laryngeal branch of the superior laryngeal nerve of CN X. Lesions of the posterior oropharynx, the inferior nasopharynx, the medial aspect of the base of tongue, and the hypopharynx tend to refer pain to the ear owing to the overlapping innervations of both CN IX and X (via the pharyngeal plexus).

◆ Evaluation

The evaluation of a patient with otalgia begins with a detailed history and a thorough head and neck examination. Questions as to the character and

Table 5.7 Etiology for Referred Otalgia

Dental disorders
Ill-fitting dentures
Bruxism
Cervical osteoarthritis
Cervicofacial myofascial pain syndrome
Posttraumatic neuralgia (greater auricular neuroma)
Chronic pharyngitis
Eagle syndrome
Postviral neuralgia (Ramsay Hunt syndrome, postherpetic neuralgia)
Temporal arteritis
Parotid neoplasia
Gastroesophageal reflux

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timing of the otalgia, exacerbating and alleviating factors of the otalgia, the patients past otologic history, the associated symptoms with the otalgia (tinnitus, hearing loss, vertigo), the presence of constitutional symptoms (to detect malignancies), and sinus and dental questions.

A head and neck examination is fundamental in evaluating otalgia. A thorough otologic examination, with a tuning fork test at two frequencies (256 and 512 Hz), is important. The CNs are examined and compared bilaterally. The nose, sinuses, oral cavity, oral pharynx, and neck are inspected and palpated to look for sources of referred otalgia. In assessing otalgia in the setting of a normal otologic examination, a fiberoptic nasopharyngolaryngoscopy is mandated to look for lesions that can be potentially noxious to the trigeminal, facial, glossopharyngeal, or vagus nerves. Attention should be directed to the endolarynx to examine the mucosa for signs of malignancy and gastroesophageal reflux.

◆ Treatment Options

Appropriate treatment of the source of otalgia in combination with pain management.

◆ ICD-9 Codes

388.7 Otalgia
388.70 Otalgia unspecified
388.72 Referred otogenic pain

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5.2.11 Neck Dissection

◆ Key Features

- The presence of cervical lymph node metastasis reduces survival of patients with head and neck squamous cell cancer by up to 50%.
- Neck dissection or lymphadenectomy is a surgical procedure in which the fibrofatty contents of the neck are removed for the prevention or treatment of cervical metastasis.
- Most commonly used in the treatment of cancers of the upper aerodigestive tract, skin of the head and neck, thyroid, and salivary glands.

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Cancers of the head and neck tend to metastasize to cervical lymph nodes. The term "neck dissection" refers to the systematic removal of lymph nodes in the neck. To eradicate cancer in the cervical lymph nodes and to help determine the need for additional therapy (staging) when no lymph nodes are clinically identified, neck dissection may be performed. Although used most commonly for the management of cancers of the upper aerodigestive tract, neck dissection is also used for malignancies of the skin of the head and neck, the thyroid, and the salivary glands.

The original surgical procedure described for the treatment of metastatic neck cancer was a radical neck dissection (RND); it was first described by Crile in 1906 and until several decades ago this was considered to be the standard procedure for the management of clinically positive neck disease in the setting of head and neck cancer. More recently, a shift toward more conservative surgical procedures has been adopted. This shift aims at removing lymphatic tissue but preserving adjacent nonlymphatic structures. In addition, specific nodal groups at risk for metastatic disease as predicted by the size, location, and other features of the primary tumor are addressed in a selective fashion in certain cases.

◆ Classification of Neck Levels

The evaluation of the drainage pattern of the primary tumor site in the upper aerodigestive tract has led to the understanding and identification of nodal groups at risk for cervical metastases. The neck has been divided into six such groups called neck levels.

Level 1: The Submandibular and Submental Triangles

Level 1 consists of the submandibular and submental triangles. The submandibular triangle is bordered by the mandible superiorly, the posterior belly of the digastric muscle posteroinferiorly, and the anterior belly of the digastric muscle anteroinferiorly. Its floor is the hyoglossus muscle. The submental triangle is the region between the bilateral anterior bellies of the digastric muscle and the hyoid bone.

Level 2: The Jugular Digastric Region

Level 2 is known as the jugular digastric region. Its boundaries are the skull base superiorly, the carotid bifurcation inferiorly, posterior border of the sternocleidomastoid muscle, and the lateral border of the sternohyoid and sternothyroid muscles medially.

Level 3: The Middle Jugular Region

Level 3 is the middle jugular region. It is bordered by the carotid bifurcation superiorly, the junction of the omohyoid and sternocleidomastoid muscle at the jugular vein inferiorly, the posterior border of the sternomastoid muscle laterally, and the lateral border of the sternohyoid muscle medially.

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Level 4: The Lower Jugular Region

Level 4 is the lower jugular region and extends from the omohyoid superiorly to the clavicle inferiorly. It extends to the posterior border of the sternomastoid muscle and the lateral border of the sternohyoid muscle medially. The fascia overlying the phrenic nerve and the brachial plexus is the deep boundary.

Level 5: The Posterior Triangle

Level 5 is also known as the posterior triangle. It includes the lymph nodes between the posterior border of the sternomastoid muscle and the anterior border of the trapezius muscle. It also extends to the clavicle inferiorly. The course of the spinal accessory nerve is encompassed in this triangle. The "supraclavicular region" is also found in level 5.

Level 6: The Anterior Compartment

Level 6 is the anterior compartment and includes the midline lymph nodes adjacent to the trachea and thyroid gland. The borders of this region are the hyoid bone superiorly, the sternal notch inferiorly, and the carotid sheath laterally.

◆ Classification of Neck Dissections

The current classification of neck dissection has been developed by the Committee of Head and Neck Surgery & Oncology of the American Academy of Otolaryngology–Head and Neck Surgery and is based on the following principles:

1. An RND is the standard basic procedure for removal of lymph nodes from the neck and all other procedures represent a modification of this procedure. It is defined as the en block removal of the nodal groups between the mandible and the clavicle. An RND includes the removal of the internal jugular vein, the spinal accessory nerve, and the sternocleidomastoid muscle.
2. When modification of the RND involves preservation of a nonlymphatic structure, the procedure is termed a modified neck dissection.
3. When modification involves preservation of one or more lymph groups that are routinely removed in an RND, the procedure is termed a selective neck dissection (SND). The procedure is usually performed if there is no palpable neck disease (clinically N0 neck), but the risk of occult metastases to the cervical lymph nodes is likely >20%. For example, a T1 N0 SCC of the oral tongue extending deeper than 4 mm is an indication to perform an SND involving levels 1 to 3, possibly level 4. SNDs include a supraomohyoid neck dissection, a lateral compartment neck dissection, a posterior lateral neck dissection, and anterior compartment neck dissection.
   - Supraomohyoid neck dissection involves removal of levels 1 through 3 and is usually performed in the setting of oral cavity tumors and N0 neck disease.

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- Lateral compartment neck dissection includes levels 2 through 4 and is used in conjunction with the surgical resection of tumors of the larynx, hypopharynx, pharynx, and thyroid.
- Posterior lateral neck dissection includes levels 2 through 5. It may include the retroauricular and suboccipital nodal regions as well. Posterior lateral neck dissection is typically performed for cutaneous malignancies of the scalp and face.
- Anterior compartment or central compartment neck dissection includes level 6 and is used for tumors found in the larynx, the hypopharynx, the subglottis cervical esophagus, and the thyroid. Anterior neck dissection is commonly performed for papillary thyroid cancer with metastases to the lymph nodes.

4. When modification involves removal of addition lymph node groups or nonlymphatic structures relative to the RND, the procedure is termed an extended RND. An extended RND involves the additional removal of muscles, nerves, vessels, and lymph nodes as dictated by primary disease or the presence of metastases found in surgery. An extended neck dissection may involve the retropharyngeal lymph nodes, the hypoglossal nerve, portions of the prevertebral musculature and the carotid artery. Also, certain parotid gland malignancies may require total parotidectomy combined with neck dissection.

◆ Complications of Neck Dissection

Complications may be divided into intraoperative or postoperative. It is important to remember that certain medical conditions such as postradiation treatment, poor nutritional status, hypothyroidism, alcoholism, and diabetes may increase the risk of intraoperative and postoperative complications.

Intraoperative complications of neck dissections include hemorrhage and damage to adjacent CNs. During a submandibular submental dissection, the marginal mandibular branch of the facial nerve, the hypoglossal nerve, and the lingual nerve are all at risk. Injury to the phrenic nerve may cause hemidiaphragm paresis, but is typically only symptomatic in patients with significant pulmonary disease. Injury to the vagus may cause vocal fold paralysis. Injury to the brachial plexus is rare but can occur, causing upper extremity weakness. Injury to the sympathetic trunk may cause Horner syndrome. Postoperative complications include hematoma, shoulder dysfunction, wound infection, salivary or chylus fistula, and carotid artery blowout.

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5.2.12 Skin Cancer of the Head, Face, and Neck

5.2.12.1 Basal Cell Carcinoma

◆ Key Features

- Basal cell carcinoma is the most common skin cancer (80%).
- It is derived from basilar keratinocytes.
- Basal cell carcinoma is slow growing, rarely metastasizing.
- It has a high cure rate from excision with minimal margins.

Basal cell carcinoma (BCC) is the most common cancer in humans. As sun exposure is the heaviest risk for developing BCC, 90% of these cancers are found in the head and neck region. Although prevention is best (by reducing sun exposure), BCC is rarely aggressive with very low rates of metastasis ($<1\%$) and can usually be treated with complete surgical excision.

◆ Epidemiology

No great epidemiologic source exists for BCC, as it is usually included under the "nonmelanoma" skin cancer data. The National Cancer Institute estimates $>1{,}000{,}000$ new cases of BCC and SCC combined, with mortality figures of <2000 individuals. In the United States, BCC is more common in the Sunbelt states. Occupations such as farmers, construction workers, and others who spend many hours in the sun have a skin cancer risk. Risk factors also include light pigmented skin and blue/green eyes. Persons of African descent rarely develop BCC, but there is higher incidence in albino Africans, pointing to the central protective role of melanin. Genetic conditions with high BCC incidence include xeroderma pigmentosum (autosomal recessive defect in DNA repair protein) and basal cell nevus syndrome (i.e., Gorlin syndrome, which is an autosomal dominant condition involving jaw cysts, bifid ribs, scoliosis, palmer pits, and multifocal BCC in situ in childhood). Other risk factors include damage to skin via ultraviolet (UV) B radiation (sun, tanning beds), ionizing radiation exposure, arsenic exposure, prior trauma or chronic irritation, and immunosuppression.

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◆ Clinical

Signs and Symptoms

Basal cell carcinoma usually presents as an indolent growth or discolored patch on the skin, rarely with itching, bleeding, and ulceration. The appearance of the lesion varies with the type (see Pathology, below).

Differential Diagnosis

- Actinic keratosis
- Seborrheic keratosis
- Keratoacanthoma
- Nevi
- Bowen disease
- SCC
- Melanoma

◆ Evaluation

Physical Exam

A routine physical exam should be performed with an emphasis on the scalp, folds around nose, lips, canthi, and ears, as these areas carry higher risk of recurrence.

Other Tests

No imaging or laboratory tests are necessary, but suspicious lesions should be biopsied.

Pathology

There are four histopathologic types of BCC:

Nodular: This the most common form of BCC (60–80%). It most often appears on the head, neck, and upper back. The lesions appear as waxy papules with central depression or rolled edges, and a pearly translucency. Often, telangiectasias appear over the surface, which can lead to bleeding and crusting, especially with ulceration.

Morpheaform: This form constitutes 10 to 20% of BCC. This is the most aggressive variety, with the worst prognosis. It appears commonly on the face as a sclerotic, sometimes depressed, plaque with yellowish color and irregular borders. Bleeding and ulceration are rare, and this lesion may be mistaken as a scar. These tumor cells express collagenases, which allow them to travel along peripheral nerves and embryonic fusion planes.

Pigmented: This type is very similar to the nodular type, but with the additional characteristic of pigmentation, which may resemble a benign

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nevus or melanoma. These lesions are more commonly found on darker-skinned individuals.

Superficial: This type appears commonly on the trunk (rarely on the head and neck) as scaly, indurated patch with an irregular border, often mimicking psoriasis or eczema or resembling actinic keratosis. Growth is slow with low rates of erosion or invasion. Multiple superficial BCCs have been associated with arsenic exposure.

◆ Treatment Options

Medical

The application of 5-FU has some success with BCC of trunk and extremities, but is not approved for use in the head and neck region.

External beam radiation can be used effectively and has gained favor over superficial x-rays by many radiation oncologists. Ionizing radiation is a good treatment option for patients who are not surgical candidates, especially those patients who have facial tumors.

Surgical

Most commonly, BCC is removed in the ambulatory setting by curettage with electrodesiccation, using a looped blade to scrape away the lesion from normal skin, vigorously and in multiple directions. This is best used for small nodular or superficial BCCs, with a 90% success rate, but should not be used with aggressive types of BCC or in cosmetically or functionally sensitive areas. Surgical excision with 4-mm margins has a 95% cure rate and can be performed for any BCC variant. Primary closure has better cosmetic outcome than healing by secondary intention. The most effective procedure (96–99% cure rate) is Mohs micrographic excision (**Fig. 5.8**). In this technique, the tumor is removed and the margin mapped and color coded, then examined thoroughly for remaining cancer. If there is remnant cancer, the surgeon returns to that specifically mapped area and removes tissues, repeating this process until all margins are clear. This procedure is best used for cosmetically or functionally sensitive areas in which wide margins cannot be easily removed, or for aggressive, recurring, or large tumors.

Cryosurgery can also reliably treat small, nonaggressive BCC.

◆ Outcome and Follow-Up

Patients who are diagnosed with BCC have a 35 to 40% chance of developing another tumor within 3 years and a 50% chance of developing another (not recurrent) basal cell carcinoma within 5 years. Therefore, regular skin screenings are recommended. Wearing hats to protect the head and neck from sun exposure and sun-block usage should be encouraged.

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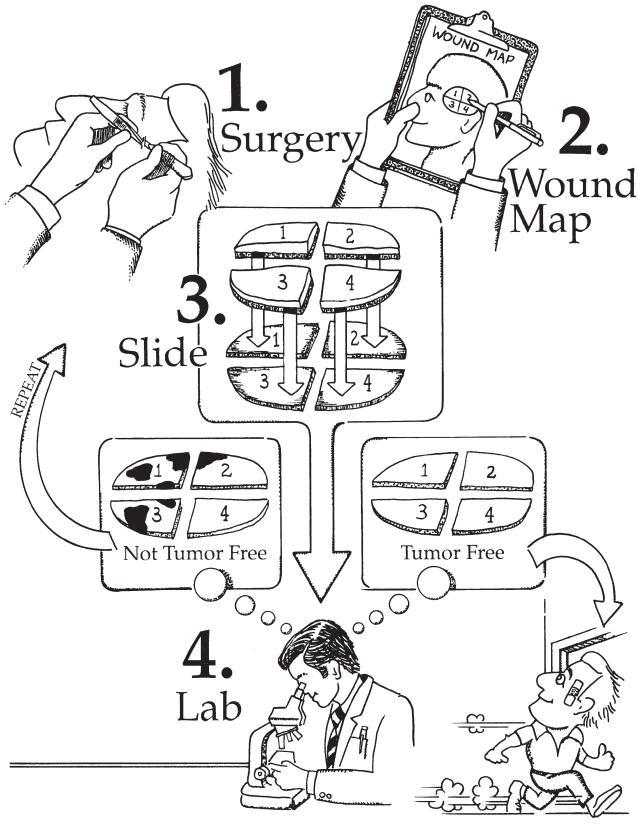

Fig. 5.8 Steps in Mohs micrographic excision. (From Papel ID, ed. Facial Plastic and Reconstructive Surgery. 3rd ed. Stuttgart/New York: Thieme; 2009:683.)

◆ ICD-9 Codes

173	Other malignant neoplasm of skin
173.0	Other malignant neoplasm of skin of lip
173.1	Other malignant neoplasm of skin of eyelid including canthus
173.2	Other malignant neoplasm of skin of ear and external auditory canal
173.3	Other malignant neoplasm of skin of other and unspecified parts of face
173.4	Other malignant neoplasm of scalp and skin of neck

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5.2.12.2 Squamous Cell Carcinoma

◆ Key Features

- Squamous cell carcinoma (SCC) is the second most common skin cancer.
- Its aggressiveness is related to size and lymphatic spread.
- Surgical excision is the mainstay of treatment.

SCC is the second most common skin cancer in humans, after BCC. SCC is more aggressive than BCC, as it frequently grows vertically, leading to lymphatic penetration and metastasis. In addition to sun exposure, chronic damage to skin and immunosuppressive states are risk factors. SCC arising from previously damaged skin (scar, burn, or chronic injury, such as Marjolin's ulcer) and large primary lesions (>2 cm) are more aggressive, with higher rates of recurrence and metastasis. Lesions <2 cm have 5% recurrence and 7% rates of metastasis. Lesions >2 cm have double the recurrence rate and three times the rate of lymphatic metastatic spread. As in the case of BCC, prevention is best by reducing sun exposure and repeated injury; however, SCC can usually be treated successfully with surgical excision of the primary lesion along with affected lymphatic tissue.

◆ Epidemiology

The National Cancer institute estimates over 1,000,000 new cases of BCC and SCC combined, with mortality figures of <2000 individuals. The epidemiologic factors of SCC are UV-B light exposure, related genetic sensitivity to the sun, chemical carcinogen exposure, previous radiation exposure, and chronic inflammation, immunosuppression, and HPV infection.

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◆ Clinical

Signs and Symptoms

The skin appearance of SCC is diverse: SCC may appear as a scaly, rough patch on an erythematous base on sun-exposed skin. The lesion may look like a scar but does not heal and often bleeds and ulcerates. Similarly, a nonhealing ulcer or wound may be housing SCC. SCC may also arise from erythroplakic lesions. The appearance of the lesion varies with the type.

Differential Diagnosis

- Bowen disease
- Cutaneous horn
- Actinic keratosis
- Keratoacanthoma
- Wart
- Blastomycosis
- Basal cell carcinoma
- Melanoma

◆ Evaluation

History

An appropriate history should include a special emphasis on previous and current cutaneous lesions or discolorations.

Physical Exam

A routine physical exam should be performed. Lymph nodes in the parotid and the neck should be assessed, especially with lesions involving the nose, ear, and temple.

Imaging

No imaging or laboratory tests are necessary for isolated cutaneous lesions (if adenopathy suspected, imaging is needed to assist with staging), but CT, MRI, and PET are useful for determining the extension of tumor if advanced.

Other Tests

Suspicious lesions require either a one-punch biopsy or two shave biopsies, which include the level of the middermis.

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Pathology

Histologically, SCC can be graded as well, moderately, and poorly differentiated. Grossly, it can be ulcerative, infiltrative, or exophytic.

Variants of SCC include the following:

Spindle cell, a biologically aggressive variant of SCC may resemble soft tissue sarcoma, but can be differentiated with a positive vitronectin antibody test. The spindle cells are elongated nuclei in a whorled pattern. Although it is a rare variant, it is more commonly seen at sites of previous scars or chronic injury.

Adenoid (acantholytic) displays pseudoglandular differentiation with a separation of keratinocytes from each other. Histologically, it may resemble adenocarcinoma, but clinically it appears as typical SCC, usually in the elderly, in the periauricular region. This variant is also considered biologically aggressive.

Indolent variants include Bowen disease (SCC in situ), which is characterized histologically by hyperkeratosis, parakeratosis, and acanthosis with thickened and elongated rete ridges.

Keratoacanthoma is a relatively common low-grade malignancy that originates in the pilosebaceous glands and closely and pathologically resembles SCC (rapid growth around hair follicle, spontaneous resolution in 4–6 months). Verrucous carcinoma is a low-grade variant of SCC with little potential for distant metastases. However, it has the potential to cause local destruction.

◆ Treatment Options

The treatment of SCC is surgical excision. Small, well-differentiated tumors can be removed safely with 4-mm margins, while lesions >2 cm, moderately undifferentiated with subcutaneous fat involvement, require at least 6-mm margins, including a portion of subcutaneous fat. If parotid or cervical lymph nodes are clinically involved, superficial parotidectomy and modified RND is recommended. Mohs micrographic excision can be employed, especially in cosmetically areas (**Table 5.8**).

Radiotherapy is effective as the primary therapy for poor surgical candidates and cosmetically sensitive areas, or as adjunct therapy to minimize recurrence, treat positive or narrow margins, treat lymphatic spread, or to debulk large lesions prior to excision.

◆ Outcome and Follow-Up

After treatment of early disease, patients should be followed every 6 months for 2 years, and then yearly. More advanced disease should be followed more frequently (3–6 months) for 2 years, with increased intervals subsequently. Previous SCC greatly increases chance of secondary tumors; patients should receive full skin exams yearly. Protection against sun exposure (wide-brim hats, sunblock) should be stressed.

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Table 5.8 Treatment Options for Squamous Cell Carcinoma (SCC) and Basal Cell Carcinoma (BCC)

Mohs micrographic excision
If one or more of these factors are present:
Recurrent tumor
Tumor size larger than 2 cm in diameter
Tumors with an aggressive histology (such as morpheaform, infiltrative, micronodular BCC or poorly differentiated SCC)
Tumors with ill-defined margins
Tumors that are incompletely excised
Tumors with perineural invasion
Tumor location where maximal conservation of normal tissue is important (such as eyelid, nose, ear, lip, digit, genitalia)
Otherwise consider
Standard excision
Use at least
4- to 6-mm margins to a depth of mid to deep subcutaneous tissue if small (62 cm) primary BCC > SCC with clinically well-defined borders and nonaggressive histology
or
7-mm margins to a depth that includes all of the subcutaneous tissue if factor(s) are present for consideration of Mohs but Mohs is unavailable or patient refuses Mohs.
Radiation therapy
If patient is a poor surgical candidate
Refuses surgery
As postoperative treatment, if Mohs or standard excision shows perineural invasion or positive margins
Consider therapies other than radiation therapy if the patient has connective tissue disease (i.e., lupus, scleroderma) or is <50 years.
Less commonly used modalities
These modalities are primarily used for superficial BCC and SCC on the trunk and extremities:
Cryosurgery
Curettage and electrodesiccation
Laser
Photodynamic therapy

Source: Papel ID, ed. Facial Plastic and Reconstructive Surgery. 3rd ed. Stuttgart/New York: Thieme; 2009:682.

◆ ICD-9 Codes

173 Other malignant neoplasm of skin
173.0 Other malignant neoplasm of skin of lip
173.1 Other malignant neoplasm of skin of eyelid including canthus
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173.3 Other malignant neoplasm of skin of other and unspecified parts of face
173.4 Other malignant neoplasm of scalp and skin of neck

◆ Staging of Squamous Cell Carcinoma

High-Risk Features for Primary (T) Tumor Staging

Clinical: Primary site on ear or glabrous lip
Histologic: ≥2-mm depth
Clark level IV/V
Perineural invasion
Lymph–vascular invasion
Poor differentiation

American Joint Committee on Cancer Stage Groupings of Squamous Cell Carcinoma

Stage 0

Tis N0 M0*

Stage I

T1 N0 M0

Stage II

T2 N0 M0

Stage III

T3 N0 M0
T3 N1 M0
T1 N1 M0
T2 N1 M0

Stage IV

T1 N2 M0
T2 N2 M0
T3 N2 M0
Any T N3 M0
T4 any N M0
Any T any N M1

*Primary site on ear or glabrous lip is considered a high-risk factor in the American Joint Committee on Cancer (AJCC), 7th edition, staging system that may be used in upstaging a tumor from pT1 to pT2. Other high-risk features include inflammatory response; association with actinic keratosis; association with HPV; association with Bowen disease; acantholytic, basaloid, small cell, signet ring, desmoplastic, or spindle cell histological subtypes; and follicular SCC.

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Anatomic (Clark) Levels

Level I

- Intraepidermal tumor only

Level II

- Tumor present in but does not fill and expand the papillary dermis

Level III

- Tumor fills and expands the papillary dermis

Level IV

- Tumor invades into the reticular dermis

Level V

- Tumor invades the subcutis

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5.2.12.3 Melanomas of the Head and Neck

◆ Key Features

- Melanomas of the head and neck arise from neuroendocrine cells and stain strongly with HMB-45 and S-100
- Melanomas are aggressive: locally invasive, distant metastasis, and poorest prognosis of skin cancers.
- Depth of invasion is key in treatment and prognosis.
- Treat with wide local excision and chemotherapy.

Approximately 20% of melanomas occur in the head and neck region; 80% of these arise from the skin (most commonly the cheek, scalp, ear, and neck); the

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rest are mucosal (most commonly the anterior septum, middle and inferior turbinates, hard palate, and gingiva). In cases of melanoma, the depth of invasion is key in staging and prognosis. Suspicious lesions should be excised completely with a margin dictated by the depth of the lesion. Risk factors include UV-B light exposure, severe sunburns (>3 before age 20), large congenital nevi, familial dysplastic syndrome, fair skin, and blue or green eyes.

Mucosal melanoma of the head and neck is relatively rare. Mucosal melanomas show far more aggressive behavior as compared with skin melanomas and are more inclined to metastasize into regional and distant sites or recur locally, regionally, or in distant locations, resulting in a high rate of cause-specific death. The prognosis is grim, with most published reports documenting a 5-year survival rate of 10 to 15%.

The presence of a pigmented lesion in the oral or nasal cavities should raise suspicion of mucosal melanoma; a biopsy of the lesion should be promptly obtained. Diagnosis of pathologic disease is dependent on the identification of intracellular melanin.

◆ Epidemiology

The National Cancer Institute estimates 68,720 new cases of and 8,650 deaths from melanoma in 2009. There has been a clear upward trend in incidence over time. Melanoma has the highest rate of increase in incidence and the third highest increase in mortality among cancers in the United States. The highest prevalence of melanoma is in white males. Late-stage presentations (metastases) are more common in persons of African descent.

◆ Clinical

Signs and Symptoms

Melanomas usually present as macular or nodular areas of discoloration on the skin, sometimes aggravated by itching, ulceration, and bleeding. The ABCDEs point to melanoma:

1. Asymmetric shape
2. Border irregularity
3. Color variation (within parts of lesion, or over time)
4. Diameter (6 mm, or increase in size)
5. Evolution (stressing change in time)

Mucosal melanomas usually present as macular, nodular, or ulcerated lesions on mucosa, causing epistaxis, nasal obstruction, or mass in the oral cavity.

Differential Diagnosis

- Seborrheic keratosis
- Pigmented basal cell cancer
- Solar lentigines

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- Atypical nevi
- Hemangioma
- Melanoacanthoma
- Focal argyrosis
- Esthesioneuroblastoma
- Other neuroendocrine carcinomas

◆ Evaluation

Physical Exam

A routine physical exam, focusing on sun damage and commonly affected areas (cheek, scalp, neck, etc.), on ABCDEs of lesions, and palpation for lymphadenopathy.

Imaging

Imaging is not often used in early disease, but in more advanced disease or for suspicious cases PET-CT and MRI are useful.

Labs

Lactate dehydrogenase (LDH) in distant metastatic work-up

Other Tests

Immunohistochemical staining; HMB-45 (homatropine methylbromide) and S-100

Pathology

Superficial spreading (60–70%): Most common type, common in younger people, multicolored with notched border, usually from preexisting nevi at dermal–epidermal junction, radial growth (diameter increase) precedes vertical growth (ulceration), can invade all levels of dermis.

Nodular (15–30%): Most aggressive, worst prognosis, appears as dark (blue/black/red) pigmented nodule, 5% amelanotic, can arise de novo (unrelated to sun exposure), no radial growth, but rapid vertical growth with frequent bleeding and ulceration, regional and distant lymphadenopathy.

Acral lentiginous (10%): High incidence in Asians and Africans, large, dark, with irregular border on soles, palms, and subungual lateral growth.

Lentigo maligna (5%): Best prognosis, common in elderly over sun-damaged skin, large, flat, tan or brown, grows slowly, stays in dermal–epidermal junction and spreads radially, bleeding or sudden change in size signal vertical growth and invasion.

◆ Treatment Options

Treatment should be based on staging: there are three commonly used staging systems (see Staging of Melanomas of the Head and Neck, below).

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Breslow, Clark, and the American Joint Committee on Cancer (AJCC) are similar staging systems in that they are all based on depth of invasion. The microstage of malignant melanoma is determined on histologic examination by the vertical thickness of the lesion in millimeters (Breslow classification) and/or the anatomic level of local invasion (Clark classification). Currently, the most widely used system is the AJCC. AJCC melanoma staging guidelines have incorporated serum LDH for the classification of stage IV disease.

Medical

Interferon is usually given after surgical excision as an adjuvant therapy. High-dose treatment with interferon A2b increases disease-free survival and overall survival over observation; low-dose treatment is less effective (ECOG 1694 trial). Side effects of treatment include immunosuppression, anorexia, nausea/vomiting, liver toxicity (increased aspartate aminotransferase [AST]), and neurologic/neuropsychiatric problems.

Surgical

Complete surgical excision provides the best prognosis. The local treatment for melanomas is surgical excision with margins based on Breslow thickness and anatomic location: for in situ lesions, 0.5- to 1-cm margins. For most melanomas 2 to 4 mm in thickness, this means 2- to 3-cm radial excision margins. Clinically enlarged or matted lymph node beds require lymphadenectomy.

Melanoma that has spread to distant sites is rarely curable with standard therapy, though high-dose interleukin-2 (IL-2) has been reported to produce durable responses in a small number of patients.

Sentinel Lymph Node Biopsy

Fifteen to 35% of clinically negative nodes are sentinel lymph node–positive. Biopsy is indicated for lesions >1 mm deep. Preoperative lymphoscintigraphy can map the first echelon of draining nodes. Intraoperatively, inject Lymphazurin dye at the primary site, locate the sentinel node with a radioactive probe, and send for a frozen section. A positive sentinel node requires neck dissection and possibly adjuvant therapy.

◆ Outcome and Follow-Up

Even with 1-mm lesions with negative margins of resection, the recurrence rate is high. Ulceration and thickness of the primary lesion make recurrence much more likely. Mucosal melanomas have a high rate of recurrence. A history of melanoma itself is a risk factor for new primaries. Also, nodal disease can appear after primary excision. Therefore, patients should be monitored closely with routine history and physical exams, and possible chest x-ray and serum LDH for metastases.

For skin melanoma, prognosis is multifactorial and primarily depends on tumor thickness, the presence or absence of histologic ulceration, and lymph node involvement (most important). Despite advances in the treatment of

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metastatic disease, the detection and treatment of cutaneous melanoma in its thin, early phase remains the best chance for cure.

Melanoma of the Mucosal Membrane

Lesions of the oral cavity have a higher prevalence of lymph node metastasis than those occurring in either the nasal or the pharyngeal cavities. Overall, 18% of patients with mucosal melanoma have lymphatic metastases at presentation. The average distant metastatic rate at presentation is 10%.

Primary site recurrence occurs in ~40% of nasal cavity lesions, 25% of oral cavity lesions, and 32% of pharyngeal tumors. Overall primary site recurrence ranges from 55 to 66% and 16 to 35% for nodal recurrence. Most recurrences occur within the first 3 years.

◆ ICD-9 Codes

172 Malignant melanoma of skin
172.0 Malignant melanoma of lip
172.2 Malignant melanoma of ear and external auditory canal
172.3 Malignant melanoma of other and unspecified parts of face
172.4 Malignant melanoma of scalp and neck
172.9 Melanoma of skin, site unspecified

◆ Staging of Melanomas of the Head and Neck

Skin Melanoma

Clark's levels: Invasion through

- Level I: Lesions involving only the epidermis (in situ melanoma); not an invasive lesion
- Level II: Invasion of the papillary dermis but does not reach the papillary–reticular dermal interface
- Level III: Invasion fills and expands the papillary dermis but does not penetrate the reticular dermis
- Level IV: Invasion into the reticular dermis but not into the subcutaneous tissue
- Level V: Invasion through the reticular dermis into the subcutaneous tissue

Breslow's levels:

Level I: Lesions $<$0.76 mm
Level II: Lesions 0.76–1.49 mm
Level III: Lesions 1.5–3.99 mm
Level IV: Lesions $>$3.99 mm

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American Joint Committee on Cancer Staging of Melanomas of the Head and Neck

Primary Tumor (pT)

pTX: Primary tumor cannot be assessed (e.g., shave biopsy or regressed melanoma)

pT0: No evidence of primary tumor

pTis: Melanoma in situ (i.e., not an invasive tumor: anatomic level I)

pT1: Melanoma ≤1.0 mm in thickness, with or without ulceration

pT1a: Melanoma ≤1.0 mm in thickness, no ulceration, <1 mitoses/mm^2

pT1b: Melanoma ≤1.0 mm in thickness with ulceration and/or 1 or more mitoses/mm^2

pT2: Melanoma 1.01–2 mm in thickness, with or without ulceration

pT2a: Melanoma 1.01–2.0 mm in thickness, no ulceration

pT2b: Melanoma 1.01–2.0 mm in thickness, with ulceration

pT3: Melanoma 2.01–4.0 mm in thickness, with or without ulceration

pT3a: Melanoma 2.01–4.0 mm in thickness, no ulceration

pT3b: Melanoma 2.01–4.0 mm in thickness, with ulceration

pT4: Melanoma >4.0 mm in thickness, with or without ulceration

pT4a: Melanoma >4.0 mm in thickness, no ulceration

pT4b: Melanoma >4.0 mm in thickness, with ulceration

Regional Lymph Nodes (pN)

pNX: Regional lymph nodes cannot be assessed

pN0: No regional lymph node metastasis

pN1: Metastasis in 1 regional lymph node

pN1a: Clinically occult (microscopic) metastasis

pN1b: Clinically apparent (macroscopic) metastasis

pN2: Metastasis in 2 or 3 regional nodes or intralymphatic regional metastasis without nodal metastasis

pN2a: Clinically occult (microscopic) metastasis

pN2b: Clinically apparent (macroscopic) metastasis

pN2c: Satellite or in-transit metastasis without nodal metastasis

pN3: Metastasis in four or more regional lymph nodes, or matted metastatic nodes, or in-transit metastasis or satellites(s) with metastasis in regional node(s)

Number of lymph nodes identified:

Number containing metastases identified macroscopically:

Number containing metastases identified microscopically:

Matted nodes:

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Distant Metastasis (pM)

pM1: Distant metastasis (documented in this specimen)
pM1a: Metastasis in skin, subcutaneous tissues, or distant lymph nodes
pM1b: Metastasis to lung
pM1c: Metastasis to all other visceral sites or distant metastasis at any site associated with an elevated serum LDH
Specify site, if known:

Clinical Stage Groupings

Stage 0

Tis N0 M0

Stage IA

T1a N0 M0

Stage IB

T1b N0 M0
T2a N0 M0

Stage IIA

T2b N0 M0
T3a N0 M0

Stage IIB

T3b N0 M0
T4a N0 M0

Stage IIC

T4b N0 M0

Stage III

Any T any N>N0 M0

Stage IV

Any T any N M1

◆ Staging of Mucosal Melanoma

Mucosal Malignant Melanoma

Primary Tumor

T3: Mucosal disease
T4a: Moderately advanced disease. Tumor involving deep soft tissue, cartilage, bone, or overlying skin.

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T4b: Very advanced disease. Tumor involving brain, dura, skull base, lower CNs (IX, X, XI, XII), masticator space, carotid artery, prevertebral space, or mediastinal structures.

Regional Lymph Nodes

NX: Regional lymph nodes cannot be assessed

N0: No regional lymph node metastases

N1: Regional lymph node metastases present

Distant Metastasis

M0: No distant metastasis

M1: Distant metastasis

American Joint Committee on Cancer Staging of Mucosal Malignant Melanoma

Stage III

T3 N0 M0

Stage IVA

T4a N0 M0
T3-T4a N1 M0

Stage IVB

T4b any N M0

Stage IVC

Any T any N M1

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5.2.13 Malignant Neoplasms of the Ear and Temporal Bone

◆ Key Features

- SCC is the most common malignancy of the middle ear and temporal bone, accounting for 60 to 80% of these lesions.
- BCC may be slightly more prevalent than SCC on the auricle but accounts for ~20% of neoplasms of the middle ear and temporal bone.
- A host of other malignant neoplasms can affect the ear and temporal bone, including melanoma, sarcomas, hematologic malignancies, and metastatic lesions.

SCC is the most common malignancy affecting the external auditory canal (EAC). Patients may present with painless bloody otorrhea. Physical examination may reveal the appearance of granulation tissue, and a biopsy is necessary to differentiate from chronic otitis.

◆ Epidemiology

For cutaneous malignancies of the auricle, controversy exists regarding whether SCC or BCC is more common. The main causative factor is sun exposure; median age is ~70 years. Melanoma is less common.

The incidence of temporal bone cancer is ~6 per million, with no clear gender predilection. Chronic suppurative otitis media (CSOM) may be a precursor, but no definitive correlation has ever been proved.

◆ Clinical

Signs

Swelling of the ear canal with or without aural polyps may be a sign of a malignant neoplasm of the ear. Facial nerve weakness or other CN palsies may be identified. Hearing loss will most likely be conductive, but an aggressive lesion with otic capsule invasion can present with sensorineural hearing loss and vertigo.

Symptoms

Tumors of the ear canal mimic otitis externa with drainage and discomfort. Tumors of the temporal bone or middle ear mimic otitis media with painless drainage but may be bloody. Patients may present with hearing loss (conductive or sensorineural) and facial palsy.

Differential Diagnosis

These must be differentiated from an infectious lesion with a biopsy. Although this chapter deals primarily with SCC, there is a long list of

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other malignancies that can affect the temporal bone, including BCC, adenocarcinoma, sarcoma, melanoma, lymphoma, and metastatic tumors. Benign lesions like ear canal osteomas, paragangliomas, schwannomas, meningiomas, hemangiomas, endolymphatic sac tumors, and eosinophilic granulomas also need to be ruled out.

◆ Evaluation

Physical Exam

A full head and neck exam is necessary. Begin with a thorough skin and scalp evaluation looking for other skin lesions. Any skin abnormality on the auricle or in the canal requires a biopsy. The parotid gland and neck are carefully palpated. Complete CN exam is documented as well. Evaluation of temporomandibular joint function is important to exclude gross involvement or tumor invasion.

Imaging

CT of the temporal bones without contrast is the study of choice for temporal bone involvement. Degree of erosion of the ear canal is noted, as is involvement of the middle ear or any deeper structures. A neck CT with contrast evaluates the parotid gland and the cervical lymph nodes. The parotid gland may be involved by both direct extension and intraparotid nodal metastasis. MRI of the brain is helpful in evaluating possible dural extension or intracranial metastatic lesions. Arteriography with balloon occlusion may be needed to evaluate suspected intrapetrous carotid artery invasion. PET scanning may be helpful in evaluating nodal disease or distant metastases.

Labs

Biopsy is necessary. An audiogram will be helpful in counseling patients regarding hearing rehabilitation after treatment.

Pathology

Same as SCC elsewhere (see Chapter 5.2.12.2). There are many different subtypes: well differentiated, moderately differentiated, poorly differentiated, clear cell, spindle cell, and verrucous.

◆ Other Malignant Neoplasms of the Ear and Temporal Bone

Melanoma

Melanoma of the auricle accounts for almost 1% of all melanomas. T stage is based on Breslow level. Surgical excision with 1- to 2-cm margin and neck dissection or sentinal node assessment for tumors 1 mm or thicker, or greater than Clark's level IV, is indicated.

Malignant Glandular Tumors

Adenoid cystic carcinoma and ceruminous adenocarcinoma present as painful obstructing ear canal masses. They are treated surgically like SCC

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with postoperative radiation. Lateral parotidectomy is also indicated, along with postoperative radiation.

Chondrosarcoma

Chondrosarcomas occur at the skull base, off midline at the petroclival junction. There are five subtypes and three grades; conventional is the most common subtype, and most tumors are grade I or II. Chondrosarcomas most commonly present with headache, diplopia, or hearing loss plus CN deficits, and most arise during the fourth or fifth decade of life. CT shows bony destruction and calcification; MRI reveals bright T2 and enhancement with contrast. Surgical excision by any number of approaches is necessary.

Chordoma

Chordoma is a locally aggressive disease process with a low rate of metastasis. It originates from notochord remnant. There are three subtypes. It is usually found midline at the clivus. Patients most commonly present with headache or diplopia and usually are 40 to 50 years of age. Over 90% of patients die of disease, but <10% have metastasis. CT shows a midline erosive mass that may enhance with contrast, and MRI images are bright on T2 and enhance with contrast. Multiple midline and lateral approaches are available.

Sarcoma

Sarcoma is the most common malignancy of temporal bone in children, especially rhabdomyosarcoma. There are multiple types, and it usually presents with otitis media, drainage, polyps, and bleeding. Most cases respond to either chemotherapy or radiotherapy, so surgery is limited to diagnostic biopsy with aggressive resection reserved for treatment failures.

Metastasis

Metastasis is most commonly hematogenous from breast, lung, and kidney. It may also spread via cerebrospinal fluid or leptomeningeal spread. Patients present with hearing loss, facial paralysis, and headache. Survival is usually less than a year.

◆ Treatment Options

Treatment is based on T stage. The modified University of Pittsburgh staging system is used:

- T1: Tumor limited to EAC without bony erosion
- T2: Tumor limited to EAC with bony erosion less than full thickness, and <5 mm of soft tissue involvement
- T3: Tumor with full-thickness erosion of the EAC with <5 mm of soft tissue involvement or middle ear or mastoid involvement or facial palsy
- T4: Tumor eroding the cochlea, petrous apex, carotid canal, jugular foramen, dura, medial wall of middle ear, or >5 mm of soft tissue involvement

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Medical

Chemotherapy may have some role in treatment as an adjunct to surgery, usually given postoperatively and in palliative management. Radiotherapy is recommended postoperatively for all lesions T2 or greater.

Surgical

The surgical approach depends on site and stage of lesion. There are many common surgical approaches beginning with sleeve resection of the ear canal with some form of mastoidectomy for T1 lesions isolated to the ear canal.

Lesions within the canal, but not extending into the middle ear, are approached with a lateral temporal bone resection that removes the ear canal en bloc. The medial margin of resection is the middle ear and the facial nerve canal.

Lesions that extend into the middle ear can be resected with subtotal temporal bone resection, which is a lateral temporal bone resection and piecemeal removal of any residual deeper tumor remnants. Total temporal bone resection is reserved for deeply invasive T4 lesions and its utility is controversial.

Neck dissection and parotidectomy are often part of the resection; certainly if there is clinically evident nodal spread. Pedicled flap or free flap may be required for reconstruction.

Melanoma is discussed elsewhere (see Chapter 5.2.12.3), and requires wider margins, and sentinal node assessment except for early-stage lesions.

◆ Outcome and Follow-Up

Accurate outcomes and survival rates are difficult to find in the literature. T1 tumors treated with surgery probably have a 5-year survival rate >90%. T2 tumors treated with surgery and radiation have 5-year survivals of ~90%. T3 and T4 tumors treated with surgery and radiation have 5-year survival rates <50%.

◆ ICD-9 Codes

170.0 Malignant neoplasm of bone and articular cartilage of skull and face
172.2 Malignant melanoma of skin of ear and external auditory canal
173.2 Other malignant neoplasm of skin of ear and external auditory canal

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5.2.14 Lymphomas of the Head and Neck

◆ Key Features

- The basic categories are Hodgkin lymphoma (HL) and non-Hodgkin lymphoma (NHL).
- NHL occurs more than five times as frequently as HL.
- NHL is the second most common malignancy in HIV-positive patients.
- Lymphomas are the most common nonepithelial tumors of the head and neck.
- The extranodal sites in the head and neck are the thyroid, orbit, salivary glands, and sinonasal passages.

The otolaryngologist–head and neck surgeon usually serves a well-defined, important role in the management of lymphomas. A high index of suspicion for lymphoma as a cause of common complaints in the head and neck region can lead to early diagnosis and improved outcome. Beyond the role of diagnostician, the head and neck surgeon will often be the one who obtains tissue for diagnosis. NHL usually originates in lymphoid tissues and can spread to other organs; it is much less predictable than HL and has a far greater predilection to disseminate to extranodal sites. The prognosis depends on the histologic type, stage, and treatment. For staging of HL and NHL, see **Table 5.9**.

◆ Epidemiology of Non-Hodgkin Lymphoma

In 2008, there was 66,120 estimated new cases of NHL and 19,160 deaths from NHL in the United States.

◆ Epidemiology of Hodgkin Lymphoma

In 2008, there were 8220 estimated new cases and 1350 deaths from HL in the United States. Over 75% of all newly diagnosed patients with adult HL can be cured with combination chemotherapy and/or radiotherapy.

The median patient age is in the low to mid-20s, with half of the patients between 15 and 30 years of age; HL is rare in patients younger than age 10 and after age 60. The age distribution is bimodal with a peak in the low 20s and a second peak in the low 40s.

◆ Clinical

HL often presents as an asymptomatic neck mass. It arises almost exclusively in nodal tissue; it manifests as enlarged rubbery painless nodes in the low neck and/or supraclavicular fossa, or above the hyoid in the submental,

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Table 5.9 American Joint Committee on Cancer/Ann Arbor Staging System for Hodgkin and Non-Hodgkin Lymphoma

Stage I	Involvement of a single lymph node region (I); or localized involvement of a single extralymphatic organ or site in the absence of any lymph node involvement (IE) (rare in Hodgkin lymphoma)
Stage II	Involvement of two or more lymph node regions on the same side of the diaphragm (II); or localized involvement of a single extralymphatic organ or site in association with regional lymph node involvement with or without involvement of other lymph node regions on the same side of the diaphragm (IIE); the number of regions involved may be indicated by a subscript, as in, for example, II_3
Stage III	Involvement of lymph node regions on both sides of the diaphragm (III), which also may be accompanied by extralymphatic extension in association with adjacent lymph node involvement (IIIE), by involvement of the spleen (IIIS), or both (IIIE,S)
Stage IV	Diffuse or disseminated involvement of one or more extralymphatic organs, with or without associated lymph node involvement; or isolated extralymphatic organ involvement in the absence of adjacent regional lymph node involvement, but in conjunction with disease in distant site(s); any involvement of the liver or bone marrow, or nodular involvement of the lung(s)
A and B classification (symptoms). Each stage should be classified as either A or B according to the absence or presence of defined constitutional symptoms.	1. *Fevers:* Unexplained fever with temperature above 38°C 2. *Night sweats:* Drenching sweats that require change of bedclothes 3. *Weight loss:* Unexplained weight loss >10% of the usual body weight in the 6 months before diagnosis

Source: Data from the 2010 American Joint Committee on Cancer: AJCC Cancer Staging Handbook. 7th ed. New York: Springer-Verlag; 2010.

submandibular, periauricular, or periparotid nodes. Disease spreads in an orderly fashion to contiguous nodal regions.

For diagnostic criteria of NHL, see **Table 5.10**.

◆ Evaluation

History

A detailed history, with special attention to the constitutional (B) symptoms of temperature higher than 38°C, night sweats, and weight loss of >10% of total body weight over 6 months. Pruritus as a systemic symptom remains controversial and is not considered a B symptom. A history relating to lymphadenopathy or extranodal disease involvement, such as unilateral tonsillar hypertrophy or nasal obstruction, is a concern.

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Table 5.10 Diagnostic Criteria for Common Lymphomas of the Head and Neck

Histologic Subtype	Cell Type	Additional CD Markers	Associated Oncogene	Translocation (Incidence in Subtype)	Cellular Activity	Typical Head and Neck Site	Typical Behavior
Small lymphocytic lymphoma	B	CD5+, CD10−, CD23+	ND	ND	ND	Anterior orbit, nodes	Indolent
Follicular	B	CD5−, CD43−, CD10+, CD23±	BCL-2	t(14;18) (q32;q11) 90%	Suppression of apoptosis	Nodes, Waldeyer's ring, salivary glands	Indolent
Diffuse large cell	B	CD5−, CD10+, CD23±, CD43−, CD30−	BCL-2	t(14;18) (q32;q11) 20%	Suppression of apoptosis	Waldeyer's ring, nodes, salivary glands, thyroid, orbit, sinonasal tract	Aggressive
			BCL-6	3q27 40%	Zinc finger transcription factor		
			BCL-8	t(14;15) (q32;q11–13) 3–4%	Transcription factor		
MALT	B	CD5−, CD10−, CD23	ND	t(11;18) (q21;q21) 35%	ND	Salivary glands, orbit, thyroid, Waldeyer's ring	Indolent

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Marginal zone (monocytoid)	B		ND	ND	ND	Nodes, neurotropic presentation	Indolent
Mantle	B	CD5+, CD10±, CD23−, CD43+	BCL-1 (cyclin-D)	t(11;14) (q13;q32) 70%	G1/S progression	Waldeyer's ring, nodes	Aggressive
Burkitt	B		C-MYC	8q24 100%	Transcription activation; cell cycle control	Waldeyer's ring, parapharyngeal site	Aggressive
Primary system C or primary cutaneous anaplastic large cell	T	CD30+	ALK/NPM	t(2;5) (p23;q35) ND	Tyrosine kinase	Skin	Variable
Angiocentric lymphoma (AKA) lethal midline granuloma, nasal T-cell lymphoma, nasal (T-NK cell) lymphoma	T	CD56+, CD4+	ND	ND	ND	Sinonasal tract	Aggressive

Source: Modified from Tsang RW, Gospodarowicz MK. Non-Hodgkin's lymphoma. In: Gunderson LL, Tepper JE, eds. Clinical Radiation Oncology. Philadelphia, PA: Churchill Livingstone; 2000:1158–1188.
Abbreviation: ND, no data.

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Physical Exam

A complete head and neck physical examination should be performed, focusing on identifying and documenting the extent of lymphadenopathy, Waldeyer's ring, or skin involvement.

Imaging

Knowledge of the anatomic extent of disease is required for treatment planning. Clinical staging for patients with HL includes a history, physical examination, laboratory studies (including erythrocyte sedimentation rate [ESR]), and thoracic and abdominal/pelvic CT scans. PET scans, sometimes combined with CT scans, have replaced gallium scans and lymphangiography for clinical staging.

Labs

A complete blood cell count with differential, ESR, serum protein electrophoresis, chemistries, and liver function tests should be obtained.

Other Tests

FNAB can be sufficient to confirm recurrence in an already well-characterized lymphoma and will occasionally be sufficient to secure initial diagnosis, but excisional biopsy is generally preferred to afford assessment of nodal or extranodal tissue architecture. Core needle biopsy is an alternative if excisional biopsy is not possible.

Bone marrow involvement occurs in 5% of patients; biopsy is indicated in the presence of constitutional B symptoms or anemia, leukopenia, or thrombocytopenia.

Pathology

Pathologists currently use the WHO modification of the Revised European-American Lymphoma (REAL) classification for the histologic classification for adult HL, see **Table 5.11**.

◆ Treatment Options

Medical

Hodgkin Lymphoma

Patients with nonbulky stage IA or IIA disease are considered to have clinical early-stage disease. These patients are candidates for chemotherapy, combined modality therapy, or radiotherapy alone. Patients with stage III or IV disease, bulky disease, or the presence of B symptoms will require combination chemotherapy with or without additional radiotherapy.

Radiotherapy can be helpful. In adult HL, the appropriate dose of radiation alone is 25 to 30 Gy to clinically uninvolved sites, and 35 to 44 Gy to regions of initial nodal involvement.

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Table 5.11 World Health Organization/Revised European-American Lymphoma (WHO/REAL) Classification for Adult Hodgkin Lymphoma

Classical Hodgkin lymphoma
Nodular sclerosis Hodgkin lymphoma
Mixed-cellularity Hodgkin lymphoma
Lymphocyte depletion Hodgkin lymphoma
Lymphocyte-rich classical Hodgkin lymphoma
Nodular lymphocyte–predominant Hodgkin lymphoma

Non-Hodgkin Lymphoma

For indolent lymphomas, no initial therapy is recommended while asymptomatic. At the onset of symptoms or complications, a single-agent oral alkylator therapy may induce and maintain clinical remission. Relapses are common.

Aggressive lymphomas are treated with a goal of cure. Localized stage I disease may be treated with radiation alone. For bulkier or clinically staged localized disease, chemotherapy is the mainstay of treatment. An intensive but abbreviated chemotherapy course followed by consolidation radiotherapy has, until recently, been the treatment of choice. Emerging experience suggests that CHOP (cyclophosphamide, doxorubicin, vincristine, and prednisone) plus rituximab (anti-CD20 monoclonal antibody) may be the most efficacious protocol in elderly patients. Disseminated aggressive disease is best treated with full-intensity, full-course chemotherapy.

Surgical

In both NHL and HL, surgery is typically performed to establish diagnosis. It consists of an excisional biopsy of enlarged or suspicious lymph nodes. Occasionally, palatine, lingual or nasopharyngeal tonsil tissue is excised and submitted. The specimen should be adequately sampled, handled, and fixed. Where possible, care should be taken to provide intact samples large enough to provide meaningful nodal architecture information on which to base pathologic diagnosis. All permanent specimens should be submitted dry or in saline but not in formalin to allow immunohistochemical and flow cytometric studies to complement traditional pathology.

◆ Outcome and Follow-Up

The most important prognostic factors in HL are subtype and stage. Lymphocyte-predominant disease has an excellent prognosis; the prognosis of the nodular sclerosis variant is also very good. Lymphocyte-depleted classic HL has the worst prognosis. Other poor prognostic factors include constitutional symptoms, age >45 years, bulky mediastinal disease, extranodal

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disease, elevation of LDH or ESR, and more than five disease foci in the spleen in pathologically staged patients.

Because HL is often curable and frequently occurs in younger populations, consideration of immediate and delayed complications of therapy is important. The most worrisome of these complications are secondary malignancies, which occur with an overall incidence of 5%. Complications of radiotherapy include those specific to head and neck irradiation, such as xerostomia and mucositis, as well as generalized fatigue and weight loss.

◆ ICD-9 Codes

200–208	Malignant neoplasm of lymphatic and hematopoietic tissue
200	Lymphosarcoma and reticulosarcoma
201	Hodgkin disease
202	Other malignant neoplasms of lymphoid and histiocytic tissue

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5.2.15 Idiopathic Midline Destructive Disease

◆ Key Features

- Idiopathic midline destructive disease is a progressive destructive lesion of the midface and upper airway region.
- Sinonasal natural killer cell or T-cell (NK/T-cell) lymphomas are associated with the disease.
- Non-Hodgkin lymphoma of the midface region is one of the rarest forms of extranodal lymphoma representing $<0.5\%$ of cases.
- The incidence of these tumors is substantially higher among Asian and South and Central American populations.

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Idiopathic midline destructive disease (IMDD), also known as lethal midline granuloma or extranodal NK/T-cell lymphoma, nasal type, is a rare disorder characterized by ulceration and necrosis of the midline facial tissues and obstruction of the upper airway region.

◆ Epidemiology

Idiopathic midline destructive disease affects a wide age range, peaking in the sixth decade. Males are predominately affected, and the disease is much more common in persons resident in East Asia (e.g., Japan, Taiwan, China, Korea, Thailand, and Hong Kong). In Asian groups, >90% of cases have T-cell markers and EBV has been consistently demonstrated in the cell genome.

◆ Clinical

Signs and Symptoms

Initial symptoms are usually those of a nonspecific rhinitis or sinusitis with nasal obstruction and nasal discharge. Epistaxis and facial swelling may occur. As the disease progresses, ulcerations spread, destroying the soft tissues, cartilage and bone. Subsequently, facial pain and facial deformities develop. Signs may include CN palsies, diplopia, and proptosis due to intraorbital or skull base extension. Death after a long duration may be the result of cachexia, hemorrhage, meningitis, or intercurrent infection.

Differential Diagnosis

- Wegener granulomatosis
- Malignant lymphoma
- Churg-Strauss syndrome
- Polyangiitis
- Drug abuse

◆ Evaluation

Physical Exam

A complete head and neck exam including a nasal endoscopy should be performed. The most common finding is the presence of a nasal septal perforation.

Imaging

No specific imaging findings are present. The main role of imaging is to evaluate the extent of the disease, monitor its progression over time, and ascertain the effect of treatment. CT with high-resolution bone algorithms is the best method to evaluate bone changes, whereas MRI should be used to determine the extent of soft tissue, orbital, and intracranial involvement.

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Pathology

Because the abnormal tissue is largely necrotic, multiple biopsy specimens are often required before a diagnosis is made. IMDD seems to be closely associated with EBV. This is unusual as typically this virus is associated with B-cell lymphomas.

◆ Treatment Options

Left untreated, IMDD is uniformly fatal.

Medical

Treatment for sinonasal NK/T-cell lymphomas consists of the combination of an anthracycline-based chemotherapy regimen (e.g., CHOP – cyclophosphamide, doxorubicin hydrochloride, vincristine sulfate, and prednisone) along with locoregional radiotherapy. Generally, the initial response to radiotherapy is so rapid and dramatic that the use of involved-field radiotherapy has been accepted as the preferred treatment option for localized disease. Overall, ~20 to 30% of patients treated with radiotherapy alone experience systemic failure in extranodal sites and local recurrence rates range from 31 to 67%.

Surgical

Biopsy for diagnosis and débridement of necrotic tissue are the only surgical interventions.

◆ Outcome and Follow-Up

Patients with IMDD have poor survival outcomes, with the cumulative probability of survival at 5 years ranging from 37.9 to 45.3%.

◆ ICD-9 Code

446.3 Idiopathic midline destructive disease (aka lethal midline granuloma)

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5.2.16 Paragangliomas of the Head and Neck

◆ Key Features

- Paragangliomas of the head and neck are highly vascular tumors.
- Other terms used for tumors of the same histology are carotid body tumor, chemodectoma, and glomus tumor.

Paragangliomas are neuroendocrine tumors that develop from paraganglia, small organs of the autonomic nervous system. Paragangliomas may develop at four sites within the head and neck: the carotid body (carotid body tumors), the ganglia of the vagus nerve (glomus vagale tumors), the jugular bulb (glomus jugulare tumors), and the middle ear (glomus tympanicum tumors). Treatment of head and neck paragangliomas is primarily surgical, although radiation and observation may be appropriate under certain circumstances.

◆ Epidemiology

Paragangliomas are rare tumors, accounting for <1% of all head and neck tumors. In most cases, these are solitary lesions; bilateral or multicentric lesions occur in 3% of cases. Ten to 15% of patients have familial paragangliomas; patients with the familial tumors have much higher rates of bilaterality and multicentricity. The carotid body and jugular bulb are the most common sites of head and neck paragangliomas. Rare sites of involvement are the larynx, nasal cavity, paranasal sinuses, and thyroid gland.

◆ Clinical

Signs and Symptoms

Presentation of paragangliomas relates to the location at which the tumor arises. Carotid body and vagal paragangliomas present as slow-growing, nontender neck masses. A typical lateral neck mass may be seen, accompanied in some cases by the oropharyngeal bulge suggestive of parapharyngeal space involvement. More advanced or neglected lesions may have associated ipsilateral cranial neuropathies.

Differential Diagnosis

Initially, all etiologies of a lateral neck mass should be considered. Radiographic or clinical localization to the parapharyngeal space focuses the investigation; the most common parapharyngeal space lesions are salivary gland tumors (most common), paragangliomas, and nerve sheath tumors (schwannomas and neurofibromas).

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◆ Evaluation

Physical Exam

A complete head and neck exam with attention to CN function is appropriate. The neck mass associated with a paraganglioma is frequently pulsatile, owing to its intimate relationship to the carotid vessels. Additional neck masses, suggesting lymph node metastasis or multifocality, should also be sought.

Imaging

CT scanning with contrast is useful, demonstrating the hypervascular tumor and its relationship with the neck vessels. MRI provides similar information with the added benefits of no radiation exposure, greater soft tissue differentiation, and three planes of view. Formal angiography has been replaced by MRI, magnetic resonance angiography (MRA), or CT in the diagnostic realm, but it is still required when embolization is needed preoperatively.

Labs

A history of signs and symptoms of excess catecholamines should be explicitly sought. If symproms such as flushing, labile or difficult to control hypertension, or excessive perspiration are described, preoperative testing for serum and urine catecholamines (and their metabolites) should be ordered. Although "functional" (catecholamine-secreting) paragangliomas of the head and neck are unusual (1–3%), pheochromocytomas of the adrenal medulla, which may constitute a portion of a syndrome of which the head and neck paraganglioma is the presenting feature, are much more frequently metabolically active.

Pathology

Grossly, paragangliomas are polypoid, well circumscribed, and firm to rubbery in consistency. Paragangliomas have a characteristic histologic appearance, with tumor cells arranged in clusters called "zellballen," which are separated by a fibrovascular stroma. Roughly 10% of paragangliomas are malignant. Malignancy is determined solely on the basis of metastasis, rather than on any histologic features of the primary tumor.

◆ Treatment Options

The major goal of treatment is prevention of morbidity due to progressive cranial neuropathies. In most cases, this goal is best achieved by surgical extirpation via a cervical incision. Many surgeons employ preoperative embolization to decrease blood loss and improve visualization with larger tumors. The need for internal carotid artery sacrifice and revascularization with saphenous vein grafting should always be considered, especially in larger tumors and tumors that radiographically encapsulate the internal carotid artery.

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On the other hand, surgery itself may result in new cranial neuropathies and significant associated morbidity. This merits consideration of other management strategies in selected cases. Other options include close observation with serial imaging and external beam radiation. Notably, external beam radiation appears to be tumorostatic rather than tumoricidal with paragangliomas.

In the very elderly or infirm patient, and in those with multiple (especially bilateral) tumors, all options are considered carefully. In the fragile patient, surgery may be postponed unless significant growth is noted radiographically or worsening of cranial neuropathies is identified clinically. In patients with bilateral disease, treatment, which may include any combination of surgery, observation, and external beam radiation, is tailored to achieve a goal of CN preservation on at least one side, as intractable swallowing dysfunction and/or tracheotomy dependence are the inevitable consequence of bilateral CN X or XII impairment.

◆ Outcome and Follow-Up

Patients should be closely observed for any local recurrence, although these are usually rare. Contralateral tumors should be sought and resected.

◆ ICD-9 Codes

194.5 Malignant neoplasm of carotid body
194.6 Malignant neoplasm of aortic body and other paraganglia
237.3 Neoplasm of uncertain behavior of paraganglia

Further Reading

Day TA, Joe JK. Primary neoplasms of the neck. In Cummings CW, Haughey BH, Thomas JR, et al, eds. Otolaryngology–Head and Neck Surgery. 4th ed. Philadelphia, PA: Elsevier Mosby; 2005:2554
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5.2.17 Peripheral Nerve Sheath Tumors

◆ Key Features

- Peripheral nerve sheath tumors (PNSTs) include schwannomas and neurofibromas.
- Schwannomas account for 6 to 8% of intracranial neoplasms.
- PNSTs are the most common tumors of the poststyloid parapharyngeal space.

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- Vestibular schwannomas (see Chapter 2.8) are the most common CN schwannomas, followed by trigeminal and facial schwannomas, and then glossopharyngeal, vagus, and spinal accessory nerve.
- Benign PNSTs include neurilemoma (schwannoma), neurofibroma, and ganglioneuroma.
- Malignant PNSTs include malignant neurofibrosarcoma, schwannosarcoma, and sympathicoblastoma.

PNSTs are divided into two major groups: schwannomas (neurilemomas) and neurofibromas. Schwannomas are slow-growing, usually benign tumors that may arise from any nerve that is ensheathed in Schwann cells. In the parapharyngeal space, this includes CNs V3, IX, X, XI, and XII; the sympathetic trunk; and the upper cervical nerves. In the parapharyngeal space, schwannomas often present as neck masses.

Neurofibromas, in contrast, are unencapsulated and intimately involved with the nerve of origin. Neurofibromas are often multiple.

◆ Epidemiology

About 30% of all schwannomas arise in the head and neck. They are the most common benign neurogenic neoplasm of the parapharyngeal space. In this setting most arise from the vagus nerve or the sympathetic trunk. Neurofibromas may occur as a manifestation of neurofibromatosis type 1 (NF-1; formerly known as von Recklinghausen disease); in these patients, the incidence of malignant transformation is increased. Malignant PNSTs constitute ~10% of all soft tissue sarcomas and are more often found in the setting of neurofibromatosis. The most common head and neck area of involvement of malignant PNSTs is the neck, though it may arise from the tongue or soft palate.

◆ Clinical

Signs and Symptoms

Benign schwannomas of the parapharyngeal space most frequently present as neck masses. Other features of PNSTs at presentation may include an oropharyngeal bulge and ipsilateral cranial neuropathies.

Differential Diagnosis

- Paraganglioma
- Deep lobe parotid tumor
- Parapharyngeal space salivary gland
- Lymphoma
- Metastatic disease

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◆ Evaluation

History

A history should include a family history of neurofibromatosis and other syndromes.

Physical Exam

A complete head and neck examination, including examination of CN function, is appropriate. Cranial neuropathies may or may not correlate with the nerve of origin of the tumor; that is, in some cases, adjacent nerves may develop palsies due to compression, while the nerve of origin continues to function normally. Other than the mass, many patients will be asymptomatic.

Imaging

MRI is the imaging modality of choice for schwannomas and is frequently diagnostic. Schwannomas have low signal intensity on T1-weighted images and high signal intensity on T2-weighted images. Administration of gadolinium causes intense enhancement of schwannomas.

Pathology

Benign schwannomas arise from the nerve sheath and consist of Schwann cells in a collagenous matrix. Histologically, schwannomas are characterized by regions of tightly packed spindle cells: Antoni type A neurilemoma and type B neurilemoma are used to describe varying growth patterns in schwannomas. Type A tissue has elongated spindle cells arranged in irregular streams and is compact in nature. Type B tissue has a looser organization, often with cystic spaces intermixed within the tissue.

Malignant PNSTs may be classified into three major categories, with epithelioid, mesenchymal, or glandular characteristics.

◆ Treatment Options

The surgical approach chosen depends on the location and extent of the lesion within the parapharyngeal space, the presence or absence of involvement of adjacent spaces, and the preferences of the surgeon. Options include cervical, cervical–parotid, cervical–parotid with mandibulotomy, and submandibular approaches. Differentiation of schwannomas from neurofibromas is of relevance to surgeons because schwannomas can be easily shelled out while preserving nerve contiguity. In most neurofibromas, however, the nerve is incorporated within the mass, and the required surgery includes resection and subsequent nerve grafting to preserve and restore function. In the case of schwannomas, this type of relationship between tumor and nerve presents the possibility of enucleation (removal of the tumor with preservation of the nerve from which it arose). Although this type of removal is often practically achievable, and recurrence rates

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are low, some surgeons are dubious about the likelihood of preservation of nerve function.

Malignant PNSTs should be treated by wide surgical excision, but local recurrence is a common occurrence and hematogenous metastasis occurs in at least half of treated cases. The tumor is resistant to radiotherapy and chemotherapy, and those occurring in neurofibromatosis type 1 behave in a more aggressive fashion than those not associated with the syndrome. Overall, the 5-year survival rate for malignant PNSTs is 40 to 75%.

◆ ICD-9 Codes

237.7	Neurofibromatosis
237.70	Neurofibromatosis unspecified
237.71	Neurofibromatosis type 1
237.9	Neoplasm of uncertain behavior of other and unspecified parts of nervous system
210.x	Schwannoma (benign neoplasm, by site)

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5.3 The Salivary Glands

5.3.1 Salivary Gland Disease

◆ Key Features

- Salivary gland disease may be infectious, inflammatory, noninflammatory, or autoimmune in nature.
- Salivary gland disease may be accompanied by a decrease in salivary flow.
- An underlying autoimmune condition and tumor must be ruled out.

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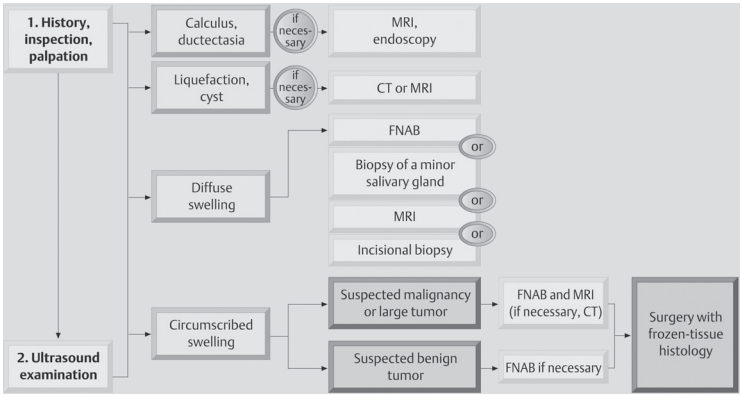

Fig. 5.9 Flowchart for the investigation of salivary gland disease. MRI, magnetic resonance imaging, CT, computed tomography; FNAB, fine-needle aspiration biopsy. (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:141.)

Salivary gland disease may be infectious, inflammatory, noninflammatory, or autoimmune in nature.

◆ Infectious Diseases of the Salivary Glands

Acute Sialadenitis

Acute sialadenitis is an acute inflammation of a salivary gland. Several viral microorganisms have been identified as causative agents for infections of the salivary glands. The most common virus that causes sialadenitis is the mumps virus (rubulavirus). Others include coxsackievirus, echovirus, influenza virus, and HIV.

Signs and Symptoms

Symptoms of acute sialadenitis include acute painful swelling of the affected salivary gland (**Fig. 5.9**). At times only one gland is affected initially. There may be swelling of adjacent cervical lymph nodes. The duct orifice may be erythematous. Other symptoms include fever and trismus.

Differential Diagnosis

- Cervical lymph adenopathy
- Acute bacterial sialadenitis
- Inflammatory sialadenitis
- Abscess
- Tumor
- Odontogenic abscess
- Infection

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Treatment Options

Treatment is supportive; it typically consists of fluid intake, sialagogues (such as lemon wedges), and analgesics.

Acute Bacterial Sialadenitis

Acute bacterial sialadenitis is a suppurative infection that most commonly infects the parotid glands in debilitated, dehydrated, or elderly patients. Acute bacterial sialadenitis is thought to be an ascending bacterial infection due to decreased salivary flow. Situations that cause a tendency for acute sialadenitis include diabetes mellitus, immunocompromised state, and poor oral hygiene. Other rarer infectious causes of sialadenitis include tuberculosis, actinomycosis, syphilis, and HIV infection.

Signs and Symptoms

Symptoms include diffuse, painful swelling of the affected gland (**Fig. 5.10**). The skin over the gland may be warm, red, and tight. The orifice duct of the affected salivary gland may be red and massage of the gland may express purulent material from the orifice. Trismus may be present.

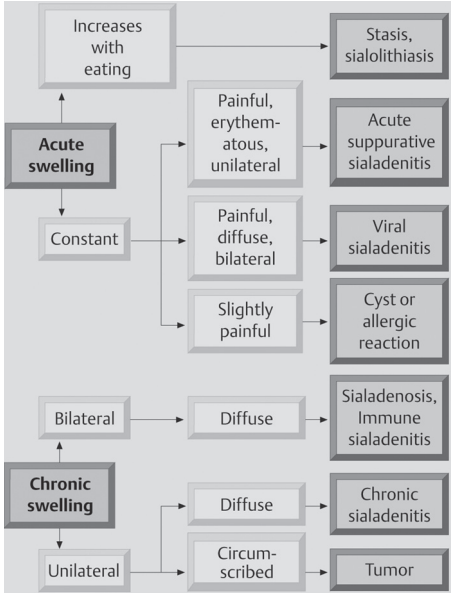

Fig. 5.10 Differential diagnosis of acute and chronic salivary gland swelling. (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:140.)

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Microbiology

The main causative pathogen is *Staphylococcus aureus*. Other bacterial organisms include *Streptococcus viridans*, *Haemophilus influenzae*, *Streptococcus pyogenes*, and *Escherichia coli*.

Treatment Options

Medical treatment includes antibiotics, hydration, sialogogues (such as lemon wedges), warm compresses, gland massage, and meticulous oral hygiene. If an abscess develops, incision and drainage should be performed with careful attention to the underlying facial nerve. The incision should be made in parallel to the branches of the facial nerve.

◆ Inflammatory Diseases of the Salivary Glands

Sjögren Syndrome

Sjögren syndrome is an inflammatory disease of the exocrine glands. This autoimmune condition may cause chronic sialadenitis. Patients may develop a gradual decrease in salivary production. Xerostomia (dry mouth), xerophthalmia (dry eyes), and lymphocytic infiltration of the exocrine glands is known as the sicca complex. The pathogenesis is antibodies directed against the antigens of the salivary duct epithelium causing gland atrophy and interstitial lymphocystic infiltration.

Epidemiology

The disease predominantly affects women.

Signs and Symptoms

Both parotid glands may be involved. Their consistency on palpation is doughy. There may be little or no pain or tenderness. Patients may have associated xerostomia, keratoconjunctivitis, lymphadenopathy, and polyneuropathies.

Labs

An elevated ESR and the presence of autoantibodies Sjögren syndrome A (SS-A) and Sjögren syndrome B (SS-B), rheumatoid factor, and antinuclear antibodies are indicators for the disease.

Other Tests

Biopsy of the minor salivary glands of the lip is diagnostic in 60 to 70% of the cases. Sialography, if performed, may demonstrate a leafless tree pattern.

Treatment Options

Treatment includes immunosuppressant therapy as well as symptomatic therapy with saliva substitutes and artificial tears as well as pilocarpine to augment salivatory stimulation.

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Complications

Complications of Sjögren syndrome involving the salivary glands include dental caries. There is an increase incidence of NHLs of the salivary glands among patients with Sjögren syndrome.

Heerfordt Syndrome

Other causes of chronic sialadenitis include Heerfordt syndrome, a form of sarcoidosis with enlargement of the parotid glands, mild fever, uveitis, and facial nerve palsy.

◆ Noninflammatory Salivary Disease

Sialolithiasis

Sialolithiasis (salivary stones or calculi) is a stone formation in the excretory duct system of the salivary glands. Sixty to 70% of these are located in the main duct of the gland; 70 to 80% occur in the submandibular gland; 20% occur in the parotid gland. A much smaller percentage may occur in minor salivary glands. Pathogenesis is ductal debris, and calcium phosphate coalesce due to infection, inflammation, or salivary stasis.

Epidemiology

Sialolithiasis typically affects adults, males more commonly affected than females.

Signs and Symptoms

Symptoms vary according to the degree of obstruction and may include pain and swelling often while or after eating.

Differential Diagnosis

- Inflammation
- Infection
- Tumor

Imaging

Ultrasound may reveal the dilation of the duct system. Seventy to 80% of submandibular salivary stones and only 20% of parotid stones are radiopaque and may be seen on imaging.

Treatment Options

Typically, the treatment for sialolithiasis is surgical. Interglandular stones in the submandibular gland typically result in the removal of the gland. Salivary stones that are located near the orifice may be manipulated out. Stones that are more distal are removed through an incision through excretory duct system.

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Complications

Complications include abscess formation and infection.

Ranula

Ranula is a sialocele of the sublingual gland typically found in the floor of the mouth. The treatment is surgical excision.

◆ ICD-9 Codes

527.0 Atrophy of salivary gland
527.1 Hypertrophy of salivary gland
527.2 Sialadenitis
527.3 Abscess of salivary gland
527.4 Fistula of salivary gland
527.5 Sialolithiasis
527.6 Mucocele of salivary gland
527.7 Disturbance of salivary secretion
527.8 Other specified diseases of the salivary glands
527.9 Unspecified disease of the salivary glands

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Witt RL, ed. Salivary Gland Diseases: Surgical and Medical Management. Stuttgart/New York: Thieme; 2005

5.3.2 Benign Salivary Gland Tumors

◆ Key Features

- Tumors of the salivary glands represent 2 to 4% of head and neck neoplasms.
- 70% of salivary gland tumors originate in the parotid gland
- 85% of salivary gland tumors are pleomorphic adenomas (benign mixed tumors)
- Salivary gland tumors most often present as painless masses.
- The most common parotid mass in a child is a hemangioma.

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Table 5.12 Classification of Benign Primary Epithelial Salivary Gland Tumors

Benign mixed tumor (pleomorphic adenoma)
Warthin tumor (papillary cystadenoma lymphomatosum)
Oncocytoma (Oxyphil adenoma)
Monomorphic tumors
Sebaceous tumors
Benign lymphoepithelial lesion
Papillary ductal adenoma (papilloma)

Pleomorphic adenomas are the most common benign salivary gland tumors. They constitute 70% of parotid gland tumors and 50% of submandibular gland tumors. Pleomorphic adenomas are often located in the tail of the parotid gland. When they are found in the minor salivary glands, the hard palate is the site most frequently involved, followed by the upper lip. Warthin's tumors (papillary cystadenoma lymphomatosum) occur almost exclusively in the parotid gland. They account for 4 to 11.2% of all salivary gland tumors. These lesions can be multifocal and bilateral (10%) and are more common in individuals who use tobacco (**Table 5.12**).

◆ Clinical

Signs

A painless mass can be felt in the affected salivary gland.

Symptoms

There is slowly increasing, painless swelling of the affected salivary gland. (Pain, rapid growth of long-standing benign parotid mass, and facial nerve paralysis should raise the suspicion of malignant disease; see Chapter 5.3.3.)

Differential Diagnosis

- Adenopathy of periparotid or perifacial lymph nodes
- Malignant salivary tumors
- Metastatic lesions
- Autoimmune, infectious, or inflammatory salivary gland lesions

◆ Evaluation

History

History should include questions about time course and onset of mass, pain, relationship of swelling to eating, xerophthalmia, known autoimmune conditions, weight loss, fever, night sweats, and infectious diseases.

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Physical Exam

A full head and neck examination including the salivary glands and the neck, and a bimanual examination of submandibular glands should be performed. Facial nerve function should be documented.

Imaging

Imaging studies are most helpful in the diagnostic evaluation. Ultrasound may help FNAB localization. MRI is the most sensitive test for establishing the borders of soft tissue tumor extension. CT is usually sufficient.

Labs

A white blood cell count should be ordered to investigate for any evidence of leukocytosis and neutrophilic shift, possible infectious process, or lymphoproliferative disease. A lymphoepithelial cyst or recurrent parotid abscess should prompt HIV testing.

Other Tests

FNAB findings provide evidence for a preoperative diagnosis.

Pathology

Pleomorphic adenomas are characterized by variable, diverse, structural histologic patterns. Frequently, they have growth patterns of sheets, strands, or islands of spindle and stellate cells, with a myxoid configuration occasionally predominating. Warthin's tumors are composed of an oncocytic epithelial component that can have a papillary, glandular–cystic, and/or solid growth pattern. Oncocytomas are composed of large oxyphilic cells (oncocytes).

◆ Treatment Options

Medical

Medical therapy is appropriate for infectious and inflammatory processes. Radiotherapy is for nonoperative candidates with pleomorphic adenomas or recurrent multiple pleomorphic adenomas.

Surgical

Management of benign salivary gland tumors includes complete removal with an adequate margin of tissue to avoid recurrences. This involves superficial parotidectomy, total parotidectomy with preservation of facial nerve for deep lobe masses, or submandibular gland removal.

◆ Complications

During submandibular gland excision, unintentional injury may be inflicted on the lingual, hypoglossal, or mandibular branch of the facial nerve.

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Parotidectomy

Recurrence is usually caused by inadequate excision, tumor enucleation, or tumor spillage. Facial nerve paralysis (paresis) takes a few weeks to resolve spontaneously but can last months or be permanent. Reported rates of permanent postoperative facial nerve paresis range from 0 to 30%. Eye care is important until there is a return of facial nerve function.

Frey Syndrome (Gustatory Sweating)

Frey syndrome is caused by an aberrant connection of the postganglionic gustatory parasympathetic fibers to sympathetic fibers of the sweat glands of the overlying skin. It is best avoided by raising a thick flap.

◆ Outcome and Follow-Up

Evaluate postoperative facial, hypoglossal, and lingual nerve function. Occasionally, transient facial nerve paresis occurs, but it usually resolves within weeks after surgery.

Outcome is typically excellent, and the recurrence rate is very low. Malignant degeneration to carcinoma ex pleomorphic adenoma occurs in 1.4% to 6.3% of untreated pleomorphic adenomas. Malignant degeneration is often associated with a prolonged history of untreated or recurrent pleomorphic adenoma.

◆ ICD-9 Codes

210.4	Benign neoplasms of minor salivary glands: NOS
235.0	Major salivary glands
210.2	Benign lesion major salivary glands
527	Diseases of the salivary glands

Further Reading

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5.3.3 Malignant Salivary Gland Tumors

◆ Key Features

- Malignant salivary gland neoplasms account for 0.5% of all malignancies.
- Malignant salivary gland neoplasms account for 3 to 5% of all head and neck cancer.
- The most common malignant major and minor salivary gland tumor is mucoepidermoid carcinoma.
- Numbness or facial nerve weakness and pain in conjunction with salivary gland mass suggest malignancy.

◆ Epidemiology

The frequency of malignant lesions varies by site. Approximately 20 to 25% of parotid tumors, 35 to 40% of submandibular tumors, 50% of palate tumors, and >90% of sublingual gland tumors are malignant. Mucoepidermoid carcinoma is the most common malignant neoplasm of the salivary glands. Most cases originate in the parotid gland. Mucoepidermoid carcinoma is a malignant epithelial tumor that is composed of various proportions of mucous, epidermoid, intermediate, columnar, and clear cells. Microscopic grading of mucoepidermoid carcinoma is important to determine the prognosis. Mucoepidermoid carcinomas are graded as low, intermediate, and high based on the degree of epidermoid and mucinous cell populations.

Adenoid cystic carcinoma (formerly known as cylindroma) is a slow-growing but aggressive neoplasm with a remarkable capacity for recurrence. This is the most common malignant tumor of the submandibular and minor salivary glands and constitutes 4% of all salivary gland tumors. Morphologically, three growth patterns have been described: cribriform or classic pattern, tubular, and solid or basaloid pattern. This tumor has a propensity for perineural spread. Regardless of histologic grade, adenoid cystic carcinomas, with their unusually slow biologic growth, tend to have a protracted course and ultimately a poor outcome, with a 10-year survival reported to be <50% for all grades. Many advocate following these patients for the duration of their lifetime as recurrence can be quite late.

Acinic cell carcinoma is a malignant epithelial neoplasm in which the neoplastic cells express acinar differentiation. In Armed Forces Institute of Pathology (AFIP) data of salivary gland neoplasms, acinic cell carcinoma is the third most common salivary gland epithelial neoplasm; >80% occur in the parotid gland, women are affected more than men, and the mean patient age is 44 years. Clinically, patients typically present with a slowly enlarging mass in the parotid region. Pain is a symptom in >33% of patients. For acinic cell carcinoma, staging is likely a better predictor of outcome than histologic grading.

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Carcinoma ex pleomorphic adenoma, also known as carcinoma ex mixed tumor or pleomorphic carcinoma, is a carcinoma that shows histologic evidence of arising from or in a benign pleomorphic adenoma. The neoplasm occurs primarily in the major salivary glands. Diagnosis requires the identification of benign tumor in the tissue sample. The incidence or relative frequency of this tumor varies considerably depending on the study cited.

Primary SCC, also known as primary epidermoid carcinoma, is a malignant epithelial neoplasm of the major salivary glands that is composed of squamous (i.e., epidermoid) cells. Diagnosis requires the exclusion of primary disease located in some other head and neck site; indeed, most SCCs of the major salivary glands represent metastatic disease (**Table 5.13**).

Table 5.13 Classification of Malignant Primary Epithelial Salivary Gland Tumors

Mucoepidermoid carcinoma
Adenoid cystic carcinoma
Adenocarcinomas
Acinic cell carcinoma
Polymorphous low-grade adenocarcinoma
Adenocarcinoma, NOS
Rare adenocarcinomas
Basal cell adenocarcinoma
Clear cell carcinoma
Cystadenocarcinoma
Sebaceous adenocarcinoma
Sebaceous lymphadenocarcinoma
Oncocytic carcinoma
Salivary duct carcinoma
Mucinous adenocarcinoma
Malignant mixed tumors
Carcinoma ex pleomorphic adenoma
Carcinosarcoma
Metastasizing mixed tumor
Rare carcinomas
Primary squamous cell carcinoma
Epithelial-myoepithelial carcinoma
Anaplastic small cell carcinoma
Undifferentiated carcinomas
Small cell undifferentiated carcinoma
Large cell undifferentiated carcinoma
Lymphoepithelial carcinoma
Myoepithelial carcinoma
Adenosquamous carcinoma

Abbreviation: NOS, not otherwise specified.

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Malignant neoplasms whose origins lie outside the salivary glands may involve the major salivary glands by

1. Direct invasion from cancers that lie adjacent to the salivary glands
2. Hematogenous metastases from distant primary tumors
3. Lymphatic metastases to lymph nodes within the salivary gland

Direct invasion of nonsalivary gland tumors into the major salivary glands is principally from squamous cell and basal cell carcinomas of the overlying skin.

Lymphomas of the major salivary glands are characteristically of the NHL type. In an AFIP review of case files, NHL accounted for 16.3% of all malignant tumors that occurred in the major salivary glands, and disease in the parotid gland accounted for ~80% of all cases.

The most common malignant major and minor salivary gland tumor is mucoepidermoid carcinoma, which constitutes ~10% of all salivary gland neoplasms and ~35% of malignant salivary gland neoplasms. Prior exposure to ionizing radiation appears to substantially increase the risk for development malignant neoplasms of the major salivary glands.

◆ Clinical

Signs and Symptoms

A painless swelling of the affected salivary gland is a sign of a salivary gland tumor. Approximately 10 to 15% of malignant parotid neoplasms present with pain. Occasionally, malignant salivary gland tumors may be characterized by rapid growth or a sudden growth spurt. Numbness or nerve weakness caused by nerve involvement and persistent facial pain are highly suggestive of malignancy. Depending on the site of the primary tumor, other symptoms include drainage from the ipsilateral ear, dysphagia, trismus, and facial paralysis. The overlying skin or mucosa may become ulcerated.

Differential Diagnosis

- Adenopathy of periparotid or perifacial lymph nodes
- Benign salivary tumors
- Metastatic lesions
- Autoimmune, infectious, or inflammatory salivary gland lesions

◆ Evaluation

History

History includes questions about time course and onset of mass, pain, facial weakness, weight loss, known autoimmune disease, fever, and night sweats.

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Physical Exam

A full head and neck examination including salivary glands, neck, and a bimanual examination of submandibular glands should be performed. CN function should be assessed.

Imaging

Imaging studies are helpful in the diagnostic evaluation. Ultrasound may help FNAB localization. MRI is the most sensitive test for establishing the borders of soft tissue tumor extension. Contrast-enhanced CT is usually sufficient.

Other Tests

FNAB findings often provide evidence for a preoperative diagnosis.

Pathology

Salivary gland neoplasms show extreme histologic diversity. These neoplasms include malignant tumors of epithelial, mesenchymal, and lymphoid origin. Histologic grading (**Table 5.14**) of salivary gland carcinomas is important to determine the proper treatment approach. However, it is not an independent indicator of the clinical course and must be considered in the context of the clinical stage (see Staging of Malignant Salivary Gland Tumors, below).

◆ Treatment Options

Medical

The use of chemotherapy for malignant salivary gland tumors remains under evaluation. Postoperative radiotherapy augments surgical resection, particularly for the high-grade neoplasms, when margins are close or involved, when tumors are large, or when histologic evidence of lymph node metastases is present. Fast neutron-beam radiation or accelerated hyperfractionated photon beam schedules have been reported to be more effective than conventional x-ray therapy in the treatment of inoperable, unresectable, or recurrent malignant salivary gland tumors.

Surgical

The minimum therapy for low-grade (and intermediate-grade mucoepidermoid carcinoma) malignancies of the superficial portion of the parotid gland is a superficial parotidectomy. For all other lesions, a total parotidectomy with facial nerve preservation is often indicated. The facial nerve or its branches should be resected if involved by tumor. Rarely a mastoidectomy or temporal bone resection may be performed to obtain a negative margin on CN VII. Neck dissection may be performed, depending on tumor type and nodal involvement.

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Table 5.14 Histologic Grading of Salivary Gland Carcinomas

Low grade
Acinic cell carcinoma
Basal cell adenocarcinoma
Clear cell carcinoma
Cystadenocarcinoma
Epithelial-myoepithelial carcinoma
Mucinous adenocarcinoma
Polymorphous low-grade adenocarcinoma
Low, intermediate, and high grade
Adenocarcinoma, NOS
Mucoepidermoid carcinoma
Squamous cell carcinoma
Intermediate and high grade
Myoepithelial carcinoma
High grade
Anaplastic small cell carcinoma
Carcinosarcoma
Large cell undifferentiated carcinoma
Small cell undifferentiated carcinoma
Salivary duct carcinoma

Abbreviation: NOS, not otherwise specified.

◆ Complications

Parotidectomy

Recurrence may be caused by inadequate excision, positive margins, or tumor spillage. Facial nerve paralysis and cosmetic deformity may need to be addressed surgically. CN dysfunction from the tumor and resultant surgery is a possible complication. Eye care is important in the presence of facial nerve paralysis.

◆ Outcome and Follow-Up

Early-stage low-grade malignant salivary gland tumors are usually curable by adequate surgical resection alone. The prognosis is more favorable when the tumor is in a major salivary gland. Large bulky tumors or high-grade tumors carry a poorer prognosis. The prognosis also depends on the following: gland in which they arise, histology, grade, the stage, perineural involvement, and spread to adjacent structures, lymph nodes, or distant sites. The prognosis for any treated cancer patient with progressing or relapsing disease is poor, regardless of histologic type or stage.

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◆ ICD-9 Codes

142 Malignant neoplasm of major salivary glands
142.0 Malignant neoplasm of parotid gland
142.1 Malignant neoplasm of submandibular gland
142.2 Malignant neoplasm of sublingual gland
142.8 Malignant neoplasm of other major salivary glands
142.9 Malignant neoplasm of salivary gland, unspecified

◆ Staging of Malignant Salivary Gland Tumors

Salivary Gland Cancer: Invasive Carcinomas of the Parotid, Submandibular, and Sublingual Glands

Primary Tumor

TX: Primary tumor cannot be assessed

T0: No evidence of primary tumor

T1: Tumor ≤2 cm in greatest dimension without extraparenchymal extension

T2: Tumor >2 cm but not >4 cm in greatest dimension without extraparenchymal extension

T3: Tumor >4 cm and/or tumor having extraparenchymal extension

T4a: Moderately advanced disease. Tumor invades the skin, the mandible, the ear canal, and/or the facial nerve

T4b: Very advanced disease. Tumor invades the skull base and/or the pterygoid plates and/or encases the carotid artery

Regional Lymph Nodes*

NX: Cannot be assessed

N0: No regional lymph node metastasis

N1: Metastasis in a single ipsilateral lymph node, ≤3 cm in greatest dimension

N2a: Metastasis in a single ipsilateral lymph node, >3 cm but not >6 cm in greatest dimension

N2b: Metastasis in multiple ipsilateral lymph nodes, none >6 cm in greatest dimension

N2c: Metastasis in bilateral or contralateral lymph nodes, none >6 cm in greatest dimension

N3: Metastasis in a lymph node, >6 cm in greatest dimension

*Superior mediastinal lymph nodes are considered regional lymph nodes (level VII).

Midline nodes are considered ipsilateral nodes.

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Distant Metastasis

M0: No distant metastasis
M1: Distant metastasis

American Joint Committee on Cancer Stage Groupings of Malignant Salivary Gland Tumors

Stage I

T1 N0 M0

Stage II

T2 N0 M0

Stage III

T3 N0 M0
T1 N1 M0
T2 N1 M0
T3 N1 M0

Stage IVA

T4a N0 M0
T4a N1 M0
T1 N2 M0
T2 N2 M0
T3 N2 M0
T4a N2 M0

Stage IVB

T4b any N M0
Any T N3 M0

Stage IVC

Any T any N M1

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5.4 Endocrine Surgery of the Head and Neck

5.4.0 Embryology and Anatomy of the Thyroid Gland

◆ Key Features

- The thyroid is a vascular H-shaped gland located in the anterior lower neck just below the larynx (it is approximately the size and shape of a bowtie).
- Its function is the synthesis and secretion of thyroid hormones thyroxine (T4) and triiodothyronine (T3).
- The gland's characteristic shape is formed by two elongated lateral lobes with superior and inferior poles connected by a median isthmus.
- The isthmus overlies the second to fourth tracheal rings.
- The thyroid weighs on average 25 g in adults (slightly lighter in males).
- A pyramidal lobe may be present as an extension of the embryologic thyroglossal duct (50%).
- The tubercle of Zuckerkandl is a lateral or posterior projection from the thyroid lobe.
- It is an anatomic landmark for the recurrent laryngeal nerve in thyroid surgery (**Table 5.15**).
- The thyroid gland is usually not visible externally; when it increases in size, it forms a characteristic swelling in the neck called a goiter.

◆ Embryology

The human thyroid gland begins to develop at around 4 weeks after conception, and moves down the neck while forming its characteristic bilobular structure, which is completed by the third trimester (**Fig. 5.11**).

◆ Ligaments and Fascia

The thyroid gland is ensheathed by the middle layer of the deep cervical fascia. The posteromedial part of the gland is attached to the cricoid cartilage, first and second tracheal ring, by the posterior suspensory ligament (Berry ligament). Under the layer of cervical fascia the gland has an inner true capsule, which is thin and adheres closely to the gland.

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Table 5.15 Landmarks for Identifying the Recurrent Laryngeal Nerve during Thyroid Surgery

Sim's triangle (the carotid triangle) is formed by – Carotid artery – Trachea – Inferior pole of the thyroid
The recurrent laryngeal nerve may be identified 0.5 cm below the inferior cornu of the thyroid cartilage.
The tubercle of Zuckerkandl is a lateral or posterior projection from the lateral thyroid lobe, which indicates the point of embryologic fusion of the ultimobranchial body and the principal median thyroid process. The tubercle of Zuckerkandl is lateral to the recurrent laryngeal nerve.

◆ Thyroid Cells

The thyroid is made up of follicular and parafollicular cells (C cells; clear, light cells). Follicular cells are responsible for the formation of the colloid (iodothyroglobulin), whose function is to store thyroid hormones prior to their secretion). The parafollicular cells are scattered between the thyroid follicles and produce the hormone calcitonin, which is central to calcium homeostasis.

◆ Vasculature Anatomy

Arteries

The principal arterial supply is from the superior (STA) and inferior (ITA) thyroid arteries. The STA originates from the external carotid artery. The ITA originates from thyrocervical trunk, which is a branch of the subclavian artery. The ITA serves as an important surgical landmark for the recurrent laryngeal nerve and parathyroid glands. Occasionally, the thyroidea ima artery sullies the thyroid isthmus directly from the innominate artery or aortic arch (1.5–12%).

Veins

There are the superior, middle, and inferior thyroid veins. The superior and middle thyroid veins drain into the internal jugular vein.

Lymphatics

The upper middle and lower deep cervical nodes receive lymph from the thyroid gland as do the pretracheal and paratracheal nodes and the prelaryngeal (Delphian) node.

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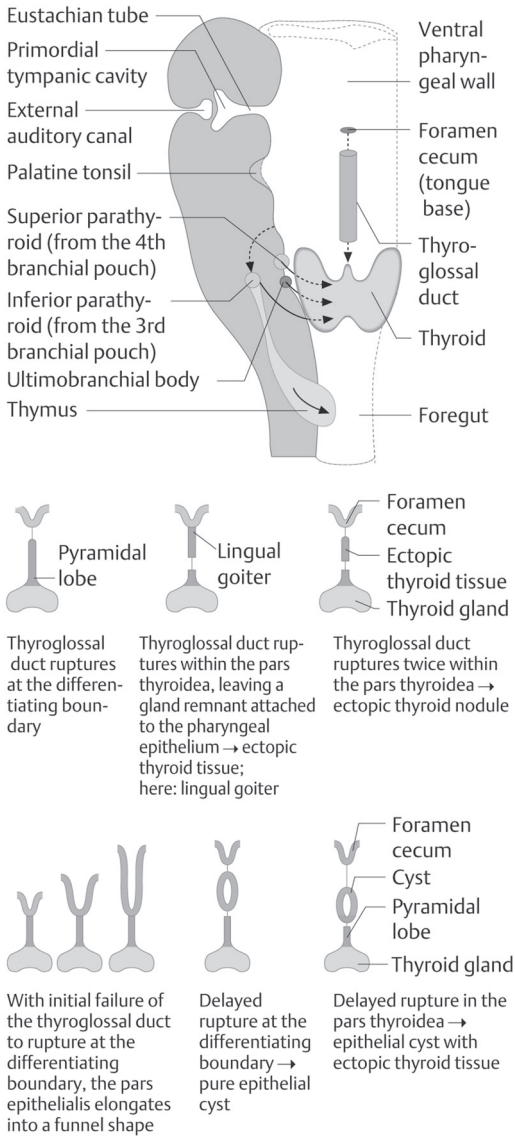

Fig. 5.11 (A–C) Embryology and malformations of the thyroid gland. (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:323.)

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◆ Nerves

The recurrent laryngeal nerve innervates all intrinsic laryngeal muscles except the cricothyroid muscle. The left recurrent laryngeal nerve lies in the tracheoesophageal groove throughout its length. The right recurrent laryngeal nerve lies laterally in the first part of its course but veers medially and ascends toward the gland (a slightly diagonal course). In rare cases, the right recurrent laryngeal nerve is nonrecurrent and comes directly off the vagus.

The superior laryngeal nerve is subdivided into external and internal branches. The external branch parallels the superior thyroid artery and descends to innervate the cricothyroid muscle. The internal branch parallels the superior thyroid artery and enters the thyrohyoid membrane to give sensation to the larynx above the goiter.

The nerve of Galen is an anastomosis between the sensory branch of the recurrent laryngeal nerve and the internal branch of the superior laryngeal nerve.

Further Reading

Schuenke M, Schulte E, Schumacher U. Thieme Atlas of Anatomy: Neck and Internal Organs. Stuttgart/New York: Thieme; 2006

5.4.1 Physiology of the Thyroid Gland

◆ Key Features

- The functional unit of the thyroid gland is the follicle (**Fig. 5.12**).
- Thyroid follicles are composed of a single layer of epithelial cells (thyroid follicular cells) surrounding a central space filled with colloid.
- The follicular cells synthesize Tg, a large tyrosine-rich glycoprotein, and secrete it into the lumen of the follicle; colloid is essentially a pool of Tg.
- Key thyroid hormones include thyrotropin-releasing hormone (TRH), thyroid-stimulating hormone (TSH), T4, and T3.
- Only 1% of total thyroid hormone is in the unbound or free state and available for metabolic purposes. The rest is bound to globulin, prealbumin, and albumin.
- Calcitonin is a peptide produced by the parafollicular cells of the thyroid gland. It reduces resorption of calcium in the bone and lowers the serum calcium.

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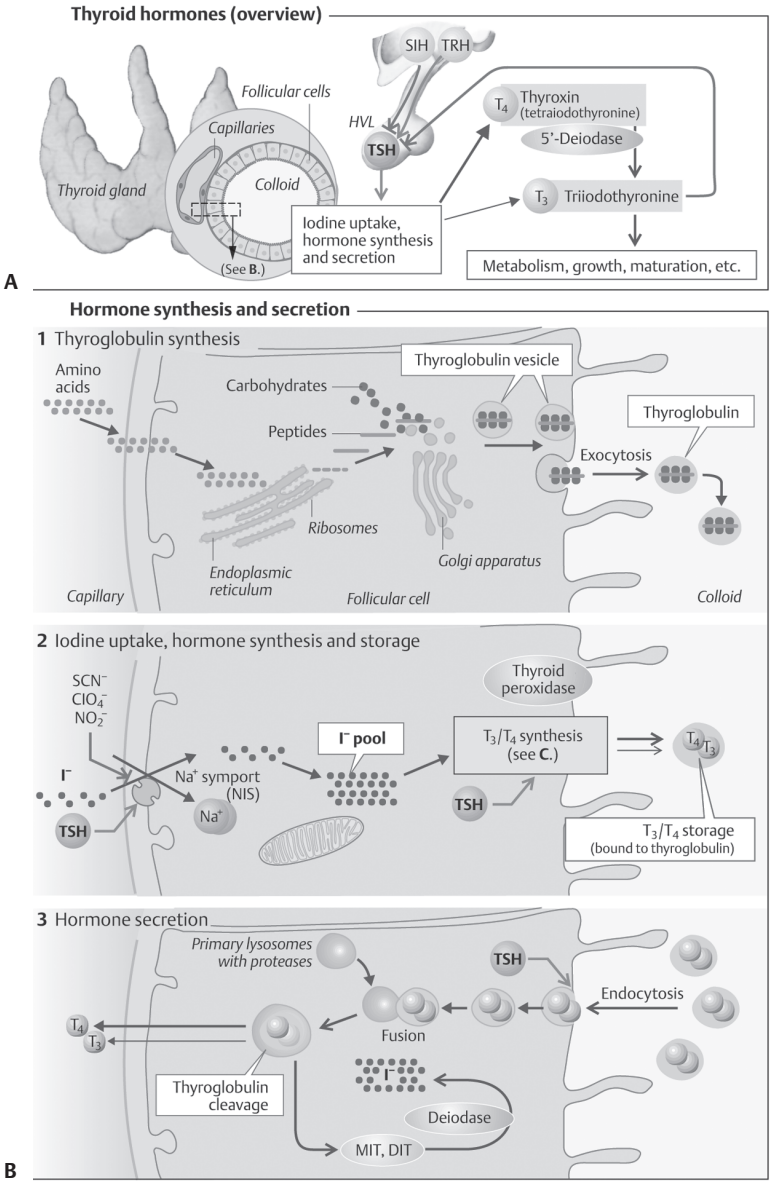

Fig. 5.12 **(A,B)** Physiology of thyroid hormone production and synthesis. (From Silbernagl S, Despopoulos A. Color Atlas of Physiology. 6th ed. Stuttgart/New York: Thieme; 2009:289.)

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◆ Thyroid Hormone Regulation

The hypothalamus secretes TRH, which passes via the portal venous system to the anterior pituitary. Here, it stimulates synthesis and release of TSH. The TSH stimulates iodide trapping, release of thyroid hormones, and thyroid growth. The thyroid hormones exert negative feedback control of the hypothalamic–pituitary axis.

◆ Steps in Thyroid Hormone Synthesis

1. Iodide uptake: The thyroid selectively concentrates inorganic iodine (iodide).
2. Organification: Iodine is produced by oxidation of iodide. Iodine reacts with tyrosine on the Tg first to produce monoiodotyrosine (MIT) and then diiodotyrosine (DIT).
3. Coupling: Coupled reactions between DIT lead to the formation of thyroxine T4 and those between DIT and MIT form T3.
4. Storage: The T4 and T3 are stored as colloid, still a part of the Tg molecule.
5. Release: Endocytosis of the stored colloid leads to formation of colloid droplets (phagolysosomes) in which Tg is digested by proteases to release T4, T3, DIT, and MIT into the blood.

Note that thyroid peroxidase (TPO) catalyzes the oxidation of iodide and its transfer to tyrosine (organification) and coupling.

◆ Thyroid Hormones and Their Actions

The thyroid gland releases two hormones into the blood, mostly T4 and some T3. T3 is the active hormone, whereas T4 is the prohormone and converts to T3 in many tissues of the body through a process called “peripheral deiodination.” Effects include:

- Calorigenesis: the thyroid hormones generate heat and increase oxygen consumption
- Carbohydrate and fat metabolism
- Growth and development

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5.4.2 Thyroid Evaluation

◆ Key Features

- Thyroid function tests are common procedures performed to determine how well the thyroid is functioning.
- Evaluation may include blood tests and imaging (ultrasound), nuclear thyroid scans, and functional stimulation tests.

◆ Thyroid Function Tests

T4 and T3 (Total)

Both T4 (>99%) and T3 (~98%) are tightly bound to transport proteins in the plasma: thyroxine-binding globulin (TBG), thyroxine-binding prealbumin (TBPA), or albumin. Therefore, only 1% of the thyroid hormones are in the metabolically active "free" state. Assays of "total" T4 or T3 measure mainly the protein-bound hormone (plus the unbound or "free" hormone). Values may vary with conditions that effect protein concentration (**Table 5.16**). Because drugs and illness can alter concentrations of binding proteins, it is therefore necessary to estimate free hormone concentrations.

Thyroid-Stimulating Hormone

The TSH assay is the screening test of choice for thyroid function. The very sensitive TSH assay (it measures TSH into very low ranges between 0.01 and 0.001 mU/L) is currently in wide use.

Free T4 and T3 (FT4 and FT3)

These assays measure the unbound, metabolically active thyroid hormones in the circulation (~0.0003 to total). Free thyroid hormone tests fall into two main categories: equilibrium dialysis (not affected by TBG abnormalities) and analogue assays (affected by protein binding but still superior to total T3 and T4 assays).

Table 5.16 Conditions Affecting Protein Concentration

Increased TBG (High T4)	Decreased TBG (Low T4)
Pregnancy	Liver disease/kidney disease
Estrogens	Androgens
Oral contraceptive pills	Congenital TBG deficiency
Drugs: narcotics, clofibrate	Drugs: steroids, NSAIDs, ASA

Abbreviations: ASA, acetylsalicylic acid (aspirin); NSAIDs, nonsteroidal antiinflammatory drugs; TBG, thyroxine-binding globulin.

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Table 5.17 Different Causes for Thyrotoxicosis

High RAI Uptake	Low RAI Uptake
Graves disease	Exogenous use of thyroxine (thyroiditis factitia)
Hot nodules Toxic multinodular goiter Solitary toxic adenoma	Destructive thyroiditis – subacute thyroiditis Postpartum Silent
TSH-secreting pituitary tumor	Iodine-induced thyrotoxicosis (contrast dye, diet, amiodarone)

Abbreviations: TSH, thyroid-stimulating hormone; RAI, radioactive iodine.

Thyroid Hormone Uptake Test

The thyroid hormone uptake test (or the T3 resin uptake test) is inversely proportional to the protein-bound T4; accordingly, T3 resin uptake is low when T4 protein binding is increased and high when protein binding is reduced. This measurement helps clinicians distinguish protein-binding disorders from true thyroid diseases.

Radioactive Iodine Uptake

Thyroid follicular cells have iodine pumps that trap iodine in cells for thyroid hormone synthesis. The activity of these pumps can be determined by measuring the radioactive iodine (RAI) uptake. Uptake of ^{131}I by the gland is measured after oral ingestion of RAI. The normal 24-hour RAI uptake is ~10 to 25% in the United States. This test is most useful in the differentiating of causes for thyrotoxicosis (**Table 5.17**).

Thyroglobulin Measurements

Thyroglobulin (Tg) measurements are taken based on two indications:

1. Detection of residual or recurrent epithelial thyroid cancers (papillary, follicular, and Hürthle cell carcinomas) after thyroidectomy
2. Differentiation of thyroiditis factitia (Tg suppressed) from all forms of endogenous hyperthyroidism (Tg normal or high)

This test is not reliable in the presence of circulating anti-Tg antibodies.

Other biochemical tests of thyroid function, such as TRH tests, have been used only rarely since the advent of the highly sensitive TSH assays (**Table 5.18**).

◆ Thyroid Imaging

Thyroid Radionucleotide Study

A thyroid radionuclide study uses a gamma camera to obtain information on the size shape and position of the thyroid gland and distribution of

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Table 5.18 Summary of Thyroid Function Work-Up

First Test: TSH	Second Test: Free T4	Clinical Status
High	Low	Primary hypothyroidism
	Normal	Subclinical hypothyroidism
	High	Pituitary hyperthyroidism
Low	High	Thyrotoxicosis
	Normal	Subclinical hyperthyroidism
	Low	Pituitary hypothyroidism

Abbreviation: TSH, thyroid-stimulating hormone.

administered isotope within it. Either technetium-99 (^{99m}Tc) or ^{131}I may be used. Both scans will distinguish between solitary and multiple nodules. They will disclose the presence of a "warm nodule" against a normally active background, a hyperfunctioning "hot" nodule against a suppressed or silent background (4% malignancy rate), or a hypofunctioning "cold" nodule (5–20% malignancy rate).

Ultrasound

Ultrasound may distinguish between a single node or a multinodular goiter and differentiate between solid and cystic lesions. When the precise location of a nodule is in doubt, ultrasound can be used to guide an FNAB. Doppler flow may show vascularity of a thyroid nodule.

Fine-Needle Aspiration Biopsy

FNAB is the gold standard assessment of a thyroid nodule. It has a 1 to 10% false-negative rate, depending on the experience of the cytologist. It is especially useful in the identification of papillary, medullary, and poorly differentiated carcinomas. It may also be used for diagnosing thyroid lymphoma, but it is not considered useful in distinguishing between follicular carcinoma and adenoma.

Further Reading

Terris DJ, Gourin CG, eds. Thyroid and Parathyroid Diseases: Medical and Surgical Management. Stuttgart/New York: Thieme; 2009

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5.4.3 Thyroid Nodules and Cysts

◆ Key Features

- Nodular disease of the thyroid gland is estimated to be present in 5 to 10% of the population.
- It is more common in women.
- <5% of thyroid nodules are malignant.

Palpable thyroid nodules occur in ~4 to 7% of the general population. The prevalence of nonpalpable thyroid nodules is ~20 to 50% by ultrasound. The important question when a thyroid nodule is detected is whether it is thyroid cancer (which accounts for 4–6.5% of all thyroid nodules).

◆ Epidemiology of Nodules

Males are affected more than females. The prevalence of cancer is higher in children, adults <30 years or >60 years old, patients with a history of head and neck radiation, and those with a family history of thyroid cancer. Contrary to this, the prevalence of cancer may be lower in patients with multinodular goiters. All autonomously hyperfunctioning ("hot") nodules are benign.

◆ Clinical

The usual presentation of thyroid nodules is a lump in the neck noted by the patient, or as an incidental finding during routine physical examination, or during a radiologic procedure, such as carotid ultrasonography or neck CT. Symptoms associated with increased risk of malignancy include a rapid increase in size, dysphagia, hoarseness (nerve involvement), pain, a family history of thyroid cancer or multiple endocrine neoplasias, and a history of radiation exposure.

◆ Evaluation of Nodules

History

Pertinent history is a history of head and neck irradiation, total body irradiation, thyroid carcinoma in a first-degree relative, rapid growth of the nodule, hoarseness, dysphagia, neck pain or pressure, and exposure to radiation from a nuclear power plant; any symptoms of hyperthyroidism (e.g., due to an autonomous hot nodule) or hypothyroidism (e.g., due to Hashimoto thyroiditis).

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Physical Exam

A complete physical exam focusing on the thyroid gland and adjacent cervical lymph nodes should be performed. The nodule should be palpated to note size, consistency, mobility, tenderness. Vocal fold mobility (nasopharyngoscope) and stridor should be noted. Cancer is suspected when nodules are >4 cm in size, when they are fixed to the surrounding structures, or when there is associated lymphadenopathy, or vocal fold paralysis.

Imaging

- If serum TSH is low, get a radionuclide thyroid scan ^{99m}Tc thyroid scan or ^{123}I thyroid scan (RAI uptake) to determine whether it is a "hot," "warm," or "cold" nodule; a hot functioning nodule rarely harbors malignancy.
- If serum TSH is normal or high, diagnostic thyroid ultrasound will help to (1) determine whether there is truly a thyroid nodule that corresponds to the palpable abnormality or a thyroid cyst, (2) look for other nonpalpable thyroid nodules that may also need to be biopsied and the location and the number of the nodules, and (3) guide FNAB.
- CT and MRI are used to evaluate nodule size or characteristics, adenopathy, multinodular goiters causing pressure symptoms, substernal goiter, airway and vascular displacement, invasion of tumor. These studies are not routinely employed, however.
- Consider pulmonary function testing with flow-volume loops to assess upper airway function.
- Consider a chest CT to evaluate displacement of trachea.

Other Tests

FNAB is the single most important procedure for differentiating benign from malignant thyroid nodules. FNAB can give one of the following results: benign, malignant, suspicious follicular neoplasms, or nondiagnostic. The following approach should be taken in interpreting and managing FNAB results:

- Benign (~67% of FNABs): Periodic follow-up is recommended with physical examination and ultrasound imaging if needed, and FNAB is repeated if there is a change in size or appearance of sonographic features suggestive of malignancy. Routine thyroid hormone suppression therapy to decrease nodule size is not recommended.
- Positive for malignancy (~4%): Surgery is recommended.
- Suggestive of malignancy (~10%): This often occurs with a follicular neoplasm or Hürthle cell neoplasm, where it is impossible to distinguish between a benign versus a malignant neoplasm on FNAB alone. Lobectomy or hemithyroidectomy is recommended for a more

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definitive diagnosis. This allows one to assess for the presence of vascular, capsular, or lymphatic invasion or metastasis, which indicates malignancy.

- Nondiagnostic (~19%; may be even less with ultrasound guidance): This result is due to an inadequate specimen, "too few cells." If the FNAB is nondiagnostic, it should be repeated, preferably under ultrasound guidance.

Other cytopathologic yield of FNAB includes uniform follicular epithelium and abundant colloid suggesting nodular or adenomatous goiters, inflammatory cells suggesting thyroiditis, papillary cells (psammoma bodies), giant cells suggesting papillary carcinoma, amyloid deposits stained with Congo red suggesting medullary carcinoma, and undifferentiated cells suggesting anaplastic carcinoma.

Serum TSH is an initial screening test and should be checked in all patients with thyroid nodules. If TSH is low, a radionuclide thyroid scan should be obtained to determine if the nodule is functioning, or hot (uptake is greater in the nodule than in the surrounding normal thyroid). Functioning nodules are rarely malignant and so do not need to be evaluated further with FNAB. Further evaluation and treatment of the hyperthyroidism may be indicated. If the TSH is normal or high, a thyroid scan is of little value and should not be done.

Serum calcitonin is a screening test used for detection of C-cell hyperplasia and medullary thyroid cancer at an earlier stage, if clinically indicated (not routinely).

Serum Tg is not routinely recommended, since it can be elevated in most thyroid diseases and is an insensitive and nonspecific test for thyroid cancer (it is the most valuable test as a marker of recurrence in follow-up of thyroid cancer after surgical treatment followed by RAI treatment; see Chapter 5.4.8).

If thyroiditis is suspected, FT4, T3, ESR, anti-TPO and anti-Tg should be checked only if they are clinically indicated. They have no routine role.

◆ Thyroid Cysts

Most cystic nodules of the thyroid are degenerating benign adenomas, which can be seen by ultrasonography within the wall of or adjacent to the cystic nodule. They may arise from cystic degeneration of malignancy. Cysts that have a partial solid component are more suggestive of malignancy.

Clinical

A thyroid cyst presents as a smooth, round mass.

Evaluation

FNAB, ultrasound, and CT may all be used to evaluate for a thyroid cyst.

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Treatment Options

The cyst may be observed for regression. FNAB may relieve pain and provide diagnostic cytology. In the case of recurrent cysts or bloody aspirates, consider thyroid lobectomy.

◆ ICD-9 Codes

241.0 Thyroid nodule

Further Reading

Gemsenjaeger E. Atlas of Thyroid Surgery: Principles, Practice, and Clinical Cases Stuttgart/New York: Thieme; 2009
Terris DJ, Gourin CG, eds. Thyroid and Parathyroid Diseases: Medical and Surgical Management. Stuttgart/New York: Thieme; 2009

5.4.4 Hyperthyroidism

◆ Key Features

- Hyperthyroidism is a hypermetabolic condition associated with elevated levels of free T4, free T3, or both.
- The incidence of hyperthyroidism is between 0.05 and 1.3%, with most cases consisting of subclinical disease.
- It is caused by excess synthesis and secretion of thyroid hormone by the thyroid.
- Its causes include diffuse toxic goiter (Graves disease), toxic multinodular goiter, and toxic adenoma.

Thyrotoxicosis is the presence of an excess of thyroid hormone (T4 and/or T3) in the body, which may be due to overproduction of thyroid hormone by the thyroid gland, increased release of thyroid hormone in conditions like thyroiditis, and exogenous ingestion of thyroid hormone preparations. Hyperthyroidism refers to causes of thyrotoxicosis in which thyroid produces excess thyroid hormone.

◆ Clinical

Signs

Signs include sinus tachycardia, atrial fibrillation, hyperreflexia with rapid relaxation of tendon reflexes, tachycardia, lid lag and stare, hair loss, goiter, warm and moist skin, muscle weakness and wasting, and onycholysis.

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Symptoms

Symptoms include fatigue, weakness, heat intolerance, weight loss with increased appetite, palpitations, diarrhea, oligomenorrhea or amenorrhea, insomnia, brittle hair, shakiness, difficulty concentrating, irritability, or emotional lability.

◆ Etiology

The etiology of thyrotoxicosis is broadly divided into two categories:

1. **Hyperthyroidism secondary to increased synthesis of hormone**: This condition is associated with high RAIU. Causes include
   A. *Graves disease*: The most common cause of hyperthyroidism.

Key features

- 30 cases per 100,000 persons per year
- Female/male ratio of 10:1
- Peak age of onset is 40 to 60 years
- Autoimmune condition with TSH-receptor antibodies (TSHR Abs, also called thyroid-stimulating immunoglobulins, or TSIs)
- Twenty-five to 30% of patients with Graves disease have clinical evidence of Graves ophthalmopathy

Etiology

Infection, iodide intake, stress, female sex, and genetic predisposition (*HLA-DRB1*, and *HLA-DQB1)* appear to be associated with Graves disease susceptibility.

Signs and Symptoms

Graves disease presents with symptoms typical of thyrotoxicosis: rapid heart rate, palpitation, nervousness, and tremor. It also has some unique features, including ophthalmopathy [a hallmark of Graves disease, with exophthalmos (proptosis), lacrimation, gritty sensation in the eye, photophobia, eye pain, diplopia, or even visual loss], and pretibial myxedema (raised, hyperpigmented violaceous, orange-peel textured papules).

Physical Examination

The thyroid gland generally is diffusely enlarged and smooth. Thyroid nodules may be present. There is widening of the palpebral fissures, tachycardia, hand tremor, proximal muscle weakness, brisk deep tendon reflexes, and warm, velvety skin. Physical findings may include ophthalmopathy, pretibial myxedema, and clubbing of fingers with osteoarthropathy (acropachy).

Differential Diagnosis

- Anxiety disorders
- Chronic autoimmune (Hashimoto's) thyroiditis
- Hyperemesis gravidarum
- Pituitary adenomas

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- Pheochromocytoma struma ovarii
- Papillary thyroid carcinoma
- Cocaine abuse
- Wolff-Parkinson-White syndrome

B. *Toxic multinodular goiter*: Arises in the setting of a long-standing multinodular goiter. It usually affects patients >50 years of age. It occurs when certain nodules develop autonomous function.

C. *Toxic adenomas*: This is a single benign thyroid nodule (adenoma), which becomes autonomous.

D. *Iodine-induced hyperthyroidism*: Though uncommon, iodine-induced hyperthyroidism can develop after an iodine load, for example, following administration of contrast agents used for angiography or CT or iodine-rich drugs such as amiodarone.

E. *TSH-producing pituitary adenomas*

2. **Hyperthyroidism secondary to thyroiditis (with release of preformed hormone into the circulation) or an extrathyroidal source of thyroid hormone**. This condition is associated with low RAIU. Causes include
   - *Thyroiditis:* painless and postpartum thyroiditis, subacute painful or de Quervain thyroiditis (see Chapter 5.4.7)
   - *Exogenous and ectopic:* factitious ingestion; excessive thyroid hormone

◆ Evaluation

Labs

The most reliable screening methods of thyroid function are:

- TSH level: TSH levels are suppressed.
- FT4 and FT3: Elevated FT4 level with suppressed TSH level establishes the diagnosis. If TSH is suppressed, FT4 is normal and FT3 is elevated, this is known as T3 thyrotoxicosis.
- TSI: If elevated, TSI confirms Graves disease; however, it may be undetectable or absent.
- Anti-TPO: Anti-TPO antibodies are usually elevated in Graves disease and usually low or absent in toxic multinodular goiter and toxic adenoma. Anti-TPO may also be elevated in autoimmune-related thyroiditis.

Imaging

- RAI scanning and measurements of iodine uptake: Useful to differentiate overproduction of thyroid hormone from excess release due to thyroiditis or exogenous thyroid hormone ingestion. In Graves disease, the RAI uptake is increased and the uptake is diffusely distributed over the entire gland.
- CT or MRI of the orbits may be necessary in the evaluation of proptosis.

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◆ Treatment Options

The treatment depends on the cause of thyrotoxicosis. If the cause is hyperthyroidism (overproduction of thyroid hormone), definitive treatment options include RAI ablation, antithyroid medications, and surgery, in addition to symptomatic treatment.

Medical

Beta blockers such as propranolol or atenolol provide symptomatic relief in all types of hyperthyroidism. Consider antiinflammatory therapy with NSAIDS (and corticosteroids in refractory cases) in patients with thyrotoxicosis due to thyroiditis.

Antithyroid drugs are used for primary therapy of thyrotoxicosis, for attainment of euthyroidism in preparation for thyroidectomy, and for use in conjunction with radioiodine therapy in selected patients. These inhibit new thyroid hormone production. These include methimazole (Tapazole; Jones Pharma, St. Louis, MO) and propylthiouracil (PTU) if needed for patients with allergy to methimazole (except agranulocytosis), who are pregnant or breast feeding, or have severe thyrotoxicosis or thyroid storm. As primary therapy, use antithyroid drugs alone for 12 to 18 months. Monitor for hepatic dysfunction and agranulocytosis.

^{131}I radiotherapy is primary therapy for thyrotoxicosis due to Graves disease, toxic multinodular goiter, or autonomously functioning thyroid nodules and in patients failing to achieve a remission after a course of antithyroid drugs.

For Graves disease, 10 to 30 mCi RAI therapy is safe and effective. Hypothyroidism occurs over time in ~75% of patients. Permanent hypothyroidism likely; pregnancy and breast-feeding should be avoided until after 6 to 12 months. It may precipitate new or worsened ophthalmopathy, especially in smokers. There is a slight risk of thyroid storm after treatment (see Chapter 5.4.6).

Surgery

Thyroidectomy or subtotal thyroidectomy is usually curative but results in iatrogenic hypothyroidism; risks include potential injury to parathyroids and recurrent laryngeal nerve. Orbital decompression may be necessary in patients with severe Graves ophthalmopathy.

◆ Outcome and Follow-Up

The outcome depends on the cause of hyperthyroidism and the treatment modality. Patients should be routinely followed by an endocrinologist with a history, physical exam, and thyroid function tests.

◆ ICD-9 Codes

242.90 or 242.91	Hyperthyroidism: Cause not specified This includes TSH-mediated, postpartum, destructive, and recurrent

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242.00 or 242.01	Hyperthyroidism: Graves disease; autoimmune stimulatory This includes postpartum, stimulatory
242.20 or 242.21	Hyperthyroidism: Toxic nodular goiter (multinodular)
242.10 or 242.11	Hyperthyroidism: Toxic nodule (uninodular)
242.80 or 242.81	Hyperthyroidism: Excessive thyroid hormone ingestion

Further Reading

Greenspan FS, Strewler GJ. Basic & Clinical Endocrinology. Stamford, CT: Appleton & Lange; 1997
Randolph GW. Surgery of the Thyroid and Parathyroid Glands. Philadelphia, PA: Saunders; 2003
Terris DJ, Gourin CG, eds. Thyroid and Parathyroid Diseases: Medical and Surgical Management. Stuttgart/New York: Thieme; 2009

5.4.5 Hypothyroidism

◆ Key Features

- Hypothyroidism is a common endocrine disorder resulting from deficiency of thyroid hormone.
- Primary hypothyroidism: The thyroid gland produces insufficient amounts of thyroid hormone.
- Secondary hypothyroidism: There is a lack of thyroid hormone secretion due to inadequate secretion of either TSH or TRH.

Hypothyroidism is reduced production of thyroid hormone by the thyroid gland; it can be caused by disease of thyroid gland itself (*primary hypothyroidism*), or by deficiency of TSH due to disease of pituitary or hypothalamus (*secondary and tertiary or central hypothyroidism*). *Subclinical hypothyroidism* is a mild asymptomatic hypothyroidism, in which TSH is mildly elevated but T4 or FT4 levels are normal.

◆ Etiology

Primary Hypothyroidism

Primary hypothyroidism accounts for ~99% of cases. Causes include:

- *Chronic autoimmune (Hashimoto's) thyroiditis:* The most common cause of hypothyroidism in iodine-sufficient regions of the world.
- *Iatrogenic disease:* Thyroidectomy, radioiodine treatment, and external radiotherapy.

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- *Iodine deficiency and iodine excess:* Both can cause hypothyroidism. The most common cause of hypothyroidism worldwide is iodine deficiency.
- *Drugs:* Medications used to treat hyperthyroidism (such as methimazole and PTU) can cause hypothyroidism. Other drugs that can cause hypothyroidism include amiodarone, lithium carbonate, interleukin-2, and interferon alfa.
- *Transient hypothyroidism:* This can occur during the course of several types of thyroiditis, followed by recovery of thyroid function. Some patients who undergo subtotal thyroidectomy become hypothyroid after 4 to 8 weeks, but recover several weeks or months later. This may also follow RAI treatment for Graves disease (see Chapter 5.4.4), when glands are not completely ablated by radioiodine and there is some remaining thyroid tissue.
- *Hypothyroidism in infants and children:* The most common causes of congenital hypothyroidism are agenesis and dysgenesis of the thyroid, but a few are delivered by mothers who were receiving an antithyroid drug for hyperthyroidism. Among children who become hypothyroid later, the most common cause is chronic autoimmune thyroiditis.

Central (Secondary and Tertiary) Hypothyroidism

Central (secondary and tertiary) hypothyroidism can be caused by:

- Most often a pituitary tumor (macroadenomas), pituitary surgery, or irradiation
- Less common causes are head injury, postpartum pituitary necrosis (Sheehan syndrome), pituitary apoplexy (bleeding in a pituitary tumor), hypophysitis, nonpituitary tumors such as craniopharyngiomas and infiltrative diseases

Tissue Resistance to Thyroid Hormone

Tissue resistance to thyroid hormone is rare.

◆ Epidemiology

Primary hypothyroidism is a common disease worldwide in both iodine-deficient and replete regions. The prevalence is 4 to 8% in the general population. It occurs more commonly in women, with a female to male ratio of 10:1. Mean age at diagnosis in women is 60 years with increasing occurrence with advancing age. Mild hypothyroidism may exist in 7 to 15% of the elderly population.

The prevalence of central hypothyroidism in the general population is roughly 0.005%. The sex distribution is equal. It occurs with peaks in childhood and in adults 30 to 60 years old.

◆ Clinical

Deficiency of thyroid hormone causes a generalized slowing of metabolic processes and/or accumulation of matrix glycosaminoglycans in the interstitial spaces of many tissues.

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Signs

Signs include slow movement, slow speech, delayed relaxation of tendon reflexes, bradycardia, dry and coarse and yellowed skin, puffy facies and loss of eyebrows, periorbital edema, and diastolic hypertension.

Symptoms

Symptoms include fatigue, weakness, myalgia, cold intolerance, weight gain, depression, cognitive dysfunction, mental retardation (in infants), constipation, menstrual irregularity, hair loss, and hoarseness.

Differential Diagnosis

- Laboratory confirmation of the diagnosis of hypothyroidism consists of measuring serum TSH and free thyroxine (T4).
  - Primary hypothyroidism is characterized by a high serum TSH concentration and a low serum FT4 concentration. Patients with a high serum TSH concentration and a normal serum FT4 concentration have subclinical hypothyroidism.
  - Central (secondary and tertiary) hypothyroidism is characterized by a low serum T4 concentration and a serum TSH concentration that is not appropriately elevated. In this setting, differentiation must be made between pituitary and hypothalamic disorders.
- Hyperlipidemia occurs with increased frequency in hypothyroidism; as a result, patients with dyslipidemia should be screened for thyroid dysfunction.
- Thyroid function should be measured in all patients with unexplained hyponatremia, as this is another laboratory manifestation of hypothyroidism.
- Thyroid function should also be measured in patients undergoing evaluation for high serum muscle enzyme concentrations or anemia.

◆ Treatment Options

- Start replacement therapy with L-thyroxine (Synthroid, Abbott Laboratories, Abbott Park, IL; Levothroid, Forest Pharmaceuticals, New York, NY; Levoxyl, Jones Pharma, St. Louis, MO).
- The average replacement dose of T4 in adults is ~1.6 μg/kg body weight per day (112 μg/day in a 70-kg adult). The initial dose can be the full anticipated dose in young, healthy patients.
- After initiation of T4 therapy, the patient should be reevaluated and serum FT4 and TSH should be measured in 3 to 6 weeks (depending upon the patient's symptoms), and the dose adjusted accordingly.
- The serum TSH concentration should be maintained between 0.5 and 3 mU/L.
- For monitoring therapy in patients with central hypothyroidism, serum FT4 should be measured and maintained in the upper half of normal range.

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Special treatment situations for hypothyroidism include:

- Elderly patients or those with known or suspected heart disease: These patients should initially be treated with low doses of levothyroxine 25 μg/day. The dose can be increased by 25 μg/day every 3 to 6 weeks until TSH is normal.
- Hypothyroidism during pregnancy: Women require more thyroid hormone during pregnancy.
- Surgical patients: Urgent surgery should not be postponed in hypothyroid patients. On the other hand, hypothyroidism should be corrected before elective surgery. Patients receiving chronic T4 therapy who undergo surgery and are unable to eat for several days need not be given T4 parenterally. If oral intake cannot be resumed in 5 to 7 days, then T4 should be given intravenously. The dose should be ~50% of the patient's usual oral dose.

◆ Complications

Myxedema coma occurs in the setting of prolonged, untreated hypothyroidism. Usually it occurs in the elderly and is precipitated by intercurrent illness, surgery, or narcotic/hypnotic drugs. It is characterized by hypothermia, bradycardia, severe hypotension, seizures, and coma. Myxedema coma holds a poor prognosis with 20% mortality rate. These patients should be promptly treated with IV T4, steroids, and supportive measures.

◆ Outcome and Follow-Up

The outcome depends on the cause of hypothyroidism and the treatment modality. Patients should be routinely followed by an endocrinologist with a history, physical exam, and thyroid function tests.

◆ ICD-9 Codes

245.1	Thyroiditis, subacute
245.2	Thyroiditis, lymphocytic
775.3	Hyperthyroidism: Neonatal
246.8	Abnormal thyroid hormone binding protein
244.9	Hypothyroidism: Any etiology
243	Hypothyroidism, congenital
244.0	Hypothyroidism, postsurgical
244.1	Hypothyroidism, postablative

Further Reading

Terris DJ, Gourin CG, eds. Thyroid and Parathyroid Diseases: Medical and Surgical Management. Stuttgart/New York: Thieme; 2009

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5.4.6 Thyroid Storm

◆ Key Features

- Thyroid storm is a rare and potentially fatal complication of hyperthyroidism.
- Approximately 1 to 2% of patients with hyperthyroidism progress to thyroid storm.
- It usually occurs in patients with untreated or partially treated hyperthyroidism who experience a precipitating event such as surgery, infection, or trauma.

Thyroid storm is a state of severe thyrotoxicosis, in which there is an exaggeration of the manifestations of hyperthyroidism. It is a life-threatening condition.

◆ Etiology

Thyroid storm usually occurs in the setting of undiagnosed or inadequately treated thyrotoxicosis and an added precipitating event such as surgery (both thyroid and nonthyroid), infection, trauma, iodinated contrast dyes, and radioiodine therapy.

◆ Clinical

The condition causes high fever (>102°F or 39°C), tachycardia, cardiac arrhythmia, thromboembolic events, congestive heart failure, confusion, agitation, psychosis, extreme lethargy, coma, diarrhea, nausea and vomiting, and abdominal pain.

◆ Evaluation

The evaluation is mainly based on clinical presentation. Obtaining laboratory data are helpful, but waiting for these laboratories should not delay life-saving treatment.

Labs

TSH-undetectable, elevated FT3 and FT4, anemia, leukocytosis, hyperglycemia, azotemia, hypercalcemia, and abnormal function tests (LFTs).

◆ Treatment Options

The following are the immediate goals:

- To decrease thyroid hormone synthesis: PTU, methimazole
- To decrease thyroid hormone release: Iodine solution

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- Blocking T4-to-T3 conversion: PTU, corticosteroids, propranolol
- Beta-adrenergic blockade: Propranolol

Remaining care is supportive, including treatment for fever with acetaminophen and cooling blankets, IV fluids, nutrition including glucose, thiamine, and treatment of the precipitating event. Consider monitoring the patient in intensive care (ICU).

Further Reading

Terris DJ, Gourin CG, eds. Thyroid and Parathyroid Diseases: Medical and Surgical Management. Stuttgart/New York: Thieme; 2009

5.4.7 Thyroiditis

◆ Key Features

- Thyroiditis is an inflammation of the thyroid gland.
- Hashimoto thyroiditis is the most common type.
- Other forms include postpartum thyroiditis, subacute thyroiditis (de Quervain), silent thyroiditis, drug-induced thyroiditis, radiation-induced thyroiditis, and acute thyroiditis.

Thyroiditis refers to a diverse group of disorders associated with inflammation of the thyroid gland. **Table 5.19** provides the main types of thyroiditis.

◆ Thyroiditis Associated with Pain and Tenderness

Subacute Thyroiditis

Also known as subacute nonsuppurative thyroiditis, de Quervain thyroiditis, giant cell or subacute granulomatous thyroiditis, it is a self-limited inflammatory disorder and is the most common cause of thyroid pain.

Etiology

Subacute thyroiditis is most likely caused by a viral infection (Coxsackievirus, mumps virus, influenza, echovirus, and adenovirus) or postviral inflammatory process. There is a strong association with HLA-B35.

Clinical

Many patients have a history of an upper respiratory infection preceding the onset of thyroiditis. Patients present with fever, malaise, extreme neck

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Table 5.19 Main Types of Thyroiditis

	Hashimoto Thyroiditis	Painless and Postpartum Thyroiditis	Subacute (Painful) Thyroiditis	Suppurative Thyroiditis
Cause	Autoimmune	Autoimmune	Viral	Infectious
Clinical features	Painless, firm, bumpy, symmetric goiter (in 10% atrophied thyroid)	Painless, normal-size thyroid or diffuse goiter	Tender, firm goiter	Tender thyroid mass
Thyroid function	Hypothyroidism	Thyrotoxicosis, hypothyroidism or both	Thyrotoxicosis, hypothyroidism or both	Euthyroidism
TPO antibodies	High titer, persistent	High titer, persistent	Low titer, absent or transient	Absent
ESR	Normal	Normal	High	High
24-hour RAI uptake	Variable	<5%	<5%	Normal

Abbreviations: TPO, thyroid peroxidase; ESR, erythrocyte sedimentation rate; RAI, radioactive iodine.

pain, swelling, or both. Pain may radiate to oropharynx or ears. Up to 50% of patients have symptoms of thyrotoxicosis.

Evaluation

On physical exam, the gland is extremely tender. In most patients, the painful thyrotoxic phase lasts 4 to 6 weeks followed by a hypothyroid phase lasting another 4 to 6 weeks followed by normalization of thyroid functions (recovery phase). Residual hypothyroidism persists in 5% of patients.

Initially, very high ESR, FT3, and FT4 (T4 levels are elevated disproportionally to serum T3) and TSH and RAI uptake and the anti-TPO Abs are usually normal (thyrotoxic phase). As the disease progresses, FT3 and FT4 drop, TSH rises, and symptoms of hypothyroidism are noted. Later RAI uptake rises, reflecting recovery phase.

Treatment Options

Treatment is symptomatic and includes pain control with NSAIDs, and β blockers for control of hyperthyroid symptoms. For more severe thyroid pain, corticosteroids may be considered.

Infectious Thyroiditis

Infectious thyroiditis is also known as suppurative thyroiditis, acute suppurative thyroiditis, pyrogenic thyroiditis, and bacterial thyroiditis.

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Etiology

Infectious thyroiditis is usually caused by *Staphylococcus* and *Streptococcus*, but many other pathogens have also been implicated including mycobacterial, fungal, and Pneumocystis infections. Typically occurs in immunocompromised, elderly, or debilitated patients.

Clinical

Patients present with acute illness, fever, chills, dysphagia, pain in the anterior neck, and swelling. On physical exam, thyroid is tender.

Evaluation

FT3, FT4, and TSH are normal. FNAB with Gram stain and culture of the organism confirms diagnosis. Ultrasonography should be considered to confirm the presence of a single abscess. In most instances, rapid diagnosis and treatment is required.

Treatment Options

The condition is treated with antibiotics, with drainage in the case of an abscess.

Radiation Thyroiditis

May be caused by radioiodine given for treatment of Graves disease (see Chapter 5.4.4). Patients present with mild thyroid pain and tenderness 5 to 10 days after receiving the radioiodine. Symptoms usually subside spontaneously in a few days to one week.

◆ Thyroiditis Not Associated with Pain and Tenderness

Chronic Autoimmune Thyroiditis

Chronic autoimmune thyroiditis is also known as Hashimoto thyroiditis (see also Chapter 5.4.5).

- TPO (thyroid peroxidase) antibodies: High titer, persistent
- ESR: Normal
- RAI uptake: Variable

Painless Thyroiditis

Painless thyroiditis (also known as silent thyroiditis, subacute lymphocytic thyroiditis, and lymphocytic thyroiditis) and postpartum thyroiditis are identical, except that the term "postpartum" is used for patients in whom painless thyroiditis develops within one year after delivery (or may occur even after an abortion). Both account for 4.9% of all types of thyrotoxicosis.

Etiology

Chronic autoimmune thyroiditis is an autoimmune disorder due to lymphocytic infiltration of the thyroid gland.

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Clinical

There is sudden onset of mild transient hyperthyroidism or hypothyroidism, which may be without localized pain. A nontender goiter may be present on physical exam.

Evaluation

The evaluation should include patient's history and the results of thyroid function tests. Anti-TPO is positive in 50% of patients. RAI uptake is low.

Note that postpartum thyroiditis must be differentiated from Graves disease, which also commonly presents in women after delivery. RAI uptake can be obtained in women who are not nursing (low in thyroiditis and high in Graves disease). Also check thyroid-stimulating immunoglobulin (typically positive in Graves). In nursing mothers, thyroid ultrasound with Doppler flow may be helpful in differentiating these two conditions. Hypervascularity typically occurs with Graves disease, whereas there is decreased vascularity in hyperthyroidism associated with postpartum thyroiditis.

Treatment Options

Patients with symptomatic hypothyroidism should be treated with Levothyroxine for 2 to 3 months and then be reevaluated. In up to 25% of women with postpartum thyroiditis, hypothyroidism may be permanent.

Fibrous Thyroiditis

Fibrous thyroiditis (also known as Riedel thyroiditis and invasive thyroiditis) is characterized by extensive fibrosis of the thyroid gland that extends into adjacent tissues.

Etiology

Its etiology is unknown.

Clinical

Patients present with a painless, hard, fixed goiter. Initially euthyroid, but patients may develop hypothyroidism after the gland becomes fibrosed.

Evaluation

- TPO antibodies: usually present
- ESR: normal
- RAI uptake: low or normal

Treatment Options

Treatment includes levothyroxine, corticosteroids, methotrexate, and surgery.

Drug-Induced Thyroiditis

Patients receiving interferon-alfa, interleukin-2, amiodarone, or lithium may develop painless thyroiditis.

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◆ ICD-9 Code

245.9 Thyroiditis

Further Reading

Shindo ML. Introduction to benign thyroid diseases. In: Terris DJ, Gourin CG, eds. Thyroid and Parathyroid Diseases: Medical and Surgical Management. Stuttgart/New York: Thieme; 2009:61–72

5.4.8 Thyroid Cancer

◆ Key Features

- Well-differentiated tumors (papillary and follicular) are highly treatable.
- Undifferentiated tumors (medullary or anaplastic) are aggressive, and have a poorer prognosis.
- Thyroid cancer affects women more than men.
- Solitary nodules are more likely to be malignant in the young and the elderly.

Most thyroid malignancies are well-differentiated cancers that originate from follicular cells (papillary and follicular carcinomas). Thyroid tumors can also originate from the other cell types in the thyroid gland, including the calcitonin-producing C cells (parafollicular cells), lymphocytes, other vascular components and metastases from other organs (**Table 5.20**).

◆ Epidemiology

Thyroid cancer represents the most common endocrine malignancy, annual incidence being ~34,000 cases in the United States. Female/male ratio is ~3:1. Annual incidence increases with age, peaking by the fifth through eighth decades. Thyroid cancer is very rare in children <15 years. Overall, the incidence is on the rise.

◆ Well-Differentiated Thyroid Carcinomas

Well-differentiated thyroid carcinomas originate from the thyroid follicular cells and include papillary and follicular carcinomas, as described below. These respond to TSH, concentrate iodine, and synthesize Tg, albeit less efficiently than normal thyroid tissue. Well-differentiated thyroid carcinomas are two to four times more common in females than males.

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Table 5.20 Overview of Thyroid Cancers

Thyroid Cancer	Type	Incidence	Cell Origin	Subtype	Treatment
Differentiated	Papillary carcinoma	80%	Endodermally derived follicular cell		Surgery and RAI
	Follicular carcinoma	10%	Endodermally derived follicular cell	Hurtle cell	Surgery and RAI
Undifferentiated	Medullary carcinoma	5–10%	Neuroendocrine-derived calcitonin-producing C cell		Surgery
	Anaplastic carcinoma	1–2%	Endodermally derived follicular cell	Small cell carcinoma	Surgery when possible Chemotherapy Radiation therapy
				Giant cell carcinoma	
Others	Lymphoma	Rare	Intrathyroid lymphoid tissue		Chemotherapy Radiation therapy

Abbreviation: RAI, radioactive iodine.

Papillary Thyroid Cancer

Papillary thyroid cancer (PTC) is the most common type of thyroid malignancy. It has a female/male ratio of 2.5:1. The tumor generally grows slowly; the overall prognosis is favorable.

Pathogenesis

Mutations or rearrangements in the genes encoding for the proteins in the MAPK pathway such as ret/PTC, NTRK1, Ras, or BRAF have been implicated in the development and progression of differentiated thyroid cancer. It has been shown that BRAF mutation is associated with more aggressive tumors with higher rates of extrathyroidal extension, lymph node metastases, and recurrence.

Epidemiology

PTC accounts for 80 to 90% of all thyroid cancers. Its peak incidence is between ages 30 to 50 years. Females are more commonly affected than males.

Risk Factors

- History of radiation exposure during childhood such as that received as a treatment of childhood malignancies. Prior to about 1960, external

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radiation to the head and neck was used to treat a wide variety of conditions, including enlarged tonsils, thymus, and even acne.
- History of thyroid cancer in a first-degree relative or a family history of a thyroid cancer syndrome is a risk factor.
- Suggestive signs of a nodule's being malignant include rapid increase in size, fixation to surrounding tissues, new-onset hoarseness or vocal fold paralysis, and the presence of ipsilateral cervical lymphadenopathy.

Clinical Presentation

PTC typically presents as a painless discrete mass in the thyroid gland. These tumors may be multicentric. The tumor generally grows slowly and is late to break through the capsule of the gland. However, once it has become extrathyroidal, it may ultimately become invasive. Nodal metastases appear classically in paratracheal nodes but may be present anywhere in the neck. Bilateral spread is found in 8% of patients. It usually spreads via the lymphatic system. It is not uncommon to find microscopic foci of papillary carcinoma at autopsy or incidentally in a thyroid removed for other indications.

Pathology

Characteristic cytopathologic features seen on FNAB or after surgical resection are diagnostic, including psammoma bodies, cleaved nuclei with an "Orphan Annie" appearance caused by large nucleoli and the formation of papillary structures.

Prognosis

With treatment, overall outcome is generally favorable. However, a small group of patients develop local recurrence and/or distant metastases. Features associated with increased risk of recurrence or mortality include age at diagnosis (age >45 years), size of the primary tumor (>2 cm), and the presence of soft tissue invasion and cervical lymph node or distant metastases.

Variants

PTC has several histologic subtypes, including a follicular variant, an oxyphilic variant, a solid or trabecular variant, and a clear cell variant. Other biologically aggressive variants include a columnar variant, a tall cell variant, a diffuse sclerosing variant, and poorly differentiated carcinoma.

Follicular Thyroid Cancer

Epidemiology

Follicular thyroid cancer (FTC) is more common in the iodine-deficient regions of the world. It tends to occur in an older population, with a peak incidence between ages 40 and 60 years. Similar to most thyroid malignancies, FTC is more common in women (by about a 3:1 ratio).

Risk Factors

- Iodine deficiency may have a role in the pathogenesis as there is a higher prevalence of FTC in iodine-deficient regions of the world, compared with iodine-sufficient regions.

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- FTC is rarely associated with radiation exposure, RET-PTC mutations, or TSH receptor mutations.
- FTC has no association with familial syndromes.
- FTC may be associated with RAS mutations. PAX8-PPAR gamma 1 (a gene rearrangement) can be seen both in follicular adenomas and cancers.

Clinical Presentation

FTC typically presents as a painless solitary, mostly encapsulated nodule in the thyroid. FTC is more aggressive than the PTC and usually spreads by hematogenous routes to bone or lung, and less commonly to brain and liver. It rarely invades lymphatics. The tumor is classified on the basis of degree of invasiveness into minimally invasive (encapsulated) or widely invasive. Metastases are more common with the latter variant.

Diagnosis

FNAB cannot distinguish between follicular adenomas and carcinomas because diagnosis of malignancy requires identification of tumor capsule and/or vascular invasion. Therefore the actual diagnosis of follicular thyroid cancer is made on permanent pathologic evaluation of the thyroid specimen after surgery.

Prognosis

Factors associated with adverse prognosis in FTC include older age, distant metastases, large tumor size, vascular invasion, capsular extension, histologic grade (widely invasive variant, Hürthle cell, insular and trabecular variants), and male gender.

Variants

Clear cell tumor, oxyphilic cell type or Hürthle cell type, and insular carcinoma are FTC variants.

Treatment of Well-Differentiated Thyroid Carcinomas

The primary treatment is surgical, followed by referral to an endocrinologist for medical management. Radioactive iodine remnant ablation treatment may be given if needed; there should be lifelong follow-up and surveillance for recurrence. Surgical treatments include

- Selected papillary carcinomas that are <1 cm in a young patient without a history of radiation exposure may be treated with hemithyroidectomy and isthmusthectomy. All others should be treated with total thyroidectomy and removal of any involved lymph nodes in the central or lateral neck areas. Elective lateral neck dissection is not recommended.
- FTC is treated with a total thyroidectomy.
- Hürthle cell carcinoma is treated with a total thyroidectomy and neck dissection in cases with clinically positive lymph nodes.

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Postoperative Complications of Well-Differentiated Thyroid Carcinomas

Permanent hypoparathyroidism, transient hypoparathyroidism, damage to the recurrent laryngeal nerve (hoarseness), and damage to the superior laryngeal nerve are possible postoperative complications.

Postoperative Management of Well-Differentiated Thyroid Carcinomas

Radioactive Iodine Remnant Ablation

All patients with FTC and those PTC patients who have features associated with increased risk of recurrence or mortality (see Prognosis of Papillary Thyroid Cancer, above) may undergo RAI remnant ablation (RAI treatment or ^{131}I remnant ablation). The RAI is taken up by the residual normal and tumor cells, leading to destruction or death of these cells. This not only reduces future recurrence risk, but also facilitates surveillance for future recurrence. Before the treatment, plasma Tg is measured and a whole body scan is performed after administration of a very small dose of RAI. Several days after the treatment, another whole body scan is obtained (posttreatment scan).

Well-differentiated thyroid cancer has a reduced capacity to concentrate iodine when compared with normal thyroid tissue. An elevated serum TSH stimulates the thyroid cancer cells to take up iodide enough to be detected by RAI imaging and synthesize and secrete Tg. TSH levels can be increased in two ways; one being withdrawal of thyroid hormone therapy. To achieve this, patients should be off of L-thyroxine (LT4) for weeks prior to the whole body scan (because of longer half-life). Alternatively, patients may be started on liothyronine (Cytomel, GlaxoSmithKline, Brentford, Middlesex, UK; or LT3) after surgery, which is continued for 4 weeks and stopped 2 weeks before the whole body scan. This has a shorter half life than L-thyroxine and TSH levels rapidly rise after its discontinuation. Alternatively, the TSH level can also be increased by administration of recombinant human TSH (RhTSH; Thyrogen, Genzyme Corp., Cambridge, MA).

TSH Suppression Therapy

After the diagnostic whole body scan or treatment, the patient is started on L-thyroxine. The goal is to suppress TSH as much as possible. This serves two purposes: maintaining the patient in an euthyroid state and suppressing TSH and growth of any residual thyroid, as most well-differentiated tumors are TSH-responsive.

Long-Term Follow-Up and Surveillance for Recurrence

TSH-stimulated whole body scan is done at 6- or 12-month follow-up. The TSH stimulation is achieved by either thyroid hormone withdrawal or Thyrogen injection (depending on the individual case). Based on the results of this scanning, the decision is then made whether further treatment with RAI is needed.

Tg is a very useful marker for detecting the recurrence of thyroid cancer. Levels are measured periodically both while on thyroid hormone suppression

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and prior to whole body scanning on TSH stimulation. A rising Tg level raises suspicion for recurrence, indicating the need for further treatment through RAI or revision neck surgery.

Imaging, such as ultrasound and PET-CT scanning, is important for detecting recurrences.

Patients need periodic clinical follow-up to monitor for symptoms such as hoarseness, hemoptysis, pain, dysphagia, cough and dyspnea, and recurrent mass, new-onset adenopathy, or a paralyzed vocal fold.

Other Therapies for Well-Differentiated Thyroid Carcinomas

External beam radiation may play a role in the treatment of non–RAI-avid tumors, gross residual tumor, and unresectable disease, as well as in nonsurgical candidates. Other options include clinical trials involving gene therapy and tumor redifferentiation agents.

◆ Other Forms of Thyroid Cancer

Anaplastic Thyroid Cancer

Anaplastic thyroid cancer, a poorly differentiated cancer, accounts for ~2% of all thyroid cancers. Anaplastic thyroid cancer is the most aggressive and lethal of human neoplasms. Median survival is 6 months, despite treatment.

Epidemiology

The annual incidence of anaplastic thyroid cancer is 1 to 2 cases per million. The female/male ratio is 1.2 to 3.1:1. A higher incidence has been reported in areas of endemic goiter. Anaplastic thyroid cancer is responsible for >50% of the 1200 deaths per year attributed to thyroid cancer. It is a disease of the elderly, typically presenting in the sixth or seventh decade of life.

Clinical Presentation

It usually presents with local symptoms caused by a rapidly growing thyroid mass and extensive local invasion. Distant metastases may occur early in the course of the disease to the lungs, liver, bones, and brain. Anaplastic thyroid cancer may arise de novo, but dedifferentiation from long-standing differentiated thyroid carcinoma is also suspected.

Diagnosis

Diagnosis is suggested by cytologic examination of cells obtained by FNAB or core biopsy although it is not always diagnostic, in which case surgical pathology is needed to confirm the diagnosis. Histopathologically, atypical cells are seen that show numerous mitoses and form different patterns. Multinucleate giant cells, spindle-shaped cells, and squamoid cells usually predominate.

Treatment

Surgical treatment may consist of complete resection in selected individuals when possible followed by a combination of chemotherapy and radiotherapy.

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Palliative therapy includes a tracheotomy and chemoradiation. Often, the tumor is not resectable, and surgery consists of a tracheotomy or cricothyroidotomy to prevent airway compromise.

Radiation therapy is indicated preoperatively to increase the tumor resectability rate, postoperatively to enhance the effect of chemotherapy or to alleviate obstruction, but its efficacy must be balanced against its toxicity.

Several chemotherapeutic agents have been used with uniformly suboptimal results. Doxorubicin has been shown to be useful in some patients.

Medullary Thyroid Cancer

Medullary thyroid cancer (MTC) is a neuroendocrine tumor of the C cells (parafollicular cells) of the thyroid gland. Calcitonin is secreted by the tumor and is a useful marker for diagnosis and follow-up. MTC can be sporadic or familial.

Epidemiology

Seventy-five percent of MTCs are sporadic; the remainder are familial. There are three familial patterns of MTC: MEN 2A, MEN 2B, and familial MTC without other features of MEN. MTC is most aggressive in patients with MEN 2B.

Clinical Presentation

Medullary thyroid cancer typically presents as a painful hard nodule or mass in the thyroid gland or as an enlargement of the regional lymph nodes. Sometimes, it comes to medical attention due to a metastatic lesion at a distant site.

Screening

RET protooncogene testing should be obtained in all patients with MTC. If the patient tests positive for the RET mutation, then genetic counseling and testing of family members should be offered. If screening is negative, no investigation of relatives is needed.

Pathology

Characteristic microscopic features are sheets of cells separated by a pink-staining substance that has characteristics of amyloid. Diagnosis can be confirmed by positive immunostaining of the tumor tissue for calcitonin and carcinoembryonic antigen. Preoperatively, patients should also be evaluated for hyperparathyroidism and for pheochromocytoma. Ultrasound or CT of the neck, a chest x-ray, and CT of the chest and upper abdomen should be obtained preoperatively to look for local and regional disease and distant metastases.

Treatment

Treatment is primarily surgical. Total thyroidectomy with removal of regional lymph nodes should be performed after excluding hyperparathyroidism and pheochromocytoma. In patients with MEN, surgery should be performed

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for pheochromocytoma, prior to surgery for MTC. Patients with MEN often undergo prophylactic thyroidectomy in childhood to prevent the development of MTC.

Patients with MTC should receive a total thyroidectomy, a complete central neck dissection, and a lateral modified radical neck procedure on the side of the neck that harbored the tumor. Some recommend bilateral neck dissection. RAI is not a therapeutic option because MTC does not take up iodine; therefore, it is imperative that surgery be thorough.

Postoperative Management

Patients should be started on thyroid hormone replacement (Levothyroxine [LT4]) immediately after surgery. Because the C cells are not stimulated by TSH, there is no need for TSH suppression.

Serum calcitonin and CEA should be checked 6 months after surgery, to detect the presence of residual disease. An elevated basal serum calcitonin 6 or more months after surgery indicates residual disease.

Patients need continued follow-up with periodic physical examination and measurements of serum calcitonin and CEA. Patients with residual disease may benefit from radiotherapy. In cases of advanced metastatic disease untreatable by surgery or radiation, cytotoxic chemotherapy, somatostatin analogues, and interferon or radioimmunotherapy may provide palliation.

Thyroid Lymphoma

Thyroid lymphomas are typical of the NHL type. Diffuse large cell lymphoma is the most common type. Thyroid lymphoma may develop in the setting of longstanding Hashimoto's thyroiditis.

Epidemiology

Thyroid lymphoma represents ~2% of thyroid cancers. It is more common in women and usually presents in the sixth or seventh decade.

Clinical Presentation

Usually thyroid lymphoma presents as a rapidly enlarging goiter. Patients may experience symptoms or signs of compression of the trachea or esophagus, including dysphagia, dyspnea, stridor, hoarseness, and neck pain. On physical examination, the thyroid is usually firm, slightly tender, and is fixed to surrounding structures. It commonly extends substernally. It is not uncommon to see enlarged cervical or supraclavicular lymph nodes. Patients presenting with hoarseness usually have vocal fold paralysis.

In addition, 10% of patients have systemic (B) symptoms of lymphoma, including fever, night sweats, and weight loss (10% of body weight or more). Patients may also present with symptoms and signs of hypothyroidism or hyperthyroidism.

Diagnosis

An ultrasound of the thyroid gland should be performed. FNAB or core biopsy distinguishes thyroid proliferation from epithelial tumors and may differentiate

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lymphoma from chronic thyroiditis; often surgical specimens are required for diagnosis. Pathology, immunohistochemical staining, or flow cytometry may be necessary to establish monoclonality and characterize surface markers, especially to diagnose small cell lymphomas.

Staging includes physical examination, CBC, serum LDH, B2 microglobulin measurements, liver function tests, bone marrow biopsy, and CT of the neck, thorax, abdomen, and pelvis, as well as an FDG-PET scan.

Treatment

Surgery is not the primary treatment and is typically used for diagnostic biopsy and surgical airway only. Treatment depends on extent of disease. If lymphoma is disseminated, chemotherapy is given. If disease is confined to the neck, treatment is guided by the histologic features of the lymphoma. Patients with large cell lymphoma are treated with chemotherapy with or without radiation. For patients with localized extranodal marginal zone lymphoma of the thyroid, follicular lymphoma and small cell lymphoma radiotherapy alone may be adequate.

◆ ICD-9 Code

193 Malignant neoplasm of thyroid gland

◆ Staging of Thyroid Cancer

TNM and Stage Groupings

According to the American Joint Committee on Cancer (AJCC), the TNM stage groupings for papillary and follicular carcinomas and variants are stratified by age into patients <45 years of age and patients ≥ 45 years. Tumor size and lymph node status are also considered in the TNM classification.

All categories may be subdivided into (a) solitary tumor or (b) multifocal tumor. With multifocal tumors, the largest one is used for classification. The lymph nodes must be specifically identified to classify regional node involvement.

Primary Tumor (pT)

pTX: Primary tumor cannot be assessed

pT0: No evidence of primary tumor

pT1: Tumor size ≤ 2 cm, limited to the thyroid

pT1a: Tumor ≤ 1 cm in greatest dimension limited to the thyroid

pT1b: Tumor >1 cm but not >2 cm in greatest dimension, limited to the thyroid

pT2: Tumor >2 cm, but not >4 cm, limited to the thyroid

pT3: Tumor >4 cm limited to the thyroid or any tumor with minimal extrathyroid extension (e.g., extension to the sternothyroid muscle or perithyroid soft tissues)

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pT4a: Moderately advanced disease. Tumor of any size extending beyond the thyroid capsule to invade the subcutaneous soft tissues, the larynx, the trachea, the esophagus, or the recurrent laryngeal nerve

pT4b: Very advanced disease. Tumor invades the prevertebral fascia or encases the carotid artery or mediastinal vessels

Note: There is no category of carcinoma in situ (pTis) relative to carcinomas of thyroid gland.

Regional Lymph Nodes (N)

pNX: Cannot be assessed

pN0: No regional lymph node metastasis

pN1a: Nodal metastases to level VI (pretracheal, paratracheal, and prelaryngeal/Delphian) lymph nodes

pN1b: Metastases to unilateral, bilateral, or contralateral cervical (levels I, II, III, IV, V) or retropharyngeal or superior mediastinal lymph nodes (level VII)

Distant Metastasis (pM)

pM0: No distant metastasis

pM1: Distant metastasis

All anaplastic carcinomas are considered T4 tumors.

T4a: Intrathyroidal anaplastic carcinoma – surgically resectable

T4b: Extrathyroidal anaplastic carcinoma – surgically unresectable

American Joint Committee on Cancer Stage Groupings of Follicular Thyroid Cancer and Papillary Thyroid Cancer

Patients Younger than 45 Years

Stage I

Any T any N M0

Stage II

Any T any N M1

Patients 45 Years and Older

Stage I

T1 N0 M0

Stage II

T2 N0 M0

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Stage III
T3 N0 M0
T1 N1a M0
T2 N1a M0
T3 N1a M0

Stage IVA
T4a N0 M0
T4a N1a M0
T1 N1b M0
T2 N1b M0
T3 N1b M0
T4a N1b M0

Stage IVB
T4b any N M0

Stage IVC
Any T any N M1

American Joint Committee on Cancer Stage Groupings of Medullary Carcinoma

All Age Groups

Stage I
T1 N0 M0

Stage II
T2 N0 M0
T3 N0 M0

Stage III
T1 N1a M0
T2 N1a M0
T3 N1a M0

Stage IVA
T4a N0 M0
T4a N1a M0
T1 N1b M0
T2 N1b M0
T3 N1b M0
T4a N1b M0

Stage IVB
T4b any N M0

Stage IVC
Any T any N M1

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American Joint Committee on Cancer Stage Groupings of Undifferentiated (Anaplastic) Carcinoma

All anaplastic carcinomas are considered Stage IV.

Stage IVA
T4a any N M0

Stage IVB
T4b any N M0

Stage IVC
Any T any N M1

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LiVolsi VA. Pathology of thyroid disease. In: Falk SA: Thyroid Disease: Endocrinology, Surgery, Nuclear Medicine, and Radiotherapy. Philadelphia, PA: Lippincott-Raven; 1997:127–175

5.4.9 Embryology, Anatomy, and Physiology of the Parathyroid Glands

There are four parathyroid glands (in two pairs), usually close to the upper and lower poles of the thyroid lobe.

◆ Embryology

The upper pair of parathyroid glands arises from the fourth branchial cleft and descends with the thyroid gland, usually at the cricothyroid junction. The lower pair arises from the third branchial cleft and descends with the thymus; the location of the lower parathyroids may be variable. Ectopic parathyroids may be found anywhere along the pathway of descent of the branchial pouches. The (lower) parathyroid glands have been described in the carotid sheath, anterior mediastinum, and intrathyroid.

◆ Anatomy

Grossly the parathyroid glands are yellow-brown, weighing 25 to 40 mg per gland. They each measure on average ~6 mm in length, and from 3 to 4 mm in breadth, and usually present the appearance of flattened oval disks.

◆ Histology

Parathyroid glands are composed primarily of chief cells and fat with a thin fibrous capsule dividing the gland into lobules; the glands may have a pseudofollicle pattern resembling thyroid follicles.

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◆ Blood Supply

The arterial supply to the parathyroid glands gland originates from the superior and inferior parathyroid arteries, both of which usually arise from the inferior thyroid artery. Parathyroid glands do not receive nourishment from the adjacent thyroid gland.

◆ Physiology

Parathyroid glands help maintain serum calcium and phosphorus homeostasis in conjunction with calcitonin and vitamin D by secreting parathyroid hormone (parathyrin or PTH). PTH contains 84 amino acids that are cleaved in the liver to its active form, producing a biologically active N-terminal segment. Secretion of PTH is stimulated by low levels of ionized calcium and suppressed by high levels of ionized calcium. PTH binding to receptor sites results in cAMP second messenger system activation. Target end organs include the kidneys, skeletal bone, and the intestine. The half-life of PTH is a few minutes, which is clinically utilized to detect falling levels following a parathyroidectomy.

Further Reading

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Smith JA, Stack BC. Pathophysiology of the parathyroid glands. In: Terris DJ, Gourin CG, eds. Thyroid and Parathyroid Diseases: Medical and Surgical Management. Stuttgart/New York: Thieme; 2009: 184–196

5.4.10 Hyperparathyroidism

◆ Key Features

- Hyperparathyroidism is caused by an excessive secretion of parathyroid hormone (PTH).
- Hyperparathyroidism is usually subdivided into primary, secondary, and tertiary hyperparathyroidism.
- Hyperparathyroidism results in elevated levels of plasma calcium by increasing the release of calcium and phosphate from bone matrix, increasing calcium reabsorption by the kidney, and increasing intestinal absorption of calcium.

Hyperparathyroidism (HPT) refers to the increased production of PTH by the parathyroid glands. There are three types of hyperparathyroidism: primary, secondary, and tertiary, which are described below.

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◆ Primary Hyperparathyroidism

Etiology

Eighty to 90% of hyperparathyroidism patients have single parathyroid adenomas and up to 5% may have double adenomas. Diffuse hyperplasia of all four glands accounts for 6% cases of primary hyperparathyroidism (PHP). One to 2% of cases are due to parathyroid carcinoma.

The diffuse hyperplasia of all four glands can be sporadic or familial; occurring usually as a part of the MEN type 1 or 2 syndromes (**Table 5.21**). Other familial conditions associated with all four gland hyperplasia include familial hyperparathyroidism-jaw tumor syndrome and familial isolated hyperparathyroidism.

Familial hypocalciuric hypercalcemia is a rare, autosomal dominant disorder that should be included in the differential diagnosis of PHP. It is caused by mutations of the calcium sensing receptor.

Epidemiology

Primary hyperparathyroidism can occur at any age, but the great majority of cases occur over the age of 45 years. Women are affected twice as often as men.

Clinical

Primary hyperparathyroidism is most often detected incidentally by routine biochemical screening. Most patients are either asymptomatic or experience subtle and vague symptoms such as fatigue, depression, difficulty in concentration, and generalized weakness.

Patients with primary hyperparathyroidism may present with classic symptoms and signs of hypercalcemia, as recalled by the famed mnemonic "painful bones, renal stones, abdominal groans, and psychic moans":

- Bones: The classic bone disease of PHP is osteitis fibrosa cystic, which is now rare in the United States. It presents with bone pain and/or

Table 5.21 Multiple Endocrine Neoplasia (MEN)

	MEN 1	MEN 2A	MEN 2B
Inheritance	AD	AD	AD
Clinical manifestations	PHP (95%) Pituitary pumors Enteropancreatic tumors	PHP (30%) MTC Pheochromocytoma Hirschsprung disease Cutaneous lichen Amyloidosis	PHP (rare) MTC Pheochromocytoma Mucosal neuromas Intestinal Ganglioneuromatosis Marfanoid habitus

Abbreviations: AD, autosomal dominant; MTC, medullary thyroid cancer; PHP, primary hyperparathyroidism.

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pathologic fractures. A more common skeletal manifestation of PHP is a decrease in the bone mineral density (osteopenia), preferentially at the cortical sites (forearm and hip). The cause is prolonged PTH excess.

- Kidneys: Nephrolithiasis occurs in ~15 to 20% of patients with primarily hyperparathyroidism. The cause is prolonged PTH excess. Hypercalciuria and chronic renal failure can also occur.
- Gastrointestinal: Hypercalcemia associated symptoms include anorexia, nausea, vomiting, constipation, and peptic ulcer disease.
- Psychiatric and neurocognitive: Patients may have depressed mode, lethargy, emotional lability, and decreased cognitive function.

Often the presentation is much more subtle. Patients with familial hypocalciuric hypercalcemia are asymptomatic.

Parathyroid carcinomas are rare. Tumors may be large, palpable, and up to 6 cm in diameter.

Evaluation

Patients suspected of having PHP should be referred to an endocrinologist for work-up and confirmation of the diagnosis.

Imaging

- Sestamibi scan: ^{99m}Tc sestamibi localizes to the mitochondria of parathyroid cells, which are rich in mitochondria. After injection, an image is taken at 10 to 15 minutes and again at 2 to 3 hours. The late phase is useful for the detection of adenomas. It has a sensitivity as high as 100%, specificity is at 90%.
- Single proton emission computerized tomography (SPECT) scan: The advantage of SPECT is that it portrays images in 3D and therefore may add localization to mediastinal lesions or ectopic adenomas in the carotid sheath.
- Ultrasound: Its advantage is ease of performance, low cost, and no radiation. Disadvantages include difficulty of localization of nonstandard locations and the potential of confusion with thyroid abnormalities, and interoperator variability.
- CT scan: CT may be useful for the detection of ectopic parathyroid; it has an up to 50% false-positive rate.
- MRI: Like the CT scan, MRI is useful for ectopic parathyroid but has a false-positive.
- Selective venous sampling: The veins draining the parathyroid region can be sampled. A two-fold gradient between PTH levels in the sampled vein vs the peripheral vein levels can help localize the parathyroid adenoma.
- Intraoperatively, localization, dye, or radioactive tracers may be used.

Labs

PTH levels can be elevated or normal. Serum calcium levels are generally elevated; however, 10 to 20% of patients with primary hyperparathyroidism

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have normal serum calcium concentrations (normocalcemic PHP). In some patients this may be due to coexisting vitamin D [25(OH)D] deficiency. Low serum phosphorus, increased 24-hour urinary calcium excretion, elevated serum 1,25-dihydroxyvitamin D may be seen. Blood urea nitrogen and creatinine should be checked as well.

It is important to rule out familial hypocalciuric hypercalcemia because usually the course of this disease is benign and parathyroidectomy is not indicated. Past medical history should be carefully obtained as these patients are asymptomatic and have a history of elevated calcium levels since childhood. Their family members may have hypercalcemia as well. Their 24-hour urinary calcium excretion is low.

Secondary hyperparathyroidism should also be ruled out (either from a renal source or from decreased calcium absorption/intake or vitamin D deficiency).

Treatment Options

Medical

Medical treatment is indicated in patients who do not meet the criteria for surgery, refuse surgery, or are poor surgical candidates. This includes adequate hydration and moderate calcium intake ~1000 mg/day. The calcium levels, renal function and a dual-energy x-ray absorptiometry (DEXA) scan are periodically followed under the care of an endocrinologist. Medications used in the treatment of osteoporosis, such as bisphosphonates, may be useful. Calcimimetics (e.g., cinacalcet hydrochloride; Sensipar, Amgen, Inc., Thousand Oaks, CA) lower PTH and calcium levels, but are not yet FDA-approved for PHP.

Surgical

Surgery is curative and is indicated in all cases with symptomatic disease. Following are the indications of surgery in asymptomatic patients: (1) serum calcium >1.0 mg/dL above normal, (2) creatinine clearance reduced to <60 mL/min, (3) 24-hour urine calcium >400 mg/dL, (4) age <50 years, (5) DEXA bone density scan with T score below −2.5 at any site, and (6) patient requests surgery. After surgery, 90% of patients' calcium levels normalize. Surgery consists of removal of the adenoma. Preoperative imaging localization allows for guided and minimally invasive parathyroidectomy in most cases.

Occasionally parathyroid adenomas may be found in an ectopic site. Common ectopic sites include the thymus/mediastinum, transesophageal groove, retroesophageal, intrathyroidal, and the carotid sheath.

Surgery in Multigland Disease

1. Multigland diffuse hyperplasia does not localize with sestamibi. Remove enlarged glands and do an intraoperative parathyroid hormone assay.
2. Non-MEN familial hyperparathyroidism: Patients tend to be younger and more likely to have multiglandular disease and persistent or recurrent hyperparathyroidism after surgery. More aggressive than MEN 2a,

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this disease is more likely to be associated with profound hypercalcemia or hypercalcemic crisis. In a bilateral neck exploration, identify all four glands; perform a subtotal or total parathyroidectomy with thymectomy and autotransplantation of gland as needed.

3. MEN I: Patients should have a bilateral neck exploration with identification of all four glands. It is controversial if a subtotal or total parathyroidectomy should be performed.
4. MEN 2a: Pheochromocytoma should be ruled out prior to surgery. After a bilateral neck exploration, with ID of all four glands – remove only the enlarged glands.
5. Renal failure-induced hyperplasia occurs if medical therapy fails. After a bilateral exploration, a subtotal or total parathyroidectomy with autotransplantation can be done.

◆ Secondary Hyperparathyroidism

Secondary hyperparathyroidism is a normal elevation in the PTH level secondary to renal failure, hypocalcemia, hyperphosphatemia, and vitamin D deficiency. Treatment consists of treating the initial cause of the secondary hyperparathyroidism.

◆ Tertiary Hyperparathyroidism

Tertiary hyperparathyroidism is due to prolonged hypercalcemia that causes parathyroid gland hyperplasia. Autonomous oversecretion of PTH by the parathyroid glands results in hypercalcemia. Most commonly seen after renal transplant for end-stage renal disease that is associated with severe secondary hyperparathyroidism. Treatment is usually surgical.

◆ ICD-9 Codes

252.0	Hyperparathyroidism
252.07	Primary hyperparathyroidism
252.02	Secondary hyperparathyroidism, nonrenal
252.08	Other hyperparathyroidism

Further Reading

Pellitteri PK. Surgical management of hyperparathyroidism. In: Terris DJ, Gourin CG, eds. Thyroid and Parathyroid Diseases: Medical and Surgical Management. Stuttgart/New York: Thieme; 2009:197–225

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5.4.11 Hypoparathyroidism

◆ Key Features

- Hypoparathyroidism is caused by low circulating levels of parathyroid hormone or insensitivity to its action.
- The most common causes are iatrogenic (surgery) and autoimmunity.

◆ Etiology

- Iatrogenic (surgical): This is the most common cause of hypoparathyroidism. This may occur after surgery on the neck (thyroidectomy, parathyroidectomy, or neck dissection). It maybe transient with recovery within days to weeks, or it may be permanent. It usually occurs as a result of manipulation of blood supply to the parathyroid glands during surgery or injury to or removal of one or more parathyroid glands.
- Hypoparathyroidism may also occur in the setting of "hungry bone syndrome," following surgery. In this case, bones readily take up calcium and phosphate. This is usually associated with severe preoperative hyperparathyroid bone disease.
- Autoimmunity: This occurs secondary to immune-mediated destruction of parathyroid glands. This may be acquired or familial.
- Hypoparathyroidism may occur due to abnormal development of the parathyroid glands. This is usually associated with DiGeorge syndrome, which is a congenital abnormality of the third and fourth branchial pouches and results in the absence of parathyroid glands and thymus.
- Idiopathic hypoparathyroidism is more common in females and has been associated with anti-PTH antibodies.
- Hypoparathyroidism may result from the destruction of parathyroid glands due to irradiation or infiltrative diseases (such as metastatic cancer and granulomatous disease) or storage diseases (such as Wilson's disease and hemochromatosis).

◆ Clinical

Signs and Symptoms

Patients present with symptoms and signs of hypocalcemia (see Chapter 5.4.12).

Differential Diagnosis

Hypoparathyroidism is usually associated with hypocalcemia (low serum calcium levels), hyperphosphatemia (high phosphate levels), and low or

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undetectable PTH levels. Note that, in contrast, "hungry bone syndrome" is associated with hypocalcemia, and hypophosphatemia (low phosphate levels).

◆ Treatment Options

For management of acute hypocalcemia, see Chapter 5.4.12. Patients with hypoparathyroidism may require lifelong supplementation with calcium and vitamin D, except for those with transient hypoparathyroidism. The goal of treatment is to maintain serum calcium levels in the low normal range. Patients are given 1.5 to 2 g of elemental calcium in divided doses daily with meals to maximize absorption. Calcitriol (Rocaltrol; Roche Pharmaceuticals, Nutley, NJ) is usually given at a starting dose of 0.25 to 0.5 μg orally daily and titrated as needed. Renal function and calcium levels should be closely monitored in these patients.

Recombinant PTH (Forteo; Eli Lilly & Co., Indianapolis, IN) may be another treatment option. It is currently under investigation.

◆ ICD-9 Code

252.0 Hypoparathyroidism

Further Reading

Terris DJ, Gourin CG, eds. Thyroid and Parathyroid Diseases: Medical and Surgical Management. Stuttgart/New York: Thieme; 2009

5.4.12 Calcium Disorders

◆ Key Features

- Hypercalcemia can result when too much calcium enters the extracellular fluid or when there is insufficient calcium excretion from the kidneys.
- Hypocalcemia is less frequent than hypercalcemia.
- Hypocalcemia occurs in patients with renal failure, vitamin D deficiency, magnesium deficiency, acute pancreatitis, and with hypoparathyroidism and pseudohypoparathyroidism.

About 99% of the body's calcium is found in the bones. The remaining 1% is found in the extracellular fluid. Of this 1%, ~40% of calcium is bound to albumin, and 50% is in the free (unbound, active, or ionized) form. A basic

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metabolic panel and a comprehensive metabolic panel measure the total calcium levels (bound plus unbound), although it is the free (unbound) form that is most important.

◆ Control of Calcium Metabolism

Calcium is absorbed from the gut, stored in the bone, and excreted by the kidneys. Calcium levels are affected by the following:

- PTH: Decreased calcium levels lead to an increase in PTH, which in turn causes a release of bone calcium stores and decreased renal excretion of calcium.
- Vitamin D: PTH stimulates conversion of 25(OH)D into its active form [1,25-(OH)2D], which in turn increases calcium and phosphate absorption from the gut.
- Calcitonin: Calcitonin is synthesized in the C cells of the thyroid and causes a decrease in plasma calcium and phosphate levels.

◆ Hypocalcemia

Etiology

- Hypoparathyroidism: The most common cause is iatrogenic (surgery); see Chapter 5.4.11.
- Pseudohypoparathyroidism: An inherited disorder that is caused by resistance to PTH by target organs. Patients present in childhood. It is associated with hypocalcemia, hyperphosphatemia, and elevated PTH levels.
- Renal failure
- Vitamin D deficiency
- Hypomagnesemia
- Acute pancreatitis
- Hyperphosphatemia

Clinical

- Neuromuscular irritability: Patients may present with tingling, paresthesias in fingers and periorally, tetany, carpopedal spasm, seizures, irritability, and confusion.
  - Chvostek's sign: Gentle tapping over the facial nerve causes twitching of facial muscles
  - Trousseau's sign: Carpopedal spasm after blood pressure cuff is inflated above patient's systolic blood pressure for 3 minutes
- Cardiac manifestations: electrocardiogram changes—prolonged QT interval, heart failure, and arrhythmias.
- Other manifestations include subcapsular cataracts, dry flaky skin, and brittle nails.

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Evaluation

Serum albumin concentration should be measured in patients with hypocalcemia. When albumin is low, the calcium level should be corrected (adjusted) for the level of albumin. Alternatively, a serum ionized calcium level should be obtained. Additionally, phosphate levels, intact PTH levels, magnesium levels, vitamin D levels, and creatinine levels should also be obtained.

Treatment Options

Treatment should be tailored to treat the underlying cause. Endocrine consult (or referral) should be considered.

- Acute hypocalcemia: Patients with symptomatic tetany, seizures, or stridor need immediate correction of hypocalcemia. Calcium is given as a slow IV bolus over 10 to 15 minutes because a rapid infusion may cause cardiac dysfunction. The patient should be in a monitored setting (telemetry). This may be followed by a slow calcium infusion over several hours. Serum calcium levels should be closely monitored during the IV infusion.
- Chronic hypocalcemia: Calcium and vitamin D supplementation are the mainstays of treatment. Daily divided doses of 1.5 to 2 g of elemental calcium are given with meals to maximize absorption. Vitamin D supplementation is given as ergocalciferol (vitamin D_2), which has a long duration of action, or calcitriol (Rocaltrol), which has a short duration of action.

◆ Hypercalcemia

Etiology

- Hyperparathyroidism is the most common cause of hypercalcemia; see Chapter 5.4.10.
- Malignancy is the second most common cause of hyperparathyroidism. Commonly associated with solid tumors (e.g., breast, lung, ovary) and hematologic cancers (e.g., myelomas and lymphomas).
- Granulomatous disorders (such as sarcoidosis, tuberculosis)
- Milk alkali syndrome
- Paget disease
- MEN syndromes
- Thyrotoxicosis
- Medications: thiazides, vitamin A and D intoxication, lithium
- Immobilization

Clinical

See **Table 5.22**.

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Table 5.22 Signs and Symptoms of Hypercalcemia

Bones	Bone pain, muscle weakness, osteopenia/osteoporosis
Renal	Polyuria, polydipsia, nephrolithiasis, nephrocalcinosis, acute and chronic renal insufficiency
Gastrointestinal	Anorexia, nausea, vomiting, constipation, pancreatitis, peptic ulcer disease
Neurologic-psychiatric	Decreased concentration, confusion, fatigue, depression
Heart	Shortening of the QT interval, bradycardia, and hypertension

Evaluation

Labs should include intact PTH level, ionized calcium level, and vitamin D. If PTH levels are low and malignancy is suspected, PTHrP levels should be obtained. Other lab values should be obtained to diagnose the suspected underlying etiology.

Treatment Options

Treatment should focus on the underlying etiology of hypercalcemia.

- Acute severe hypercalcemia: The volume status should be assessed and the patient should be aggressively rehydrated with IV normal saline. Use with caution in patients with heart failure or renal failure to avoid fluid overload. The serum electrolytes, calcium, and magnesium levels should be monitored closely. Once the fluid deficit has been corrected, consider adding furosemide (loop diuretic). Bisphosphonates can be given as these inhibit bone resorption by osteoclasts and are effective in lowering calcium levels to the normal range within 2 to 5 days. Calcitonin rapidly lowers serum calcium and can be administered subcutaneously as an adjunct.
- Chronic severe hypercalcemia: The cause should be identified and treated accordingly (see Chapter 5.4.10). Bisphosphonates, gallium nitrate, or glucocorticoids can be used in the treatment of malignancy associated hypercalcemia. Hypercalcemia associated with granulomatous diseases is also treated with glucocorticoids. If hypercalcemia is caused by medication overdose, then that medication should be stopped.

◆ ICD-9 Codes

275.41 Hypocalcemia
275.42 Hypercalcemia

Further Reading

Terris DJ, Gourin CG, eds. Thyroid and Parathyroid Diseases: Medical and Surgical Management. Stuttgart/New York: Thieme; 2009

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6 Pediatric Otolaryngology

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6.1 Pediatric Airway Evaluation and Management

◆ Key Features

- The airway is relatively narrower and more tenuous in children.
- The potential for airway emergency is high.
- Many conditions causing respiratory distress in infants resolve spontaneously with growth.

The pediatric airway is proportionally smaller than the adult: the tongue is relatively larger and more anterior, the soft palate descends lower, the adenoid is larger, the epiglottis is omega shaped, larger, and more acutely angled toward the glottis; the cricoid ring is narrower, the trachea is shorter and narrower, the surrounding soft tissue is looser, and cartilaginous structures are less rigid. Thus the pediatric airway is more prone to compromise by infection, inflammation, neoplasia, and normal breathing.

Foreign body aspiration may be a life-threatening emergency. An aspirated solid or semisolid object may lodge in the airway. If the object is large enough to cause nearly complete obstruction of the airway, asphyxia may rapidly cause death. Infants are at risk for foreign body aspiration because of their tendency to put everything in their mouths and because of immature chewing. Children may be asymptomatic. If present, physical findings may include stridor, fixed wheeze, or diminished breath sounds. If obstruction is severe, cyanosis may occur.

◆ Clinical

Signs and Symptoms

- Stridor: Harsh high-pitched sound of turbulent airflow past partial obstruction in upper airway
  - Inspiratory stridor signifies supraglottic obstruction.
  - Biphasic stridor signifies glottic or subglottic obstruction.
  - Expiratory stridor signifies tracheal or large bronchial compression.
- Stertor: Low-pitched, snorting sound resulting from partial nasal/nasopharyngeal/hypopharyngeal obstruction
- Wheezing: A continuous whistling or musical sound on expiration from a small bronchiole constriction

For subjective assessment of respiratory distress, see **Table 6.1**. Indications for intubation for airway compromise include PaO_2 <60 mm Hg with FiO_2 >0.6 (without cyanotic heart disease), $PaCO_2$ >50% (acute, unresponsive to other intervention), actual or impending obstruction, neuromuscular

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Table 6.1 Subjective Assessment of Respiratory Distress

	None	Mild	Moderate	Severe
Stridor	None	Mild	Moderate at rest	Severe on inspiration and expiration or none with markedly decreased air entry
Retractions	None	Mild	Moderate at rest	Severe, marked use of accessory muscles
Color	Normal	Normal	Normal	Dusky or cyanotic
Level of consciousness	Normal	Restless when disturbed	Anxious; agitated; restless when disturbed	Lethargic, depressed

Source: Davis HW, Dartner JC, Galvis AG, et al. Acute upper airway obstruction: croup and epiglottitis. Pediatr Clin North Am 1981;28(4):859. Reprinted with permission from Elsevier.

weakness (maximum negative inspiratory pressure over -20 cm H_2O, vital capacity $<$12–15 mL/kg), and an absent cough/gag reflex.

Differential Diagnosis

- Supralaryngeal
  - Adenoid hypertrophy
  - Macroglossia
  - Mass (nasopharyngeal, base of tongue)
  - Choanal atresia
  - Foreign body
  - Cellulitis
  - Neck/pharyngeal abscess
- Laryngeal
  - Laryngomalacia (most common overall cause of pediatric stridor)
  - Vocal fold paralysis (unilateral often is iatrogenic or trauma related, bilateral is often due to central nervous system [CNS] lesion or dysfunction)
  - Laryngeal web
  - Subglottic stenosis or hemangioma
  - Papillomata
  - Laryngeal cleft
  - Viral croup
  - Epiglottitis
  - Gastric reflux
- Tracheobronchial
  - Tracheomalacia
  - Bronchomalacia

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- Stenosis
- Vascular ring
- Airway foreign body
- Bronchitis
- Bronchiolitis
- Tracheoesophageal fistula (TEF)

◆ Evaluation

History

A thorough history of respiratory distress (cyanosis, apnea, dyspnea, retractions, grunting) including age of onset, frequency, degree, and rate of progression, ameliorating and exacerbating factors (including positional), as well as feeding difficulties and fevers should be elicited. Difficult delivery, history of prematurity, and postpartum complications (asphyxia, duration of intubation) should be noted.

Physical Exam

On the physical exam, note respiratory rate, nasal flaring, intercostal and supraclavicular retraction, gasping, or respiratory fatigue. Examine the mouth using a tongue blade, unless epiglottitis is suspected. Auscultate the chest and neck. Note change in breathing when positioning child upright, supine, prone, and on each side. Visualize airway with flexible laryngoscope, unless epiglottitis is suspected. More severe cases may require bronchoscopy and possibly esophagoscopy. Bronchoscopy (rigid or flexible) may be both diagnostic and therapeutic (see A1 in Appendix A).

Imaging

Upright anteroposterior (AP) and lateral soft tissue x-rays of the airway (ideally on full inspiration with head in extension) best evaluate the upper airway. Additionally, AP and lateral chest x-rays can detect extrinsic compression or evaluate for evidence of airway foreign body. If child has feeding difficulties, obtain a barium esophagram. Magnetic resonance imaging (MRI) with MR angiography (MRA) is a good alternative to angiography for vascular malformations. Obtain gastric emptying scans and esophageal pH monitoring if reflux is suspected.

For foreign body aspiration, employ posteroanterior inspiratory and expiratory chest radiography to look for hyperinflation, computed tomography (CT), or fluoroscopy. Studies may be false-negative; if index of suspicion remains high, bronchoscopy in the OR is indicated.

Other Tests

Although pulmonary function testing with a flow-volume loop can help distinguish inspiratory versus expiratory as well as intrathoracic versus extrathoracic obstruction, the test requires a cooperative subject and is not

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often feasible in children <6 years. Polysomnography is very helpful in the evaluation of possible pediatric sleep-related respiratory disorders; a differentiation between obstructive versus central apnea can be obtained. This is highly recommended in children with a history of neurologic disorder.

◆ Treatment Options

Acute choking, with respiratory failure associated with airway foreign body obstruction, may be successfully treated at the scene using standard first aid techniques such as the Heimlich maneuver, back blows, and abdominal thrusts. Even in less urgent settings, expeditious removal of airway foreign bodies is recommended and a workup may be performed.

Medical

Definitive management will, of course, depend upon the specific diagnosis. But in general terms, for the child with airway compromise continuous monitoring with pulse oximetry is necessary. Supplemental humidified oxygen, racemic epinephrine, or heliox may be implemented. Systemic steroids are often employed.

Reflux medications such as lansoprazole (Prevacid, Takeda Pharmaceutical Company, Deerfield, IL), to decrease gastric acid production, as well as metoclopramide (Reglan, Schwarz Pharma, Inc., Milwaukee, WI), to promote gastric emptying, can help with reflux irritation of the airway. Upper respiratory infections should be empirically treated as an inpatient with ceftriaxone 75 mg/kg/day intravenously (IV), or comparable antibiotic covering *Streptococcus pneumoniae*, *Streptococcus pyogenes*, and *Haemophilus influenzae*.

Surgical

Again, definitive management will, of course, depend upon the specific diagnosis. In cases of severe compromise, pediatric tracheotomy may be necessary. In laryngomalacia, redundant laryngeal tissue can be excised, and aryepiglottic folds can be divided using endoscopic scissors or CO_2 laser. However, many cases of laryngomalacia may be managed with observation (see Chapter 6.2). Subglottic stenosis can be dilated, or a cricoid split or cartilage graft reconstruction performed (see Chapter 6.7). Obstructing masses should be removed, if possible. Subglottic hemangioma management is discussed in Chapter 6.13.

◆ Outcome and Follow-Up

The child should be monitored closely overnight in case of bleeding or edema compromising airway. Oxygen saturation should be monitored. Specific conditions are discussed in detail in the following chapters.

Outcome depends on the diagnosis and management. Child can be followed with the usual well-child checks, and immunizations should be kept up to date.

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◆ ICD-9 Codes

786.1 Stridor
748.3 Other anomalies of larynx, trachea, and bronchus

Further Reading

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Wetmore RF, Muntz HR, McGill TJ, et al. Pediatric Otolaryngology: Principles and Practice Pathways. Stuttgart/New York: Thieme; 2000

6.2 Laryngomalacia

◆ Key Features

- Most common cause of stridor in infants (accounts for ~75% of infantile stridor).
- Often self-limited; most patients are symptom-free by 12 to 24 months of age.
- Treatment for 90% of cases is expectant observation.
- Its etiology is unknown.

Laryngomalacia is a temporary physiologic dysfunction due to abnormal flaccidity of laryngeal tissues or incoordination of supralaryngeal structures.

◆ Epidemiology

Laryngomalacia is the most common congenital airway abnormality. Overall, laryngomalacia accounts for 60% of cases of chronic laryngeal stridor. It is more common among males than females (2:1). Concomitant airway abnormalities are found in 12 to 37% of patients. Comorbidities, including prematurity, cardiovascular malformation, and neurologic and congenital or chromosomal abnormalities, are present in ~41% of patients.

◆ Clinical

Signs and Symptoms

Most commonly, patients present with intermittent inspiratory stridor that is relieved by neck extension and a prone position. Stridor is exacerbated by agitation. In extreme cases, patients become cyanotic, have a poor oral intake, have chest retractions, and develop pectus excavatum.

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Differential Diagnosis

Other causes of stridor in the early pediatric age group are

- Unilateral or bilateral vocal fold paralysis
- Laryngeal cleft
- Choanal atresia
- Airway hemangioma
- Laryngeal web
- Airway foreign body
- Acquired or congenital subglottic stenosis
- Craniofacial anomalies
- Glottic cysts
- Laryngeal reflux
- Saccular cyst
- Tracheomalacia
- Papillomatosis

Acid-reflux disorders (see Chapter 4.5) have been documented in up to 80% of patients with laryngomalacia.

◆ Evaluation

Physical Exam

An examination of any child with a possible breathing problem should discern if there is an oxygenation problem, and if so, an oxygen requirement. In the physical examination, one should assess for the location of a possible obstruction and include auscultation, inspection for chest retraction, assessment for cyanosis and other anomalies such as micrognathia.

To diagnose laryngomalacia and assess for other upper airway abnormalities, direct flexible endoscopic examination during respiration must be performed. Inward collapse of supraglottic structures are visualized on inspiration. The vocal folds are normal in appearance and motility.

Direct laryngoscopy and bronchoscopy in the operating room is the definitive evaluation.

Imaging

Radiologic examination is not generally necessary. However, if there is concern about dysphagia, barium swallow is helpful to assess esophageal function and to assess for evidence of vascular rings or other obstructive lesions or evidence of TEF.

Pathology

Histologically, submucosal edema and lymphatic dilatation are seen. Mechanisms of obstruction have been described by numerous authors. Chen and

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Hollinger considered the occurrence of two or more synchronously as causal in airway obstruction. These factors include an inward collapse of aryepiglottic folds, an elongated epiglottis (flaccid) curled on itself, anterior and medial collapsing movements of the arytenoid cartilages, posterior and inferior displacement of the epiglottis, short aryepiglottic folds, and an overly acute angle of epiglottis.

◆ Treatment Options

Medical

If an infant has good progress, which is indicated by adequate weight gain and normal development, then surgical therapy is not necessary. Instead, supportive care can be the mainstay of treatment.

Surgical

In one series of 985 patients with laryngomalacia, 12% required surgical intervention. Patients who should be considered for surgical management are those with severe stridor and failure to thrive, obstructive apnea, weight loss, severe chest deformity, cyanotic attacks, pulmonary hypertension, or cor pulmonale. Supraglottoplasty is performed using carbon dioxide laser or laryngeal microscissors, or other cold instruments such as pediatric ethmoid thru-cutting forceps. Most commonly, surgery involves removal of the prolapsing aryepiglottic fold with cuneiform cartilage or division of tight, short aryepiglottic fold. Unilateral supraglottoplasty has been advocated by some to reduce the risk of supraglottic stenosis. If epiglottis is displaced posteriorly, an epiglottopexy can be performed.

Potential complications include continued airway obstruction and posterior stenosis. In the event of continued airway obstruction, a tracheotomy may be necessary until the child "outgrows" laryngomalacia. The use of postoperative antibiotics has not been well evaluated in the literature and is controversial. Antireflux medications are used routinely.

◆ Outcome and Follow-Up

Supraglottoplasty relieves symptoms of airway obstruction in 90% of patients. Most infants require only a short hospital stay (1–3 days).

◆ ICD-9 Code

748.3 Laryngomalacia

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6.3 Bilateral Vocal Fold Paralysis

◆ Key Features

- Bilateral vocal fold paralysis is the second most common cause of infantile stridor.
- It typically requires a tracheotomy for maintenance of airway until vocal fold mobility returns, or definitive airway surgery is performed.
- Acquired causes are most common, even in infants.

Bilateral vocal fold paralysis (BVFP) is a potentially lethal problem requiring aggressive management, typically tracheotomy, at least in the short term. Depending on the cause, spontaneous recovery can occur. Recovery, however, is generally a slow process, taking up to a year. A variety of surgical approaches to widen the airway have been described. Usually, however, the voice quality is degraded when there is an intervention to enlarge/improve the laryngeal airway.

◆ Epidemiology

Twenty-five percent of BVFP is congenital. Most of the remaining cases are late presentations of central lesions. Acquired cases are most likely to occur as a result of surgery in the chest or from forceps delivery or infections.

◆ Clinical

Signs

Vocal fold immobility can be seen using flexible laryngoscopy in the clinic. Stridor is a sign of BVFP.

Symptoms

- Stridor
- Typically a normal voice, episodic respiratory distress (e.g., with upper respiratory tract infections)
- Weak cough; aspiration if underlying cause is a neural lesion above the nodose ganglion

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Differential Diagnosis

- Vocal fold fixation
- Laryngeal mass
- Laryngeal web

◆ Evaluation

Physical Exam

Assess for stridor, respiratory effort and rate, color, and weight (plot on a growth chart). Flexible laryngoscopy is mandatory (it may show twitching of cords with respiration; it typically shows paramedian vocal folds).

Imaging

An MRI should scan from skull base to mediastinum (to image the complete course of laryngeal nerves).

Other Tests

Tests depend on the clinical scenario. For example, consider audiometry if a CNS disorder is present; consider a biopsy of nonvascular laryngeal mass lesion, if present.

Pathology

Congenital Causes

- Laryngeal anomalies, malformations
- CNS anomalies: Arnold-Chiari malformation, hydrocephalus, encephalocele, leukodystrophy, cerebral dysgenesis
- Peripheral neuropathy: neonatal myasthenia gravis, benign congenital hypotonia, Werdnig-Hoffmann disease, Charcot-Marie-Tooth disease, arthrogryposis, viral neuropathy
- Birth trauma, including perinatal hypoxia

Acquired Causes

- Sequelae of cardiac or esophageal surgery
- Neoplasia
- Infections
- Trauma

◆ Treatment Options

Medical

No medical treatment options are available other than supportive care initially to maintain ventilation and oxygenation during definitive treatment planning.

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Surgical

- Tracheotomy and monitoring for spontaneous recovery (minimum of 1 year)
- Arytenoidopexy, endoscopic vocal fold lateralization procedures
  - Lateralization sutures passed through the vocal process and thyroid ala lateralize the vocal fold without mucosal destruction
- Arytenoidectomy
  - External approach (lateral cervical or via laryngofissure)
  - CO_2 laser via endoscopic approach
- Posterior cartilage graft
- Laryngeal reinnervation – not widely used in children

◆ Complications

Accidental tracheotomy displacement can be fatal in the early recovery period. Other complications related to laryngeal airway surgeries include dysphonia, aspiration in 4 to 6%, and dyspnea in 3 to 8% of patients undergoing arytenoid procedures. Other procedures have not been studied enough to ascertain their complication rates. Assessment for aspiration should be done preoperatively, as posterior glottic expansion surgery can increase risk of aspiration.

◆ Outcome and Follow-Up

Initially, the patient is monitored in an intensive care unit (ICU) setting with continuous pulse oximetry. Placement of "stay sutures" for any pediatric tracheotomy is mandatory. These two Prolene sutures, placed through cartilage adjacent to the tracheal opening at the time of surgery and secured to the neck skin with Steri-Strips, greatly facilitate the replacement of a tracheotomy tube into the airway if there is accidental displacement. To help prevent displacement, the tracheotomy appliance should be secured to the skin with four sutures as well as the umbilical neck tie.

Overall, arytenoidopexy and arytenoidectomy yield high rates of successful decannulation. These patients require long-term follow-up.

◆ ICD-9 Codes

478.34 Bilateral vocal fold paralysis
786.1 Stridor

Further Reading

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6.4 Laryngeal Clefts

◆ Key Features

- Laryngeal clefts are a rare congenital anomaly.
- They are frequently associated with other anomalies.
- Clinical presentation varies with the extent of the cleft. The patient may have problems with the airway, feeding, and voice.
- Most clefts are short, but complete laryngotracheoesophageal clefts have a mortality rate greater than 90%.

Laryngeal clefts represent a rare cause of stridor. Failed fusion of the posterior cricoid lamina and incomplete development of the tracheoesophageal septum results in a laryngeal cleft, an abnormal communication of the larynx and esophagus. Laryngeal clefts are usually sporadic nonsyndromic congenital abnormalities. Most are associated with other nonsyndromic congenital abnormalities including TEF, esophageal atresia, congenital heart disease, cleft lip and palate, micrognathia, glossoptosis, laryngomalacia, gastrointestinal and genitourinary anomalies. Rarely, they are attributable to a specific syndrome [Opitz-Frias and Pallister-Hall syndrome, VATER (vertebrae, anus, trachea, esophagus, and renal) Association].

◆ Epidemiology

Laryngeal clefts are rare. Congenital anomalies of the larynx are found in only 0.5% of the population, and clinically symptomatic laryngeal clefts constitute only 0.3 to 0.5% of all congenital laryngeal anomalies. Males are more likely to be affected than females (3:1). Thirty percent of laryngeal clefts are associated with maternal polyhydramnios. TEF is present in ~25% of patients with a laryngeal cleft and is associated with a higher failure rate of surgical repair.

◆ Clinical

Signs and Symptoms

There are no pathognomonic findings. Clinical symptoms depend on the extent of the cleft. Small clefts, whose anatomic involvement is limited to the interarytenoid musculature, present with stridor and feeding problems. There may be coughing, choking, stridor, aspiration pneumonias, or cyanotic episodes. Occasionally, patients with small laryngeal clefts may be asymptomatic. However, the most severe clefts are accompanied by aphonia, severe upper airway obstruction, and respiratory distress. Stridor occurs due to anterior collapse of posterior supraglottic structures. Cyanosis and stridor are exacerbated with feeding. In utero, polyhydramnios due to impaired swallowing of amniotic fluid by the fetus is associated with laryngeal clefts.

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◆ Differential Diagnosis

- Subglottic stenosis
- Laryngomalacia
- Unilateral or bilateral vocal fold paralysis
- Subglottic hemangioma
- TEF

◆ Evaluation

Physical Exam

A full head and neck exam should be performed, including a fiberoptic airway exam. Attention should be directed at possible sources of airway

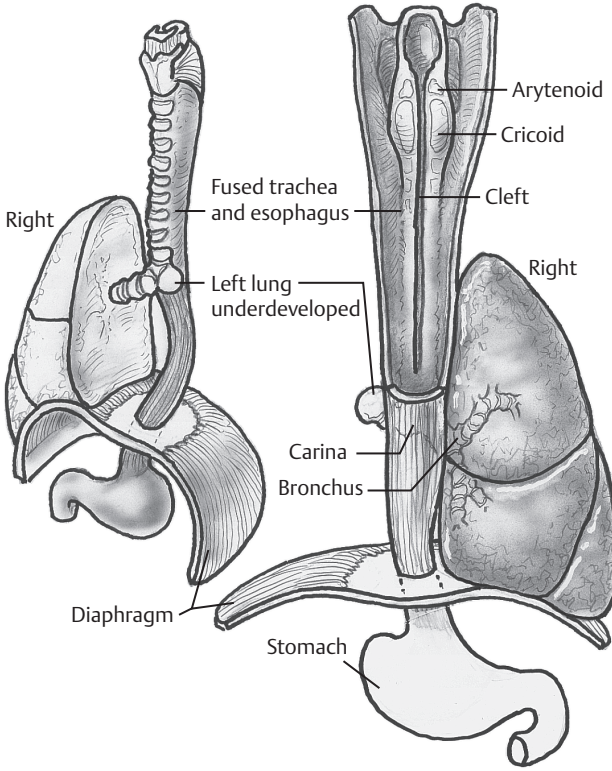

Fig. 6.1 Left anterolateral view and posterior view of type IV laryngotracheoesophageal cleft with left-side pulmonary agenesis and microgastria.

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symptoms, such as choanal atresia, craniofacial anomalies, laryngomalacia, and vocal fold motion impairment.

Laryngeal clefts must be visualized. Suspension microlaryngoscopy is used to visualize and palpate a laryngeal cleft. Palpation is performed on the lateral spread of the interarytenoid mucosa. The normal interarytenoid height from the vocal fold is 3 mm, and is severely reduced in laryngeal clefts. Bronchoscopy and esophagoscopy are necessary to adequately assess the airway and to investigate concomitant anomalies (**Fig. 6.1**).

Imaging

Modified barium swallow and a chest radiograph should be performed. Aspiration with thin liquids is the most common finding. A fiberoptic endoscopic evaluation of swallowing (FEES) is helpful preoperatively.

Pathology

There are several classification schemes in the literature. Because of the rare nature of this disorder, there is no consensus, although the Benjamin/Inglis Classification is commonly used. The classification is based on the inferior extent of cleft:

- Type 1: Interarytenoid soft tissue cleft without extension into cricoid cartilage.
- Type 2: The cleft extends into cricoid cartilage.
- Type 3: The cleft extends through the entire posterior cricoid cartilage.
- Type 4: The cleft extends into the thoracic trachea; it may extend to the carina.

◆ Treatment Options

Medical

Speech and feeding therapy aimed toward decreasing aspiration may be used in conjunction with medical therapy if the cleft is short. For clefts that extend only through interarytenoid musculature but not into cricoid, antireflux therapy in conjunction with thickened diet may be sufficient to control symptoms. However, most clefts require repair. In general, any cleft associated with significant aspiration is ruptured.

Surgical

The surgical approach should be individualized based on the symptoms, other associated findings on airway endoscopy, and type of cleft. The decision for surgical repair initially hinges on the extent of the cleft with respect to cricoid and its relationship with local anatomy. Endoscopic techniques may be feasible for type 1 and same type 2 clefts. However, if the cleft extends into inferior anatomic structures, open surgery is generally necessary. Type 3 and 4 clefts require open repair. This is also an option for patients

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who fail endoscopic management. Early repair is important to minimize irreversible pulmonary damage due to persistent aspiration.

Laryngeal clefts that extend into cricoid or trachea, without carinal involvement, are accessed via anterior laryngofissure to avoid neurovascular damage. Clefts that extend into the cricoid *and* involve carina require either a lateral pharyngotomy and lateral thoracotomy or an anterior laryngofissure and median sternotomy. Sternotomy requires cardiopulmonary bypass during the intrathoracic portion and perioperative tracheotomy. Clefts repaired via open approach in young children most often require tracheotomy. This likely is kept in place for an extended period as these patients tend to have substantial tracheomalacia for several years.

◆ Complications

Potential complications include recurrent laryngeal nerve injury, mediastinitis, respiratory distress, and dysphagia. Mediastinitis must be aggressively treated with IV antibiotics. Positive pressure ventilation and pulmonary care are necessary for respiratory distress, although aggressive positive pressure ventilation may compromise the anastomosis in the airway. Dysphagia is a chronic problem; many patients require feeding tube. Input from a skilled pediatric swallow therapist is necessary. Mortality associated with a type 4 cleft approaches 90%.

◆ Outcome and Follow-Up

Sedation and paralysis is employed in the early recovery period. Splinting is useful to maintain the neck in a neutral midline position. An oral or nasogastric tube is contraindicated; pressure compromises the anastomosis and may result in breakdown, tissue necrosis, and fistula formation. Antireflux medications and good pulmonary toilet are maintained. Enteral feeding is preferable (via jejunostomy or gastrostomy).

Outcomes vary greatly with the extent of anomaly, specific treatment, and possible comorbidities. For type 1 clefts repaired endoscopically, there is a 94% success rate reported in one series.

◆ ICD-9 Code

748.3 Laryngeal cleft

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6.5 Tracheoesophageal Fistula and Esophageal Atresia

◆ Key Features

- Tracheoesophageal fistula (TEF) and esophageal atresia (EA) are the result of a congenital communication between the trachea and esophagus.
- EA is also present in most cases.
- These congenital anomalies present with respiratory and/or feeding difficulties in the newborn.

Congenital TEF and EA are common congenital anomalies that usually occur together. Most cases are diagnosed immediately following birth or during infancy due to the associated life-threatening complications. However, isolated TEF may escape diagnosis until later. Problems with growth and feeding, pulmonary complications, and gastroesophageal morbidity may result from the condition.

◆ Epidemiology

TEF and EA in their various forms occur in ~1 in 3000 live births, with a slight male predominance. In over 50% of cases, TEF and EA are linked with other defects, such as in the VACTERL sequence and with chromosomal anomalies.

◆ Clinical

Signs and Symptoms

A sign of the condition, though nonspecific, is polyhydramnios on prenatal ultrasound. Symptoms of TEF include recurrent chest infections, cyanosis and choking on feeding, and abdominal distension. When EA is also present, the patient will not be able to swallow saliva or feed, and will drool excessively.

Differential Diagnosis

- Aspiration pneumonia
- Laryngeal cleft
- Tracheomalacia
- Esophageal stricture
- Esophageal diverticula
- Vascular ring
- Gastroesophageal reflux
- Other feeding problems

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TEF and EA develop as a result of incomplete separation of the respiratory and digestive divisions of the primitive foregut. Several variants of TEF and EA have been described, but five types predominate, as shown in **Fig. 6.2**.

◆ Evaluation

Physical Exam

The exam is likely to be normal unless marked abdominal distension is present. Other congenital anomalies may be noted. Passage of a tube from the mouth to the stomach may be impossible in the presence of EA. The subsequent chest x-ray will typically show the tip of the catheter coiled in the proximal esophageal pouch.

Imaging

A chest x-ray will show gaseous distension of the bowel when TEF is present. There may be pulmonary changes from persistent respiratory infections. A contrast esophagram can be used to demonstrate an isolated TEF but may provide a false-negative because of the slanted orientation of the fistulous tract. Abdominal ultrasonography is used to look for associated renal pathology. Echocardiography is used to examine the heart for any associated anomaly and to aid in planning surgical treatment. Bronchoscopy may help find the location of the fistula and guide operative strategy.

◆ Treatment Options

To prevent aspiration and gastric reflux, a sump catheter should be immediately placed into the upper esophageal pouch and connected to constant suction. The patient should be placed in a prone, head-up position.

Most infants undergo immediate primary repair. Reasons for the delay of surgical treatment include severe associated anomalies, severe pneumonia or respiratory distress, and a long gap between the esophageal pouches.

Repair consists of ligation of the fistula and primary esophageal anastomosis via a right posterolateral thoracotomy. If a right-sided aortic arch is present, a left thoracotomy is used. A cervical incision may be used if there is an isolated TEF. If more than two vertebral bodies separate the upper and lower esophageal segments (“long-gap”), extramucosal circular myotomies can be used. If the gap persists or if there are significant complications, replacement of the esophagus is necessary.

If a patient cannot undergo immediate primary repair, a gastrostomy may be used for gastric decompression and access for feedings.

◆ Complications

Anastomotic Leaks

Anastomotic leaks usually resolve with parenteral nutrition and posterior drainage. Repeat thoracotomy is required if healing does not occur.

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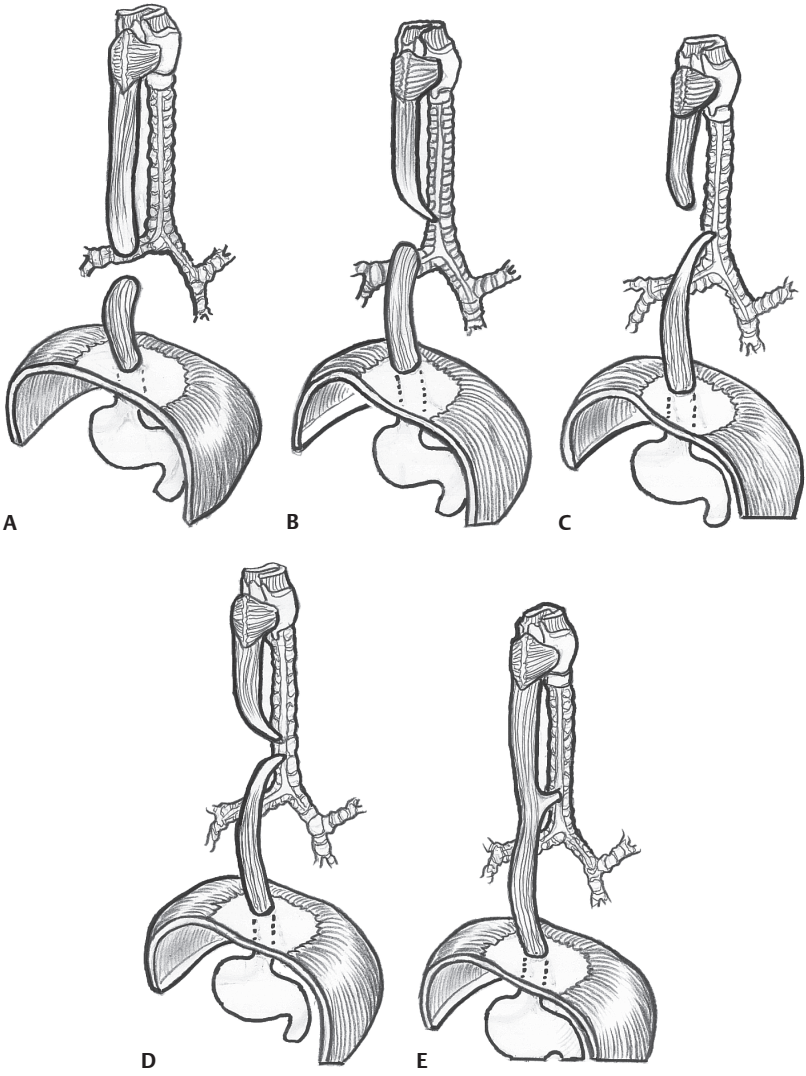

Fig. 6.2 Classification of tracheoesophageal fistula (TEF) and esophageal atresia (EA). **(A)** EA (Gross classification A, Vogt classification 2, approximate frequency 8%). **(B)** Proximal TEF with distal EA (Gross classification B, Vogt classification 3A, approximate frequency 0.8%). **(C)** Distal TEF with proximal EA (Gross classification C, Vogt classification 3B, approximate frequency 88.5%). **(D)** Proximal TEF and distal TEF (Gross classification D, Vogt classification 3C, approximate frequency 1.4%). **(E)** TEF without EA, or "H"-type TEF (Gross classification E, approximate frequency 4%).

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Stricture

Stricture is usually treated with repeated dilatation.

Dysphagia

Peristalsis is abnormal in the vast majority of patients with a history of TEF or EA. Patients are advised to eat slowly and may need to avoid meats. Esophageal obstruction may occur and foreign body removal may be required.

Acid-Reflux Disorders

All patients should be placed on antireflux medical treatments and these should be continued until at least the time when an upright posture is achieved. Antireflux surgery may be necessary in some cases.

Tracheomalacia

Treatment is generally reserved for those with "dying spells" or recurrent pneumonia. Aortopexy is generally employed. If this fails, tracheotomy may be required. Rarely tracheal stents are employed, but they are not used in infants and are uncommonly employed in young children.

Recurrent Tracheoesophageal Fistula

Recurrent TEF requires reoperation.

◆ Outcome and Follow-Up

Oral feeding is delayed until a contrast study done several days after the operation shows no leaks or narrowing around the anastomosis. In the case of isolated TEF repair, oral feeding can resume immediately if the integrity of the repair is certain. Complications occur relatively frequently following operative repair of TEF/EA and one or more additional operations are needed in half the cases. Strictures are the most common complication, and a barium swallow or esophagoscopy is needed before hospital discharge in all patients.

Although complications occur relatively frequently, patients have an excellent chance of leading normal lives in the absence of severe associated anomalies. Inherent structural and functional defects in the trachea and esophagus result in significant respiratory and gastroesophageal sequelae, including poor growth, feeding problems, tracheomalacia, bronchomalacia, recurrent chest infections, and reflux. However, the frequency of such events appears to decrease significantly with age.

◆ ICD-9 Code

750.3 Tracheoesophageal fistula, esophageal atresia, and stenosis

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6.6 Vascular Rings

◆ Key Features

- The trachea and esophagus are completely or incompletely surrounded by vascular structures.
- Compression of the trachea, the bronchi, and/or the esophagus may occur.
- Most symptomatic malformations present during infancy or early childhood.

The term vascular ring refers to an aortic arch abnormality in which the trachea and esophagus are surrounded by vascular structures. It may be complete or incomplete. The greater the degree of compression the vascular ring causes, the more severe the symptoms are and the earlier they present. For symptomatic patients, treatment is generally surgical.

◆ Embryology and Anatomy

Vascular rings arise during embryonic development from the abnormal evolution of the arterial branchial arch system. In normal embryonic vascular development, ventral and dorsal aortae are connected by six pairs of aortic arches. The first, second, and fifth arches regress, as well as a portion of the right fourth arch. This leaves the usual left aortic arch. Residual segments of the third, fourth, and sixth arches develop into the mature anatomy of the mediastinal vascular structures. Inappropriate persistence or development of segments leads to congenital aortic arch anomalies.

The most common vascular ring is a double aortic arch, accounting for 50 to 60% of symptomatic vascular rings. A right aortic arch with an aberrant left subclavian artery is the second most common, accounting for 12 to 25% of cases. Other vascular anomalies include a right aortic arch with mirror image branching and left ductus arteriosus, a pulmonary artery sling, an anomalous innominate artery, and left aortic arch anomalies.

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◆ Epidemiology

At autopsy, 3% of the population has a congenital anomaly of the aortic arch system. Most are asymptomatic. Vascular rings account for less than 1% of congenital cardiovascular malformations.

◆ Clinical

Signs and Symptoms

Symptoms depend on the location and degree of vascular compression. Wheezing, stridor, aspiration, cyanotic or apneic attacks, and dysphagia are characteristic. Feeding may exacerbate stridor. Dysphagia is worsened by solid foods. Recurrent respiratory infections such as aspiration pneumonia may also be present.

Differential Diagnosis

- Asthma
- Tracheomalacia
- Bronchiolitis
- Laryngeal stenosis
- Congenital stridor
- Laryngeal web
- Croup
- Foreign body aspiration
- Laryngomalacia

◆ Evaluation

Physical Exam

Physical findings vary, often in accordance with the patient's history. Stridor is characteristically expiratory. It is often associated with cough, tachypnea, and rhonchi. Expiratory, high-pitched wheezes and intercostal retractions can also be appreciated. Patients may hold their neck in hyperextension to alleviate respiratory distress. Respiratory findings typically do not improve with nebulized bronchodilator treatment and are worsened by exertion. Pulmonary infection may be the presenting symptom, especially in older children.

Imaging

A chest radiograph will reveal laterality of the aortic arch by typical contralateral deviation of the trachea. Two arches may be suspected if there is compression of the trachea at the level of the arches. On the lateral view, anterior tracheal compression may be evident. Tracheal constriction evidenced by narrowing or obliteration of the distal tracheal air column and lung hyperinflation may also be seen.

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A barium esophagram is diagnostic in over 90% of patients with a vascular ring. With most aortic arch anomalies, a posterior indentation of the esophagus will be seen. Bilateral and posterior indentations are common in double aortic arch and both types of right aortic arch. Retroesophageal subclavian arteries will show slanted filling defects in the posterior esophagus. A pulmonary artery sling will typically show an anterior esophageal indentation. An anomalous innominate artery usually produces a normal esophagram.

Bronchoscopy is often the diagnostic technique of choice to evaluate structural and dynamic anomalies of the airway. Bronchoscopy can also evaluate the tracheobronchial tree for coexisting or intrinsic lesions such as tracheomalacia, stenosis, complete tracheal rings or aberrant bronchi. In many centers, MRI is becoming the diagnostic technique of choice. Sedation, airway management, and intubation may be challenging in the patient with signs and symptoms of airway compression.

CT may show location, degree, and extent of tracheal narrowing. It is faster and requires less sedation than MRI but is not as good at defining vascular anatomy unless CT angiography is obtained. Other disadvantages include exposure to ionizing radiation and IV contrast.

Frequently but not invariably, echocardiography can show the presence and define the anatomy of a vascular ring. It will show associated cardiovascular anomalies and can be done at the bedside. It does not image the airways.

Cardiac catheterization with angiography provides a clear delineation of abnormal vessels and is helpful in evaluating associated congenital heart defects. With the advent of MRI, it is rarely necessary for isolated aortic arch anomalies.

Other Tests

Pulmonary function tests can be used in the evaluation of infants and children with suspected tracheal obstruction of vascular origin. The shape of partial expiratory flow-volume curve can help to localize the site and assess the severity of airway obstruction. Usually, pulmonary function testing cannot be obtained in children under age 6.

◆ Treatment Options

Medical

Asymptomatic or mildly symptomatic patients can be managed medically with humidification of inspired air, drainage of bronchial secretions, antibiotics, and supplemental oxygen when needed, and a soft diet or tube feedings if dysphagia exists.

Surgical

Surgery is indicated in all patients with symptomatic vascular rings. Asymptomatic complete rings should undergo elective surgery if there is suspicion for progressive airway compromise. The goal of surgical treatment is to divide the compressive vascular ring, relieve tracheobronchial and esophageal compression, and maintain normal perfusion of the aortic arch. For a double aortic arch, the atretic or hypoplastic arch is divided along with the ligamentum or ductus arteriosus. For right aortic arch variants, the left ductus or

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ligamentum arteriosum is divided. If surgery is indicated for an anomalous innominate artery, the preferred treatment is aortopexy. For a pulmonary artery sling, the ductus or ligamentum arteriosum is divided and the left pulmonary artery is divided and reimplanted into the main pulmonary artery.

The approach is generally through a left posterolateral thoracotomy, but certain types of vascular rings require a right thoracotomy. A sternotomy incision is indicated for concomitant repair of intracardiac defects, or for the repair of pulmonary artery sling, or for repair of complete tracheal rings with slide tracheoplasty on cardiac bypass. Intraoperative bronchoscopy is helpful in evaluating the effects of surgery on airway patency; tracheomalacia will persist after repair but will usually improve with time.

◆ Complications

Tracheomalacia and Bronchomalacia

Prolonged intubation may be required to maintain airway patency in long-segment malacia but can lead to endoluminal airway complications such as granuloma formation. Endoluminal stenting procedures have also been described. Occasionally, reconstruction of the affected airway segment is required. Generally, with growth of the trachea and gradually increasing stiffness of the cartilage, symptoms are likely to improve. Tracheotomy may be needed to stent substantial tracheomalacia.

Recurrent Laryngeal Nerve Injury

In unilateral vocal fold paralysis, the need for intervention is based on the degree of hoarseness and the risk of aspiration. In bilateral fold paralysis, surgery is needed to alleviate glottic obstruction. Tracheotomy may also be required.

Chylothorax

This is an uncommon perioperative complication. Treatment of choice is implantation of a pleuroperitoneal shunt.

◆ Outcome and Follow-Up

Intensive respiratory care is always needed postoperatively. Humidified oxygen, antibiotic prophylaxis for pulmonary infections, frequent suctioning of tracheal secretions, and diligent chest physiotherapy are vital.

Major postoperative issues relate to concurrent cardiac defects and residual airway disorders. Successful tracheal extubation is possible in most patients. Successful repair of the vascular ring may not immediately relieve airway obstruction.

The patients who are asymptomatic or mildly symptomatic with incomplete rings are likely to improve with age. Of the patients who received surgical repair, 95% are expected to survive, and most of them will become completely asymptomatic. However, persistence of various degrees and types of pulmonary function anomalies may be found in a significant number of patients.

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◆ ICD-9 Code

747.21 Vascular ring, other anomalies of aortic arch

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6.7 Subglottic Stenosis

◆ Key Features

- The subglottis is the narrowest part of the infant airway.
- Acquired subglottic stenosis is most commonly found; the most common related factor is intubation.
- Subglottic stenosis of 70% of the lumen or greater is associated with daily symptoms.

The subglottis, the narrowest part of the pediatric airway, is composed of a complete cartilaginous ring, the cricoid, and loose submucosa that swells when irritated (as in, for example, croup). It is the region most likely to be affected by pressure from a too-large or frequently moving endotracheal tube. Congenital subglottic stenosis most commonly results from an abnormally shaped cricoid, with intraluminal lateral shelves, resulting in an oval shape to the lumen. Intervention is individualized to the patient: sometimes a watch and wait approach works; other children benefit from some type of surgery.

◆ Epidemiology

Incidence is ~1.5 cases per million, but it occurs in 1 to 8% of neonates requiring intubation.

◆ Clinical

An abnormal, narrow airway causes stridor. It is, therefore, worth reviewing the typical dimensions of the “normal” airway (**Table 6.2**).

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Table 6.2 Normal Airway Size by Age

Age	Normal Subglottic Airway Diameter (mm)	Expected Endotracheal Tube Size	Expected Bronchoscope Size
Premature	3.5–4.5	2.5–3.0	2.5
0–3 mo	5.0	3.5	3.0
3–9 mo	5.5	4.0	3.5
9–24 mo	6.0	4.5	4.0
2–4 yr	6.5–7.0	5.0	4.0
4–6 yr	7.5	5.5	5.0
6–8 yr	8.0	6.0	6.0

Source: Van de Water TR, Staecker H, eds. Otolaryngology: Basic Science and Clinical Review. Stuttgart/New York: Thieme; 2006:213.

Signs

The signs include stridor, respiratory distress, and typically a normal voice.

Symptoms

- Unsuccessful extubation of a patient in the neonatal intensive care unit (NICU)
- Stridor, may be biphasic, may present only with agitation
- Respiratory distress exacerbated by upper respiratory tract infection
- Recurrent croup
- Feeding problems
- Slow growth, failure to thrive

Differential Diagnosis

Other causes of stridor with normal voice include laryngomalacia, subglottic cyst, subglottic hemangioma, and tracheal stenosis.

◆ Evaluation

Physical Exam

Flexible laryngoscopy should be done to assess vocal fold movement. The subglottis is sometimes seen with this exam, but the scope should not be passed through the glottis because of the risk of inducing vasovagal reflexes. Bronchoscopy will show subglottic stenosis. The degree of narrowing and the length of the narrowed segment are important to measure.

The Cotton-Meyer grading system apparently correlates to symptoms and prognosis (**Table 6.3**). Grade I stenoses typically are asymptomatic unless there is an upper respiratory tract infection.

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Table 6.3 Grading for Subglottic Stenosis

Grade	Degree of Narrowing
I	<50%
II	50–70%
III	71–99%
IV	Total obstruction

Imaging

A soft tissue lateral x-ray, airway fluoroscopy, or CT may show the subglottic anatomy. Bear in mind that airway fluoroscopy involves significant radiation exposure.

Other Tests

A complete evaluation for reflux (including a barium swallow, a gastric scintiscan, a pH probe) may be helpful. Pulmonary function tests and videostroboscopy are indicated in cooperative older patients.

Pathology

Congenital subglottic stenosis may be cartilaginous or membranous (a thickened submucosa). The subglottis is the narrowest part of the pediatric airway in normal circumstances, so pressure necrosis from an endotracheal tube is more likely here than elsewhere in the airway. Acquired stenosis may be caused by mucosal necrosis with healing by granulation tissue and subsequent fibrosis, although deeper injuries including cartilage necrosis can occur. Factors related to injury include size of endotracheal tube, number of reintubations, tube movement, and tube material (polyvinyl chloride is considered safest).

◆ Treatment Options

Medical

Antireflux medications have a role in management. If an acid-reflux disorder is not diagnosed with traditional tests, prophylactic reflux medications should be given, especially if there is a suggestion of reflux laryngitis on examination.

Surgical

Dilation

Dilation is an option for mild soft stenosis.

Laser Division

Laser division (CO_2, argon, or KTP laser) is an option for early stenosis (granulation tissue), crescent-shaped bands, and thin circumferential webs.

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Cricoid Split

A cricoid split is indicated in neonates with solitary subglottic stenosis and failure to extubate. Traditionally, the criteria for the procedure are weight at least 1500 g, no ventilator support for 10 days prior to repair, oxygen requirement less than 30%, no congestive heart failure in the month prior to the repair, no acute upper respiratory infection, and no antihypertensive medications required in the 10 days prior to extubation. Basically, the approach is like a tracheotomy, but the anterior midline incision in the trachea extends through the cricoid and the inferior part of the thyroid cartilage. An endotracheal tube is placed such that the incision is open by 2 to 3 mm, left in situ for 7 to 14 days, and steroids are used prior to extubation.

Laryngotracheoplasty

If more than 3 mm of circumference gain is needed, a cartilage graft is necessary. Auricular or costal cartilage may be used. Grafts can be placed anterior and posterior in the subglottis. Stenting is required if the posterior cricoid is divided; this may be achieved by endotracheal intubation for 1 to 2 weeks.

Only mature stenoses should be reconstructed with an open procedure. Cricotracheal resection is reserved for amenable airway lesions.

◆ Complications

Emergency complications can involve airway obstruction or respiratory problems. Causes include mucous plugs, granulation tissue, aspiration of stenting materials (if used), hematoma, hemorrhage into the airway, or pneumothorax. Treatment is aimed at the underlying problem. Treatment may require airway suctioning for mucous plugs, use of aerosolized steroids (e.g., dexamethasone 0.25 to 1.0 mg/kg per day, to a maximum of 20 mg/d) to reduce granulations, chest tube placement for pneumothorax, hemorrhage or hematoma, or return to the operating room for bronchoscopic or possible open treatments.

Other complications include cricoid split failure (repeat intubation for 72 additional hours with a half-size smaller tube; if that fails, then tracheotomy) and laryngotracheoplasty failure (infection may lead to graft necrosis, treated by antibiotics and revision surgery).

◆ Outcome and Follow-Up

A postoperative chest radiograph should always be obtained for open reconstructive procedures. Continuous O_2 saturation monitoring should be overnight; an ICU setting should be available for any open procedures. Sedation is required but paralysis is not desired, so that in the event of accidental extubation, the child can make breathing efforts. Also, spontaneous ventilation is preferred because of improved airway clearance of secretions and decreased muscle weakness that can develop from not breathing for 7–10 days.

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Humidification and chest physiotherapy are helpful. Racemic epinephrine may be used to treat postextubation edema. Steroids are judiciously avoided in patients with new cartilage grafts as steroids may inhibit mucosalization and compromise neovascularity. Only one dose is given the morning of extubation.

Antireflux medications are typically employed. Prophylactic postoperative antibiotics are typically given. Management by a skilled pediatric intensivist is required, to reduce potential morbidities.

These children require long-term follow-up. Depending on the procedure employed and the patient's symptoms, serial bronchoscopy may be indicated.

◆ ICD-9 Code

478 Other diseases of upper respiratory tract

Further Reading

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6.8 Pierre Robin Sequence

◆ Key Features

- Pierre Robin sequence requires the presence of micrognathia, glossoptosis, and usually a cleft palate (frequently U-shaped).
- Infants present with airway obstruction because the tongue falls into the pharyngeal airway, and feeding difficulties.
- Hearing loss is frequent, from otitis media (OM) with effusion (OME; 90%), middle ear anomalies (60%), and inner ear anomalies (40%).

First described in 1891 by Lannelongue and Ménard, then further by Pierre Robin in 1923, the sequence requires the presence of micrognathia and glossoptosis. Most (90%) also have a cleft palate. Neonates have feeding difficulties. They have upper airway obstruction and are usually difficult to intubate. These children have a high incidence (up to 80%) of other systemic anomalies.

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◆ Epidemiology

Incidence is 1 in 8500 births. Up to 80% are syndromic, most commonly Stickler syndrome (autosomal dominant; 1 per 10,000 incidence in the United States; flat midface, cleft palate, retinal detachment, cataracts, arthropathy) and Velocardiofacial syndrome (autosomal dominant; cleft palate, cardiac anomalies, almond-shaped palpebral fissures, tubular nose, small mouth, learning disabilities). Some cases have autosomal recessive or X-linked inheritance.

◆ Clinical

Signs

- Micrognathia, glossoptosis, cleft palate
- Airway obstruction with desaturations
- Pinna abnormalities, OME may be associated

Symptoms

- Airway obstruction with stertor, cyanosis, respiratory failure
- Prone position may improve airway obstruction in mild cases
- Failure to thrive

Differential Diagnosis

- Stickler syndrome
- Velocardiofacial syndrome
- Fetal alcohol syndrome
- Treacher-Collins syndrome
- Nager syndrome
- Beckwith-Wiedemann syndrome

All of these syndromes are associated with Pierre Robin sequence.

◆ Evaluation

The most important issue is to first ensure an adequate airway and feeding. Then, one should determine whether there is an associated syndrome. Syndromic patients will generally require more involved interventions.

Physical Exam

A complete head and neck exam will reveal the signs of the sequence. A maxillary–mandibular discrepancy can be measured by placing the infant upright, passively closing the jaw, placing the wooden end of a cotton applicator on the anterior surface of the mandibular alveolar ridge in the midline,

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then marking where the anterior surface of the maxillary alveolar ridge falls. This measure can be used to monitor growth and surgical outcome.

Pinnae and tympanic membranes should be evaluated. A flexible laryngoscopy is required to rule out concomitant laryngomalacia.

Imaging

Imaging is not typically required to assess the airway obstruction. Imaging may be indicated to evaluate other anomalies coincident with the sequence.

Labs

Continuous monitoring and oxygen saturation monitoring in a NICU environment is required.

Other Tests

All children with Pierre Robin sequence should have early vision and hearing screening, given the association with Stickler syndrome. A sleep study may be indicated in mild cases. Bronchoscopy is indicated in severe cases.

Pathology

This sequence is initiated by mandibular hypoplasia in utero. Because of insufficient room in the mouth for the tongue, it remains positioned between the palatal shelves preventing their normal fusion and leading to a cleft palate.

◆ Treatment Options (**Table 6.4**)

Medical

- Positioning: prone position works in about half of patients
- Nasopharyngeal airway
- Oral airway
- Short term intubation
- Manage reflux

Table 6.4 Strategies for Treating Airway Obstruction in Patients with Pierre Robin Sequence

Prone positioning
Nasopharyngeal airway
Glossopexy, tongue–lip adhesion
Tracheotomy for severe obstruction, synchronous airway lesions, failure of other methods
Mandibular distraction osteogenesis

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Surgical

About half of patients require surgery to support their airway.

Temporary Tongue–Lip Adhesion

This involves raising a flap on the inner lower lip and ventral tongue, suturing these together, and sometimes suspending a button placed on the tongue base via a suture that goes around the anterior mandible (this button stays in only for the first week). This brings the tongue forward. It is all taken down by the first year of life; by this time the mandible has usually grown enough that the airway is clear. Fifteen percent will fail and require a tracheotomy.

Mandibular Distraction Osteogenesis

Proximal and distal screws are placed into the mandible bilaterally, then an osteotomy is performed between them. An external device is attached to gradually distract the mandibular segments, typically 1 mm per day. The distractor needs to be left in place for 6 to 8 weeks for consolidation.

Tracheotomy

A tracheotomy is indicated in syndromic children, patients with aspiration, patients with reflux or severe sleep apnea, those with second sites of obstruction below the hypopharynx, and those who fail tongue–lip adhesion and/or distraction osteogenesis.

◆ Complications

- Tongue–lip adhesion: Dehiscence or failure to relieve airway obstruction is treated by either mandibular distraction or tracheotomy.
- Mandibular distraction osteogenesis: Pin tracks may become infected, loosening the pins, which will then require replacement; note also that tooth buds may be damaged. TMJ ankylosis and malocclusion problems may occur.
- Tracheotomy (see A6 in Appendix A)

◆ Outcome and Follow-Up

Infants require continuous close monitoring in an ICU setting. Initial feeding requires a nasogastric tube, and if there are no desaturations, trials of oral feeding start. Children may be discharged home once the airway and feeding are stabilized.

Elective repair of the cleft palate is also required, but management of airway obstruction takes priority. Nonsyndromic patients can generally undergo tracheotomy decannulation following palatoplasty, if standard decannulation criteria are met.

Nonsyndromic children, particularly those not requiring surgical intervention, do very well with catch up growth and are likely to have a normal facial profile by age 5. Syndromic children are more likely to require multimodality treatment. Follow-up only needs to continue until the airway obstruction is resolved.

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◆ ICD-9 Codes

749 Cleft palate
748 Congenital anomalies of the respiratory system

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6.9 Genetics and Syndromes

Congenital anomalies, whether single or multiple, can be induced by environmental and teratogenic insults as well as chromosomal or single gene defects. This chapter includes only the most common and relevant syndromes with associated craniofacial anomalies. The otolaryngologist may be primarily involved in managing the otolaryngologic manifestations of these syndromes but may also play an important role in the early identification and referral for genetic counseling in children with suspected genetic or syndrome features.

◆ Epidemiology

Major congenital anomalies diagnosed within the first year of life affect ~3% of neonates and congenital defects contribute to nearly 20% of infant deaths. Of the defects affecting children with multiple congenital anomalies, 62% are otolaryngologic in nature. In the case of multiple anomalies where the underlying cause has been identified, 84% have an otolaryngologic feature.

◆ Relevant Definitions

Association: Nonrandom occurrence of a pattern of anomalies that are not identified as a sequence or syndrome; for example, CHARGE (coloboma of the eye, heart defects, atresia of the nasal choanae, retardation of growth and/or development, genital and/or urinary abnormalities, and ear abnormalities and deafness) association.

Sequence: Pattern of multiple defects resulting from a single primary malformation or insult, i.e., Pierre Robin sequence. The fact that a cluster of anomalies is defined as a sequence does not exclude Mendelian inheritance.

Syndrome: Cluster of anomalies where all features are pathologically related; for example, Down syndrome, fetal alcohol syndrome.

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◆ Relevant Associations, Sequences, and Syndromes

- **Achondroplasia**
  - Inheritance: Autosomal dominant
  - Genetic loci: Spontaneous mutation leading to a defect in fibroblast growth factor receptor-3
  - General: Most common cause of short-limbed dwarfism; advanced paternal age is a risk factor
  - Relevant features: Frontal bossing, mid-face hypoplasia, obstructive sleep apnea
  - Associated features: Shortened limbs, long narrow trunk, lumbar lordosis, limited elbow extension, genu varum, compression of craniovertebral junction (can lead to central apnea and sudden death), hypotonia
- **Branchio-oculo-facial syndrome (BOF syndrome)**
  - Inheritance: Autosomal dominant
  - Genetic loci: Not yet identified
  - Relevant features: Branchial cleft sinuses, lacrimal duct obstruction, conductive hearing loss, pseudocleft of upper lip, auricular malformations (low-set, malformed)
  - Associated features: Low birth weight, growth and developmental delay, premature aging
- **Catel-Manzke syndrome**
  - Inheritance: X-linked (speculated)
  - Genetic loci: Not yet identified
  - Relevant features: Cleft palate, micrognathia, auricular malformations
  - Associated features: Cardiac septal defect, growth delay, hyperphalangy (increased number of phalanges) of index finger
- **CHARGE association**
  - Inheritance: Sporadic
  - Genetic locus: 8q12.1, 7q21.1
  - General: Coloboma iris, heart defects, choanal atresia, retarded growth, genital hypoplasia, ear abnormalities
  - Additional relevant features: Auricular anomalies (prominent, folded ears, absent helix) conductive/sensorineural hearing loss, micrognathia, midface hypoplasia, dysphagia, short neck
  - Additional associated features: Short stature, ptosis, microphthalmos, omphalocele, cryptorchism, syndactyly, renal hypoplasia, delayed skeletal maturation, pituitary defects, hypocalcemia
- **Costello syndrome**
  - Inheritance: Autosomal dominant and/or gonadal mosaicism
  - Genetic loci: HRAS or KRAS gene mutation on chromosome 11p15.5
  - Relevant features: Auricular malformations (low-set, thick lobes), oral, nasal, and anal papillomas

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- Head and neck features: Macrocephaly, epicanthic folds, coarse facial features, strabismus, thick lips, depressed nasal bridge, curly hair
- Associated features: growth and developmental delay, hypertrophic cardiomyopathy, thin, deep-set nails, skin hyperpigmentation, deep plantar/palmar creases, short neck, tight Achilles tendons

- **Cri-du-chat syndrome**
  - Inheritance: De novo mutations most common, can result from unbalanced translocation/recombination in a parent (12%)
  - Genetic loci: Partial and variable deletion of short arm of chromosome 5
  - Relevant features: Narrowed endolarynx (diamond-shaped), persistent interarytenoid cleft, microcephaly, round face, hypertelorism, micrognathia, epicanthal folds, low-set ears
  - Associated features: Hypotonia, severe psychomotor and developmental delay
- **Down syndrome**
  - Inheritance: Sporadic
  - Genetic loci: Trisomy 21
  - General: Most common genetic disorder associated with mental retardation and developmental delay; advanced maternal age risk factor (>35 years); 1:800 live births
  - Relevant features: Microcephaly, midface retrusion, upslanting palpebral fissures, epicanthal folds, macroglossia, frequent OM with effusion, sleep apnea, auricular anomalies (small, low-set, overfolding of upper helices), middle and inner ear anomalies
  - Associated features: Congenital cardiac defects (40%), hypotonia, joint laxity, and underdeveloped cervical vertebra (risk of atlantoaxial subluxation/dislocation), flat occiput, three fontanels, excess nuchal skin, short stature, clinodactyly 5th digit, single palmar crease
- **Ectrodactyly-ectodermal dysplasia-clefting syndrome**
  - Inheritance: Autosomal dominant (variable expression)
  - Genetic loci: 7q11.2–q21.3
  - Relevant features: Cleft lip and/or cleft palate, blue irides, lacrimal duct defects, blepharophimosis, partial anodontia, light, spare, thin, wiry hair
  - Associated features: Hyperkeratosis, hypotrichosis, hypohidrosis, hypoplastic nipples, anomalies of the extremities (syndactyly, ectrodactyly), genitourinary defects
- **Fragile X syndrome**
  - Inheritance: X-linked
  - Genetic loci: Xq27.3
  - General: Second most common cause of genetic developmental delay
  - Relevant features: Prominent ears, large jaw, long face, high-pitched speech
  - Associated features: Mitral valve prolapse, behavioral disturbance, macroorchidism, joint hypermobility, pes planus

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- **Fraser syndrome (cryptophthalmos)**
  - Inheritance: Autosomal recessive
  - Genetic loci: FRAS1 or FREM2 gene 13q13.3, 4q21
  - Relevant features: Ear anomalies (aural atresia, cupped ears), laryngeal stenosis/atresia, hypoplastic, notched nares, bilateral cryptophthalmos, eyebrow anomalies
  - Associated features: Developmental delay (50%), partial cutaneous syndactyly, genital anomalies, renal hypoplasia/agenesis
- **Larsen syndrome**
  - Inheritance: Autosomal dominant (known), possible recessive form speculated
  - Genetic loci: 3p21.1–p14.1
  - Relevant features: Cleft palate, flat facies, depressed nasal bridge, hypertelorism, prominent frontal boss
  - Associated features: Congenital joint dislocation, long, nontapering fingers with short fingernails, spinal deformities
- **Marshall syndrome**
  - Inheritance: Autosomal dominant
  - Genetic loci: 1q21
  - Relevant features: Sensorineural hearing loss, short nose with flat nasal bridge and midface, anteverted nares, large eyes, prominent protruding upper incisors
  - Associated features: Cataracts, myopia, short stature, calvarial thickening, spondyloepiphyseal abnormalities
- **Miller syndrome (postaxial acrofacial dysostosis)**
  - Inheritance: Autosomal recessive
  - Genetic loci: Not yet identified
  - General: Facial features similar to Treacher-Collins syndrome with limb anomalies
  - Relevant features: Cleft lip and/or cleft palate, malar hypoplasia and/or vertical bony cleft, micrognathia, hypoplastic cup shaped ears, down-slanting palpebral fissures, coloboma, ectropion
  - Associated features: Absence of 5th digit on all limbs, accessory nipples
- **Moebius syndrome**
  - Inheritance: Sporadic
  - Genetic loci: 13q12.2–q13
  - Relevant features: Congenital cranial nerve VI and VII palsy, orofacial dysmorphism
  - Associated features: Limb malformations, developmental delay
- **Mucopolysaccharidoses**
  - General: Storage diseases secondary to lysosomal enzyme deficiencies
  - Examples: Hunter, Hurler, Morquio, Sanfilippo, etc.

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- Relevant features: OME, sensorineural hearing loss, obstructive sleep apnea
- Associated features: Developmental delay, short-stature

- **Nager syndrome (Nager acrofacial dysostoses)**
  - Inheritance: Autosomal dominant
  - Genetic loci: 9q32
  - General: Facial features similar to Treacher-Collins with limb anomalies or deficiencies
  - Relevant features: Aural atresia, cleft palate, auricular malformations (low set, rotated posteriorly), malar hypoplasia, high nasal bridge, down-slanting palpebral fissures, absence of lower eyelashes
  - Associated features: Hypoplasia of radial limbs, development and cognition normal
- **Noonan syndrome**
  - Inheritance: Autosomal dominant
  - Genetic loci: 12q24
  - Relevant features: Neck webbing, auricular anomalies (low set), down-slanting palpebral fissures, ptosis, hypertelorism
  - Associated features: Low posterior hairline, short stature, pectus excavatum, pulmonic stenosis, bleeding diathesis
- **Opitz G syndrome (BBB syndrome)**
  - Inheritance: X-linked recessive and autosomal dominant
  - Genetic loci: 22q11
  - General: Midline defects
  - Relevant features: Cleft lip and/or cleft palate, laryngeal clefting, auricular anomalies (ears rotated posteriorly), micrognathia, flattened nasal bridge, anteverted nostrils, hypertelorism
  - Associated features: Developmental delay, hypospadia, cryptorchidism, hernias
- **Oro-facial-digital syndrome**
  - Inheritance
    - Type I: X-linked dominant, lethal in males
    - Type II: Autosomal recessive (speculated)
  - Genetic loci
    - Type I: Xp22.2–p22.3 CXORF5 gene
    - Type II: Not yet identified
  - Relevant features
    - Type I: Median cleft lip, cleft palate, bifid tongue, oral frenula and clefts, hypoplastic nasal alae, lateral displacement of inner canthi
    - Type II: Midline partial cleft palate, cleft tongue, low nasal bridge, broad nasal tip, conductive hearing loss, lateral displacement of inner canthi

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- Associated features
  - Type I: Developmental delay (variable), asymmetric digits, polycystic kidney disease
  - Type II: Partial reduplication of the hallux and 1st metatarsal, bilateral polydactyly of the hands and polysyndactyly of the feet

- **Oto-palatal-digital syndrome**
  - Inheritance
    - Type I: X-linked (intermediate expression in female carriers)
    - Type II: X-linked (mild expression in female carriers)
  - Genetic loci
    - Type I: Xq28
    - Type II: Xq28
  - Relevant features
    - Type I: Moderate conductive deafness, cleft palate, frontal and occipital bossing, thickened frontal bone and skull base, hypertelorism, small mouth and nose
    - Type II: Conductive hearing loss, cleft palate, frontal bossing, auricular anomalies (low set, malformed), flat nasal bridge, micrognathia, small mouth, down-slanting palpebral fissures
  - Associated features
    - Type I: Broad distal digits with short nails, developmental delay, small stature and trunk, pectus excavatum
    - Type II: Late closure of fontanels, microcephaly, flexed overlapping finger, short broad thumbs and hallux, bowing of radius, ulna, femur and tibia, flattened vertebral bodies

- **Pierre Robin sequence**
  - Genetic loci: 2q32.3–q33.2 (in some cases)
  - General: Up to 80% of children with Pierre Robin sequence are also affected by a named syndrome (most commonly Stickler).
  - Relevant features: Triad = glossoptosis, micrognathia, cleft palate; airway and feeding difficulty.
  - Associated features: Developmental delay, hypospadia, cryptorchidism, hernias
  - For further information on Pierre Robin sequence, see Chapter 6.8.

- **VATER association**
  - Inheritance: Sporadic
  - Genetic loci: No chromosomal anomaly identified
  - Relevant features: Malformation of the vertebrae, anus, trachea, esophagus, radial and renal structures
  - VACTERL association: As above + cardiac and limb anomalies

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- **Velocardiofacial syndrome (Shprintzen syndrome, 22q11 deletion syndrome)**
  - Inheritance: Autosomal dominant
  - Genetic loci: 22q11 deletion
  - General: 10% associated with DiGeorge syndrome (hypocalcemia, thymic aplasia)
  - Relevant features: Velopharyngeal incompetence, high arched/cleft palate (overt or submucous), conductive hearing loss, square nasal root, prominent nasal dorsum, deficiency of nasal alae, retrognathia, medial displacement of carotid artery (25%)
  - Associated features: Mild developmental delay, short stature, cardiac defects (85%), increased risk of psychiatric disorders, slender hands/fingers
- **Van der Woude syndrome**
  - Inheritance: Autosomal dominant
  - Genetic loci: 1q32 (incomplete penetrance)
  - Relevant features: cleft lip and/or cleft palate, lower lip pits, absent central/lateral incisors, canines, bicuspids
  - Associated features: Developmental delay, hypospadia, cryptorchidism, hernias

The following syndromes (where hearing loss is a primary feature) are covered in Chapter 6.11:

- Alport syndrome
- Apert syndrome
- Branchio-oto-renal syndrome
- Connexin-26 deafness
- Crouzon syndrome
- Goldenhar syndrome
- Jervell and Lange-Nielsen syndrome
- Neurofibromatosis (NF1 and NF2)
- Norrie syndrome
- Osteogenesis imperfecta
- Oto-palato-digital syndrome
- Pendred syndrome
- Stickler syndrome
- Treacher-Collins syndrome
- Usher syndrome
- Wildervanck syndrome
- Waardenburg syndrome
- X-linked hearing loss

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◆ Evaluation

History

- Three-generation family history
- Parental consanguinity
- Parental ethnicity
- Maternal, paternal age
- Teratogen exposure
- Prior pregnancies, miscarriages, still births
- Illness during pregnancy

Physical Exam

A complete head and neck exam with a focus on dysmorphologic examination is needed. A general examination should be undertaken to identify syndromic features (see above for description of characteristic syndromic features).

Imaging

Specific imaging recommendations are based on associated features of specific syndromes. CT or MRI of the brain will often be undertaken.

Labs

- Karyotyping
- DNA analysis
- Molecular genetic testing
- Fluorescence in situ hybridization (FISH) evaluation
- Targeted studies for metabolic disorders

Other Tests

None are routinely done.

◆ Treatment Options

- Genetic counseling for patient and family
- Referral to medical and surgical specialists based on specific features of syndrome
- Developmental assessment

Further Reading

National Institutes of Health. Online Mendelian Inheritance in Man. Available at: http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=OMIM
Tewfik TL, Manoukian JJ, eds. Congenital Anomalies of the Ear, Nose, and Throat. New York: Oxford University Press; 1997

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6.10 Diseases of the Adenoids and Palatine Tonsils

6.10.1 Adenotonsillitis

◆ Key Features

- Adenotonsillitis is most commonly viral in etiology.
- Bacterial etiology is similar to acute otitis media (OM).
  - Group A β-hemolytic *Streptococcus pneumoniae*
  - *Moraxella catarrhalis*
  - *Haemophilus influenzae*
- Chronic infection is typically polymicrobial.
- There is growing evidence for the role of biofilm.

Acute adenotonsillitis most commonly presents in children aged 5 to 10 years of age and young adults 15 to 25 years of age. The primary consideration in its accurate diagnosis and treatment is prevention of secondary complications particularly associated with group A β hemolytic *Streptococcus pneumoniae* including rheumatic fever and poststreptococcal glomerulonephritis. Suppurative complications avoided by early and appropriate management include peritonsillar abscess and deep neck space infection.

◆ Epidemiology

The average incidence of all acute upper respiratory infections is five to seven per child per year. It is estimated that children have one streptococcal infection every 4 to 5 years. Group A *streptococcus* is isolated in 30.0 to 36.8% of children with pharyngitis and asymptomatic carriage of group A *streptococcus* is ~10.9% for children aged 14 or less.

◆ Clinical

Signs

- Erythremic, exudative palatine tonsils
- Tonsilloliths
- Trismus
- Cervical adenopathy
- Palatal petechiae (infectious mononucleosis)

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Symptoms

- Halitosis
- Sore throat
- Odynophagia
- Purulent rhinorrhea
- Postnasal drip
- Nasal obstruction
- Fever

Differential Diagnosis

- Adenotonsillar hypertrophy
- Acute pharyngitis (bacterial or viral)
- Peritonsillar abscess
- Infectious mononucleosis
- Lymphoma, leukemia, or other neoplasm (unilateral/asymmetric tonsillar enlargement)

◆ Evaluation

Physical Exam

In the head and neck exam, focus on examination of the oral cavity, inspection of palatine tonsils looking for enlargement, erythema, peritonsillar cellulitis, abscess, and exudates. Palpation of the neck is performed to assess for cervical adenopathy.

Imaging

Contrast-enhanced CT if concerned about retropharyngeal or deep neck space infection.

Labs

Complete blood count (CBC) with differential, monospot, if clinically indicated.

Other Tests

Do a throat swab for culture and sensitivity and latex agglutination tests for group A β-hemolytic *Streptococcus*.

◆ Treatment Options

Medical

Analgesics and antipyretics are prescribed.

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Relevant Pharmacology

Antibiotic therapies include:

- **Amoxicillin:** Penicillin-based β-lactam antibiotic with bactericidal action due to interference with bacterial cell wall synthesis
  - 45 mg/kg per day divided every 8 hours for 7–10 days
- **Amoxicillin + Clavulanate:** Clavulanate has itself little antibacterial action but it is a potent β-lactamase inhibitor protecting the penicillin-based antibacterial action.
  - 45 mg/kg per day (amoxicillin component) divided every 12 hours for 7–10 days
- **Azithromycin:** Macrolide, semisynthetic derivative of erythromycin. Bactericidal action is through inhibition of protein synthesis via binding to the 50S ribosomal RNA subunit.
  - 10 mg/kg × 1 dose then 5 mg/kg per day for 5 days
- **Cefuroxime axetil:** Second-generation cephalosporin's bactericidal action is due to the inhibition of peptidoglycan synthesis interfering with cell wall synthesis similar to penicillin's.
  - 30 mg/kg per day divided every 12 hours for 7–10 days

Surgical

Surgery entails a delayed tonsillectomy and/or adenoidectomy for recurrent disease (**Table 6.5**). Currently, it is rare to perform a tonsillectomy in the setting of an acute infection (quinsy tonsillectomy). The presence of a peritonsillar abscess requires acute incision and drainage; removal of the tonsil to allow drainage is rarely needed.

◆ Complications

See Chapter 6.10.2 for a detailed discussion of complications.

Primary tonsillar hemorrhage often requires operative management (0.5 to 2.2 per 100). Secondary (delayed) tonsillar hemorrhage is often managed conservatively with close observation (0.1 to 3 per 100).

◆ Outcome and Follow-Up

In an uncomplicated surgery in an otherwise healthy child, it is a same-day procedure with analgesia and possibly postoperative antibiotics depending on the technique and the surgeon's preference. Hospital admission criteria are also discussed in Chapter 6.10.2. Follow-up is 3 to 6 weeks after tonsillectomy and adenoidectomy.

◆ ICD-9 Codes

464	Acute tonsillitis
474.00	Chronic tonsillitis

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Table 6.5 Adenotonsillectomy Indications and Contraindications

Absolute Indications
Complication of sleep apnea secondary to tonsillar hypertrophy (i.e., cor pulmonale)
Suspected tonsil malignancy
Febrile convulsions secondary to tonsillitis
Tonsillar hemorrhage
Severe failure to thrive with enlarged tonsils
Relative Indications
Obstructive sleep apnea
Recurrent acute tonsillitis
5–7/yr for 1 yr
5/yr for 2 yr
3/yr for 3 yr
>2 wk of school missed over 1 yr
Peritonsillar abscess – persistent or recurrent
Chronic tonsillitis (throat pain, halitosis, cervical adenitis)
Severe odynophagia
Tonsillolithiasis (if severe, persistent)
Acquired dental or orofacial abnormalities
Chronic carrier of *Streptococcus*
Recurrent/chronic otitis media (adenoidectomy alone)
Contraindications
Cleft palate*
Velopharyngeal insufficiency*
Bleeding diathesis (unless correctible medically; i.e., von Willebrand patients can undergo surgery with appropriate treatment perioperatively.

*Relative contraindication for tonsillectomy, absolute contraindication for adenoidectomy (although partial adenoidectomy may be considered)

Further Reading

Gislason T, Benediktsdóttir B. Snoring, apneic episodes, and nocturnal hypoxemia among children 6 months to 6 years old. An epidemiologic study of lower limit of prevalence. Chest 1995;107(4):963–966

Hoffmann S. The throat carrier rate of group A and other beta hemolytic streptococci among patients in general practice. Acta Pathol Microbiol Immunol Scand [B] 1985;93(5):347–351

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6.10.2 Adenotonsillar Hypertrophy

◆ Key Features

- Adenoids and palatine tonsils are a common source of upper airway obstruction in childhood.
- Peak size is achieved by ~5 years of age.
- Airway compromise presents as obstructive sleep apnea (OSA).

Adenotonsillar hypertrophy leads to a spectrum of airway obstruction in children and is a common indication for surgery in the pediatric age group. Adenoids and palatine tonsils enlarge over the first to fifth years of life. Progressive involution occurs by 12 to 18 years of age, though the course is highly variable and is influenced by both allergy and the frequency of recurrent tonsillar and upper respiratory tract infections.

◆ Epidemiology

Adenotonsillar hypertrophy is the most common cause of sleep-related upper airway obstruction in children. Forty percent of children snore; the incidence of true obstructive apnea is estimated to be 3%.

◆ Clinical

Signs

- Mouth open posture
- Failure to thrive and feeding difficulties
- Hyponasal speech
- Adenoid facies (high arched palate, flattened midface, "allergic shiners," mouth-open posture)
- Behavioral disturbances
- Daytime somnolence

Symptoms

- Snoring and/or apneic pauses
- Restless sleep
- Choking or gagging noises while asleep
- Hyperactivity, attention deficit symptoms
- Enuresis
- Nasal obstruction

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Differential Diagnosis

- Acute or chronic tonsillitis, adenoiditis (bacterial or viral)
- Acute pharyngitis (bacterial or viral)
- Peritonsillar abscess
- Infectious mononucleosis
- Lymphoma
- Other causes of upper airway obstruction include nasal polyposis, deviated nasal septum, and rhinitis, sinusitis (infectious, allergic, or nonallergic).

◆ Evaluation

History

History usually indicates irregular snoring with gasping or witnessed pauses and daytime somnolence. A history of behavior problems, attention-deficit hyperactivity disorder, and enuresis should be elicited. Any previous sleep studies should be reviewed. It is important to ask for any family history of known bleeding disorders, or a patient or family history of abnormal bruising or bleeding.

Physical Exam

Complete head and neck exam with focus on

1. Assessment of the status of the middle ear (there is an increased incidence of acute OM and OME with adenoid hypertrophy)
2. Examination of the oral cavity, inspection, and grading of palatine tonsils (grade 1 is tonsils within the tonsillar pillars, grade 2 (<50% obstruction), grade 3 (>50% obstruction) to grade 4 where tonsils abut in the midline)
3. Exclusion of obvious craniofacial malformation or syndromic features; assess for cleft palate

Consider the direct examination of adenoid size using small flexible fiberoptic scope, especially in the cooperative child.

Imaging

Consider a lateral neck plain film to assess the size of the adenoid pad.

Labs

A CBC should be done, with partial thromboplastin time (PTT) and prothrombin time/international normalized ratio (PT/INR) tests preoperatively if there is any family history or suspicion for potential coagulopathy. A platelet function test (PFA-100 analyzer) is a rapid inexpensive screen for platelet dysfunction, although it is nonspecific. A positive test should prompt von Willebrand studies and a referral to hematology.

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Other Tests

Nasal endoscopy should be performed to assess the degree of nasopharyngeal obstruction. Rhinomanometry is rarely used to assess the degree of nasal obstruction.

An otherwise generally healthy child with parents who provide a good description of irregular snoring with gasping, pausing, or choking noises, and an exam that reveals obvious obstructive hypertrophy does *not* need a polysomnography; proceed with an adenotonsillectomy. If the child has an unconvincing exam or the parents cannot provide a good history, polysomnography is helpful. If the child has other comorbidities, and *especially* if the child has neurologic problems, polysomnography is important, as central rather than obstructive apnea may be occurring. Central apnea will not be improved by an adenotonsillectomy.

Pathology

Hyperplasia of the lymphoid tissue of the adenoids and tonsils is found.

◆ Treatment Options

Medical

- Watchful waiting
- Topical nasal steroids
- Antibiotics may shrink tonsils occasionally.
- Continuous positive airway pressure for OSA (see Chapter 1.8.1)

Surgical

The surgical procedures employed include adenoidectomy, tonsillectomy, and adenotonsillectomy. For instrumentation, most surgeons currently use the electrocautery. Also, some surgeons perform cold dissection, use a bipolar radiofrequency ablation device (coblation), or use a microdébrider for so-called intracapsular or subtotal tonsillectomy. Other devices have been introduced and marketed, but are less widely used.

◆ Complications

Bleeding

The most common significant complication is postoperative hemorrhage, which occurs at a rate of 2 to 3%. The patient/family must understand risk of delayed bleeding and the need to present to the emergency department promptly if there is any bleeding. Children in rural areas should not be allowed to be more than 30 minutes from a hospital for 2 weeks.

Primary tonsillar hemorrhage (0.5–2.2 per 100) often requires operative management. This is due either to a failure to control a vessel that was likely in spasm or to an unrecognized clotting problem, typically platelet dysfunction.

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Table 6.6 Management Options for Posttonsillectomy Bleeding

If active bleeding, go directly to the operating room for definitive control
If inactive or scant bleeding: Admission with 24-hr observation If rebleed, go to operating room. If no bleeding for 24 hr, discharge
All patients: Check hemoglobin, hematocrit, PT, PTT. Establish intravenous access, hydrate.
Possible measures to control mild/scant bleeding in emergency department: silver nitrate or other chemical cautery

Abbreviations: PT, prothrombin time; PTT, partial thromboplastin time

Secondary (after first 24 hours) tonsillar hemorrhage (0.1–3 per 100) is often managed conservatively with close observation. This is most common at ~1 week postoperatively.

Recurrent secondary hemorrhage should prompt a thorough hematologic evaluation and mandates admission with close observation based on concern for a possible "sentinel" bleed (**Table 6.6**).

Other Complications

Early postoperative airway obstruction may require humidified oxygen, steroids, and reintubation. Chronically obstructed patients are at risk of postoperative pulmonary edema, diagnosed with physical exam and chest radiography. This may require furosemide, humidified oxygen, chest physiotherapy, positive end expiratory pressure ventilation, and reintubation.

Postoperative dehydration may require readmission. Treat with IV hydration, antiemetics, analgesia as needed, and parental education.

Nasal reflux is commonly mild and resolves; if severe or persistent this may be difficult to correct. Speech therapy can be helpful.

Nasopharyngeal stenosis/scarring is uncommon and extremely difficult to correct. Prevention is best.

Airway fire: This should be extremely rare. One should check for cuff leak before operating; should not leak below 20. Communicate with the anesthesiologist, and be sure FIO_2 is kept less than 40%. If there is a fire, then immediately extubate, turn off all the oxygen, and reintubate.

◆ Outcome and Follow-Up

Close postoperative monitoring in children with OSA, given the risk of ongoing or worsening apnea in the immediate postoperative period. Following tonsillectomy, children with a positive preoperative sleep study and/or any children 3 years or younger are typically observed overnight with continuous pulse oximetry, although there is a lack of consensus of this issue.

Other measures: Liquid/soft diet for 2 weeks postoperative. Pain management is important to enable the patient to maintain adequate hydration.

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Although there is no consensus, a recommended combination is Tylenol (Ortho-McNeil-Jansson Pharmaceuticals, Titusville, NJ) liquid up to 15 mg/kg/dose three or four times daily, using plain oxycodone liquid 0.1 mg/kg/dose (5 mg maximum) every 4 hours as needed for breakthrough pain. Separating the Tylenol and the narcotic enables the patient to minimize narcotic use in many cases. Nonsteroidal antiinflammatory drugs (NSAIDs) are avoided due to concern for platelet inhibition, although some studies suggest this does not increase bleeding risk. Oral liquid antibiotic is used for 1 week postoperatively, usually amoxicillin if the patient is nonallergic. Intraoperatively, a single dose of dexamethasane 0.5 mg/kg IV is helpful to reduce postoperative pain and swelling; the dose is typically 4 to 12 mg.

Continued routine follow-up if conservative treatment approach adopted. Repeat sleep study may be considered 3–6 months postoperatively if symptoms persist. Follow-up is scheduled 3 to 6 weeks after adenotonsillectomy.

Adenotonsillectomy achieves a 90% success rate for childhood sleep-disordered breathing. Of all tonsillectomies and adenoidectomies performed in the United States each year, 75% are performed to treat sleep-disordered breathing.

◆ ICD-9 Codes

780.57 Unspecified sleep apnea
474.1 Hypertrophy of tonsils, adenoids

Further Reading

Brodsky L. Tonsillitis, tonsillectomy, and adenoidectomy. In: Bailey BJ, Johnson JT, Newlands SD, et al, eds. Head and Neck Surgery – Otolaryngology. 2nd ed. Philadelphia, PA: Lippincott-Raven; 1998:1221–1235

6.11 Pediatric Hearing Loss

◆ Key Features

- Age at identification has significantly decreased since the advent of newborn hearing screening.
- Early identification and treatment of hearing loss is essential to obtain an optimal outcome.
- Connexin-26 related deafness is the most common cause of inherited hearing loss.
- Acquired hearing loss often occurs within the antenatal period.
- Perinatal history is key to identifying risk factors for both congenital and acquired hearing loss.

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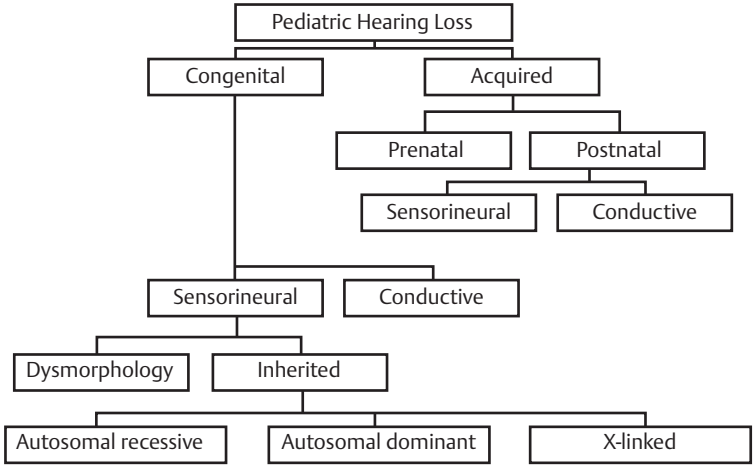

Fig. 6.3 Types of pediatric hearing loss.

The onset of hearing loss in children regardless of etiology often occurs prior to the development of language. The disorders causing both congenital and acquired hearing loss range from the simple and common to the rare and complex. The list of potential diagnoses below is not exhaustive; additional syndromes with hearing loss as a minor characteristic can be found in Chapter 6.9. For the purpose of this chapter, we will examine hearing loss using the structure seen in **Fig. 6.3**, with the caveat that some diagnoses may fall under more than one category.

◆ Epidemiology

Hearing loss is the most common congenital sensory deficit with an estimated incidence of 1 to 2 per 1000 children. Likewise acquired loss is exceedingly common with most children experiencing at a minimum a transient conductive hearing loss (CHL) due to chronic serous otitis. An estimated 50% of childhood sensorineural hearing loss (SNHL) is due to genetic factors with this proportion increasing with the ongoing detection of new mutations. Genetic causes may eventually account for a large proportion of the hearing loss currently labeled as unknown in etiology. For cases of congenital hereditary hearing loss, about two-thirds are nonsyndromic. In the setting of genetic-related deafness, most cases (70–80%) are autosomal recessive, roughly 20% are autosomal dominant, and the remainder are due to X-linked chromosomal or mitochondrial anomalies. About 50% of the cases of congenital SNHL are considered nonhereditary (i.e., acquired), and most of these are due to TORCHES (toxoplasmosis, rubella, cytomegalovirus, herpes simplex encephalitis, and otosyphilis) infections, sepsis, or severe prematurity.

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◆ Clinical

Signs

- Speech delay or regression of speech
- Vestibular dysfunction
- Delays in ambulation
- Gait disturbances
- Otorrhea

Symptoms

- Hearing loss (may be progressive or fluctuating)
- Tinnitus
- Vertigo
- Otalgia
- Aural fullness

◆ Congenital Sensorineural Hearing Loss

Dysmorphologies

Most cochlear dysmorphologies are membranous in nature (80–90%) and not identifiable on CT; the remainder have a discernible bony anomaly that is identifiable on CT imaging.

Mondini Dysplasia

- Incomplete partition of cochlea
- Autosomal dominant
- Unilateral or bilateral hearing loss may be progressive or fluctuating (interspersed with normal hearing)
- CT: Cystic dilation of cochlea with absence of modiolus, enlarged vestibular aqueduct, semicircular canal anomalies
- Associated with Pendred, Waardenburg, and Treacher-Collins syndromes and branchio-oto-renal syndrome (see below for description)

Michel Aplasia

- Autosomal dominant
- Anacusis
- CT: Absent cochlea and labyrinth, hypoplasia of petrous pyramid

Alexander Aplasia

- Autosomal recessive or sporadic inheritance
- Most common inner ear aplasia

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- High-frequency loss with residual low-frequency hearing
- Aplasia of cochlear duct (membranous defect)
- CT: No characteristic features
- Associated with congenital rubella, and Jervell Lange-Neilsen, Usher, and Waardenburg syndromes

Scheibe Aplasia

- Autosomal recessive
- Most common inner ear dysplasia
- Partial to complete aplasia of cochlea and saccule (pars inferior) with normal semicircular canals and utricle (pars superior)
- CT: No characteristic features (membranous defect)
- Associated with Usher and Waardenburg syndromes

Enlarged Vestibular Aqueduct

- Most common radiologic deformity of the inner ear
- Progressive SNHL that may be sudden or stepwise decrease
- Can occur in isolation or with Mondini malformation
- Associated with progressive and/or fluctuating vestibular dysfunction and Pendred syndrome
- Associated with perilymph gusher

Bing-Siebenmann Dysplasia

- Rare, complete dysplasia of membranous labyrinth

Inherited Disorders

Autosomal Recessive

Connexin 26

- Chromosomal mutation on 13q11
- Most common genetic cause of deafness (>50% of recessive nonsyndromic hearing loss)
- Most commonly autosomal recessive (90 mutations identified)
- Can be autosomal dominant (nine mutations identified)
- Gene mutation leads to quantitative and qualitative defects in protein coding for gap junctions (GJB2), which are responsible for maintaining endolymphatic potential in cochlea [K^+].
- 35delG is the most common mutation; however, >100 mutations have been identified.

Usher Syndrome (**Table 6.7**)

- Chromosome arm 14q (type I), chromosome arm 1q32 (type II)
- SNHL, retinitis pigmentosa with or without mental retardation, and cataracts

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Table 6.7 Classification of Usher Syndrome

Type	Sensorineural Hearing Loss	Vestibular Function	Blindness
I	Profound	Areflexia	Early adulthood
II	Moderate to severe	Normal	Midadulthood
III	Progressive	Progressive	Variable

Pendred Syndrome

- Chromosomal mutation on 7q coding for pendrin (sulfate transporter)
- SLC26A4 is the most common mutation.
- Defect in tyrosine iodination
- Severe to profound SNHL
- CT: Mondini malformation or isolated enlarged vestibular aqueduct
- Associated with euthyroid multinodular goiter in childhood

Jervell and Lange-Nielsen Syndrome

- Chromosomal mutation on 11p15 as well as 3, 4, 7, 21
- Bilateral SNHL
- Long Q-T syndrome (treated with β-blockers)

Goldenhar Syndrome

- Hemifacial microsomia
- Chromosomal mutation on 9
- Developmental anomalies of the first and second branchial arches affecting facial nerve, stapedius muscle, semicircular canals, and oval window
- Associated with preauricular tags, pinnae anomalies, aural atresia, facial asymmetry, colobomas, and mental retardation

Autosomal Dominant

Waardenburg Syndrome

- Chromosomal mutation on 2q and 14q
- Unilateral or bilateral SNHL and/or vestibular dysfunction
- Associated with pigment anomalies [white forelock, heterochromia iritides (different colored irises)], telecanthus, synophrys (unibrow), skeletal anomalies, Hirschsprung megacolon

Stickler Syndrome

- Progressive arthroophthalmopathy
- Chromosomal mutation on 6p and 12q
- Variable expression

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- Progressive SNHL and/or conductive loss (eustachian tube dysfunction due to cleft palate)
- Associated with myopia and/or retinal detachment, cataracts, Pierre Robin sequence, marfanoid habitus, joint hypermobilization, and early arthritis

Treacher-Collins Syndrome

- Mandibulofacial dysostosis
- Chromosomal mutation on 5
- Conductive hearing loss: EAC atresia, ossicular malformations, replacement of tympanic membrane with bony plate
- Sensorineural hearing loss: widened cochlear aqueduct
- Associated with aberrant facial nerve, auricular anomalies, preauricular fistulas, mandibular hypoplasia, coloboma, palate defects, and downward-slanting palpebral fissures

Branchio-Oto-Renal Syndrome

- Chromosome mutation on 8q
- EYA1 and SIX1 mutations have been identified.
- Developmental anomalies of the branchial arches and kidneys
- Variable SNHL
- CT: Possible Mondini deformity
- Associated with auricular deformities, preauricular pits, fistulas, tags, mild renal dysplasia progressing to agenesis

Neurofibromatosis

- Neurofibromatosis type 1 (NF1): Long arm of chromosome 17; 5% risk of unilateral acoustic neuroma
- Neurofibromatosis type 2 (NF2): Long arm of chromosome 22; frequently bilateral acoustic neuromas, multiple meningiomas
- Progressive profound SNHL due to acoustic neuroma
- Associated with café-au-lait spots, groin/axillary freckling, cutaneous, CNS and peripheral nerve neurofibromas, Lisch nodules (eye hamartomas), and pheochromocytomas

Apert Syndrome

- Acrocephalosyndactyly
- Chromosomal mutation on 10
- Autosomal dominant or sporadic inheritance
- CHL due to stapes fixation
- CT: patent cochlear aqueduct, large subarcuate fossa
- Associated with syndactyly, midface anomalies including hypertelorism, proptosis, saddle nose deformity, high-arched palate, trapezoid mouth, and craniofacial dysostosis

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Crouzon Syndrome
- Craniofacial dysostosis
- Chromosomal mutation on 10q; CHL to aural atresia and ossicular deformities
- Associated with cranial synostosis, small maxilla, hypertelorism, exophthalmos, prognathic mandible, and short upper lip

Osteogenesis Imperfecta
- Chromosomal mutation on 5p or 17q
- Typically autosomal dominant inheritance although a familial recessive form has been described
- Disorder of type I collagen (quantitative and qualitative collagen defect); progressive conductive, mixed, or sensorineural hearing loss
- Associated with hypermobility of joints and ligaments, bone fragility, and blue sclera

X-Linked

Alport
- Chromosome Xq, some autosomal dominant mutations in chromosome 2q
- Abnormality of type IV collagen formation in basement membrane
- Progressive SNHL due to degeneration of organ of Corti and stria vascularis within first decade
- Associated with progressive nephritis (hematuria, proteinuria, chronic glomerulonephritis, uremia), myopia, and cataracts

Oto-palato-digital Syndrome (see also Chapter 6.9)
- CHL due to ossicular malformations
- Associated with cleft palate, broad fingers and toes, hypertelorism, mental retardation, and short stature

Norrie Syndrome
- Chromosomal mutation on Xp11
- Progressive SNHL (in one third of patients; in second or third decade)
- Rapidly progressive blindness (bilateral pseudoglioma, exudative vitreoretinopathy, and ocular degeneration)

Wildervanck Syndrome
- Dominant X-linked
- SNHL (one third of cases)
- Associated with CN VI paralysis and Klippel-Feil malformation (fusion of cervical vertebrae)

X-Linked Hearing Loss
- Chromosomal mutation on Xq
- Rare syndrome with mixed hearing loss due to stapes fixation and perilymph gushers

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◆ Congenital Conductive Hearing Loss

Atresia

Atresia may be unilateral or bilateral.

Syndromic

Treacher-Collins syndrome, Apert syndrome, Crouzon syndrome, oto-palato-digital syndrome, osteogenesis imperfecta, X-linked hearing loss (see above for syndrome descriptions)

◆ Acquired Hearing Loss

Acquired hearing loss is often due to prenatal or perinatal insult, such as infection.

Acquired Prenatal

Infection *before* birth results in hearing loss.

Rubella

- Hair cell loss, atrophy of the organ of Corti, thrombosis of stria vascularis
- Severe to profound SNHL and/or delayed endolymphatic hydrops
- Associated with mental retardation, lower limb deformities, anemia, cataracts, cardiac malformations, microcephaly, and thrombocytopenia

Syphilis

- *Treponema pallidum*
- Often fatal
- Profound SNHL (within first 2 years, or in second or third decade)
- Hennebert's sign (nystagmus with fluctuating pressure on EAC in the presence of an intact tympanic membrane – in delayed congenital syphilis)
- Delayed endolymphatic hydrops
- Penicillin for acquired infection and/or corticosteroids for SNHL

Cytomegalovirus (CMV)

- Mild to profound SNHL
- May be unilateral and/or progressive
- Associated with hemolytic anemia, microcephaly, mental retardation, hepatosplenomegaly, cerebral calcifications, and jaundice

Hypoxia

- SNHL and/or auditory neuropathy
- Risk factors include birth anoxia, postnatal hypoxia and/or ventilatory support, and extracorporeal membrane oxygenation (ECMO)

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Hyperbilirubinemia

- SNHL and/or auditory neuropathy

Acquired Postnatal

Infection or injury *after* birth results in hearing loss.

Sensorineural Loss

Meningitis

- SNHL typically bilateral, permanent, profound to severe (15–20% of cases)
- Onset of SNHL occurs early in this disease.
- Bacterial etiology: *Haemophilus influenzae* (most likely to cause hearing loss), *Streptococcus pneumoniae* (most common cause of meningitis in children; most likely to cause labyrinthine ossification)
- Bacteria and their toxins likely access inner ear via the cochlear aqueduct and the internal auditory canal (IAC).
- Inflammatory, hypoxic injury of neural elements may play a role.
- Repeated auditory brainstem response assessment starting early in the course of disease
- CT: May have labyrinthine ossification. Lateral semicircular canal is often first to ossify. If child medically stable and fulfills audiologic candidacy, cochlear implantation should occur immediately ($\leq$6 weeks) if scan shows fibrosis, because once the cochlea has ossified, implantation then requires cochlear drill-out, allowing only a partial electrode, which portends a poorer outcome.

Trauma

- Possible temporal bone fracture
- SNHL due to fracture through otic capsule, stapes subluxation with perilymphatic leak or excessive displacement and shearing of basilar membrane (leads to a high frequency loss).
- CHL due to soft tissue and debris in the EAC, tympanic membrane perforation (20–50%), hemotympanum (20–65%), ossicular dislocation or injury (incus most commonly dislocated, more commonly associated with longitudinal fracture).
- Associated with facial nerve injury, vertigo, and posttraumatic benign paroxysmal positional vertigo

Auditory Neuropathy/Dyssynchrony

- Disorder of central processing
- Poor discrimination scores out of keeping with pure tone thresholds (absent auditory brainstem response, normal OAEs)
- Risk factors include hyperbilirubinemia, hypoxia or mechanical ventilation, low birth weight, congenital anomalies of the CNS, Stevens-Johnson syndrome
- May accompany neurologic diagnosis (Friedrich ataxia, Ehlers-Danlos syndrome, Charcot-Marie-Tooth syndrome)

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- Most commonly acquired although inherited variants exist
- May perform well with cochlear implantation due to suprathreshold synchronous stimulation it supplies to auditory nerve

Conductive Loss

- Cholesteatoma
  - Ossicular erosion leading to maximal conductive loss
- Tympanic membrane perforation
  - CHL (30–50 dB)
  - Successful repair may not improve hearing.
  - Perforation due to acute OM, chronic OM, barotraumas, complication of tympanostomy tube insertion or middle ear surgery
  - Should obey water precautions, particularly in bath water and lake water
- Chronic serous otitis
- Trauma
  - See above for full description.
- Ear foreign body
  - See Chapter 2.1.4.

◆ Evaluation (**Table 6.8**)

Physical Exam

See Chapter 2.4.2 for pediatric audiologic assessments. A complete head and neck exam should include otoscopy, pneumatoscopy, Rinne and Weber tuning fork tests if age-appropriate, and inspection of the auricle and preauricular skin for pits or tags. A clinical examination of the vestibular system appropriate for the child's age should be undertaken, as well as a general examination to identify syndromic features (see above for differential diagnosis for characteristic syndromic features).

Imaging

High-resolution CT of the temporal bones will identify cochleovestibular anomalies. Consider an MRI with attention to the IAC to examine for the presence and size of the auditory nerve, white matter changes due to sequelae of prematurity, CMV, hyperbilirubinemia, or in cases of neurofibromatosis.

Labs

- Pendred syndrome: thyroid-stimulating hormone (TSH) test
- Alport, branchio-oto-renal syndrome: Urinalysis, BUN (blood urea nitrogen), creatinine
- TORCH: IgM assay (toxoplasmosis, syphilis, rubella, CMV, herpes simplex)

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Table 6.8 Suggested Evaluation for Congenital Sensorineural Hearing Loss

Syndromic
Refer to geneticist
Consider: electrocardiography, urinalysis
Other imaging as directed by specific syndrome
Nonsyndromic
Tests for possible acquired causes:
RPR/ FTA-ABS
TORCH IgM assay
Genetic testing:
Various genetic panels, typically testing for Connexin mutations, others
All
CT of temporal bone
Referral to ophthalmology
Serial hearing testing to follow possible progression

Abbreviations: CT, computed tomography; IgM, immunoglobulin M; RPR/FTA-ABS, rapid plasma reagin/fluorescent treponemal antibody absorbed; TORCH, toxoplasmosis, rubella, cytomegalovirus, herpes simplex encephalitis, and otosyphilis.

- Rubella: Viral culture of amniotic fluid, urine, and throat
- Syphilis: fluorescent treponemal antibody absorbed (FTA-ABS), venereal disease research laboratory (VDRL)

Other Tests

- Repeated audiometric assessment with tympanometry appropriate for age (**Table 6.9**)
- Assessment of visual acuity due to exponential increase in morbidity in presence of dual sensory impairment
- Genetic evaluation, counseling: Connexin-26, syndromic and/or inherited loss
- Jervell and Lange-Nielsen syndromes: Electrocardiogram to detect prolonged QT interval
- Pendred syndrome: Thyroid ultrasound
- Branchio-oto-renal syndrome: Renal ultrasound, pyelogram

◆ Treatment Options

Medical

Appropriate and timely fitting of amplification (by 6 months if identified by newborn hearing screening) with a frequency modulation (FM) system or

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Table 6.9 Audiometric Assessment by Age

Birth to 6 mo
Behavioral observation audiometry
Otoacoustic emission
Auditory brainstem response
6 mo to 3 yr
Visual response audiometry
Otoacoustic emission
Auditory brainstem response
3 to 6 yr
Conventional play audiometry
>6 yr
Standard audiometry

tactile aids. An auditory verbal therapist or a total communication program, including American sign language where appropriate should be considered. Liaise with the school, teachers, and the educational audiologist.

Surgical

- Tympanostomy tubes: Mixed loss due to chronic serous OM
- Cochlear implantation for profound bilateral SNHL (see Chapter 2.5.4)
- Bone-anchored hearing aid for aural atresia, chronic OM (see Chapter 2.5.5)
- Surgical correction of atretic ear and canal
- Middle ear exploration and/or ossiculoplasty, tympanoplasty for suspected congenital or acquired conductive loss
- Auditory brainstem implant (see Chapter 2.5.5) for:
  - Bilateral acoustic neuroma in NF2
  - Cochlear or auditory nerve agenesis (i.e., Michel aplasia)

◆ Complications

Treat TM perforation due to chronic serous OM or as a complication of tympanostomy tube insertion with delayed tympanoplasty once a dry ear is established. Treat OM aggressively with antibiotics and tympanostomy tubes in children following cochlear implantation due to risk of meningitis.

◆ Outcome and Follow-Up

Postoperatively, apply analgesia and/or systemic antibiotics where the auditory prosthesis has been placed. Children receiving cochlear implantation should receive pneumococcal and meningitis vaccination as prophylaxis prior to implantation.

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Ongoing and repeated audiologic assessment should be maintained with attention to the child's speech and language development.

With a child with tympanostomy tubes, follow up every 6 months until the tubes have extruded and the myringotomy site has closed without reaccumulation of effusion.

◆ ICD-9 Code

389 Hearing loss

Further Reading

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6.12 Infectious Neck Masses in Children

◆ Key Features

- Cervical lymphadenitis is the most common infectious neck mass in children, but an abscess is also a possibility.
- The age of the patient, the location, and the timeframe to development of adenitis are important clues to the underlying etiology.
- The diagnosis is commonly based on history and physical exam, not laboratory findings.
- Medical management is appropriate for many causes.

Enlarged or inflamed lymph nodes are the cause of 95% of pediatric neck masses. A thorough physical examination is necessary because the lymph node groups are associated with different diseases, which will dictate proper management. Most of the lymphatics from the head and neck region drain to the submaxillary and deep cervical lymph nodes, which explains why these nodes are most commonly affected by cervical lymphadenitis. In the absence of cervical adenopathy, enlargement of the supraclavicular nodes can be indicative of thoracic or abdominal disease. The most common causes of isolated right supraclavicular node enlargement are Hodgkin and non-Hodgkin lymphoma. Isolated enlargement of the left supraclavicular nodes is most commonly associated with intraabdominal tumor or inflammation (Troisier sign).

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◆ Etiology

The underlying etiology of cervical lymphadenitis can be predicted based on the patient's age. In neonates, group B streptococci are the most common cause of lymphadenitis. *Staphylococcus aureus* is usually the causative organism in patients 2 months to 1 year old. Along with *Bartonella henselae* (cat-scratch disease) and nontuberculous mycobacteria, *S. aureus* is also a common cause in 1- to 4-year-old patients. These organisms can be the cause of cervical adenitis in older patients along with tuberculosis, anaerobic bacteria, and toxoplasmosis. The timeframe is also an important factor to consider when determining the etiology of cervical adenitis. Acute bilateral disease is usually a response to acute pharyngitis, but can also occur with Epstein-Barr virus, cytomegalovirus, herpes simplex virus, roseola, and enteroviruses. Acute unilateral lymphadenitis commonly presents with an associated cellulitis and is typically caused by *S. aureus*, *Streptococcus pyogenes*, and group B streptococci. Subacute or chronic unilateral lymphadenitis is much less common. It can be due to cat-scratch disease, mycobacterial infection, or toxoplasmosis.

◆ Clinical

Signs

A thorough physical examination is essential. Note the size, location, laterality, firmness, and number of nodes. Erythema and tenderness are also important signs. Associated illnesses such as pharyngitis or systemic infection need to be noted.

Symptoms

Patients can present with unilateral or bilateral neck swelling, with or without any other symptoms. Tenderness of the affected nodes is commonly associated with acute infection.

Differential Diagnosis

Infectious causes are the most common. Malignancy is an important consideration in patients with no other signs of infection, recent weight loss or fevers, or those with isolated supraclavicular involvement. Noninfectious causes are much less common and include Kawasaki disease, sarcoidosis, sinus histiocytosis, histiocytic necrotizing lymphadenitis, and Kimura disease.

◆ Evaluation

Physical Exam

Cervical lymphadenitis typically presents with acute unilaterally or bilaterally enlarged (>3 cm) and tender lymph nodes in the jugulodigastric area. Atypical mycobacteria infection generally presents with an enlarged erythematous single mass, distinct from reactive adenopathy or fluctuant abscess.

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Imaging

Imaging is not necessary if cervical lymphadenitis is suspected based on history and physical exam findings. If there is concern for tuberculosis or an abscess, an ultrasound or CT of the neck can be performed. Cervical lymphadenitis will manifest as enlarged, enhancing nodes with low central attenuation if necrosis is present. Tuberculosis-infected lymph nodes will have thick peripheral enhancement on contrasted CT, low central attenuation, a relative lack of fat stranding, and calcification.

Labs

CBC is useful for determining whether the enlarged lymph nodes are secondary to systemic infection. Other laboratory studies, including Gram staining, acid-fast staining, and culture, can be done if aspiration is undertaken. Perform cat-scratch titer or monospot if infection is suspected.

Other Tests

Fine-needle aspiration biopsy (FNAB) can be both diagnostic and therapeutic. Obtain the aspirate from the largest, most fluctuant node using a 23- or 20-gauge needle. Gram stain, acid-fast stain, and culture should be obtained. The etiology is discovered in 60 to 90% of patients who undergo needle aspiration.

Excisional biopsy is indicated if the node is hard, fixed, fails to regress following aspiration or antibiotic use, enlarges, or is associated with fever or weight loss, or if the diagnosis is uncertain. Biopsy of more than one node is preferable. Placing a portion of the specimen in a flow cytometry medium is important if lymphoma evaluation is required.

Pathology

Pathology varies depending on the underlying etiology.

◆ Treatment Options

Medical

Patients who have asymptomatic, small (<3 cm), bilaterally enlarged cervical lymph nodes can be observed. If mass persists or enlarges over several weeks, biopsy is warranted. Those with signs and symptoms typical of acute bacterial lymphadenitis (large, tender, erythematous unilateral node with no systemic symptoms) can be treated empirically for *S. aureus* and *S. pyogenes* with amoxicillin–clavulanate, cephalexin, or clindamycin. If cellulitis is present or if the patient is having severe symptoms, parenteral nafcillin, cefazolin, or clindamycin is appropriate. When lymphadenitis is secondary to dental infection, anaerobic infection should be suspected and clindamycin or penicillin plus metronidazole is effective. Azithromycin, trimethoprim–sulfamethoxazole, or rifampin is effective early in the course of disease in preventing abscess formation if cat-scratch disease is suspected.

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Relevant Pharmacology

- Amoxicillin–clavulanate: Amoxicillin binds to penicillin-binding protein, thereby preventing bacterial cell wall synthesis. Clavulanate inhibits β-lactamases, which increases amoxicillin's spectrum of activity. Dosing is based on the amoxicillin component.
  - <3 months old: Total dose is 30 mg/kg/day. Divide dose and administer twice daily using the 125 mg/5 mL suspension.
  - Children <40 kg: 25–45 mg/kg/day divided every 12 hours using either 200 mg/5 mL or 400 mg/5 mL suspension. Alternatively, 200- or 400-mg chewable tablets can be used.
  - Children ≥40 kg and adults 875 mg twice daily
- Clindamycin: Inhibits bacterial protein synthesis by binding to bacterial 50S ribosomal subunits
  - Children <16 years
    - Oral: total dose of 8–25 mg/kg/day in 3–4 divided doses
    - Parenteral: 15–20 mg/kg/day
  - Adults
    - Oral: 300 mg three times daily
    - Parenteral: 1.2–1.8 g/day in two to four divided doses
- Trimethoprim-sulfamethoxazole: Both agents inhibit bacterial folic acid production. Trimethoprim inhibits dihydrofolic acid reduction and sulfamethoxazole interferes with dihydrofolic acid. Dosing is based on trimethoprim component.
  - Children >2 months
    - Total dose trimethoprim 8–12 mg/kg/day in divided doses every 12 hours or 20 mg/kg/day in divided doses every 6 hours for serious infections
  - Children >40 kg or adults
    - Oral: Trimethoprim 160 mg every 12 hours
    - Parenteral: Trimethoprim 2 mg/kg every 6 hours

Surgical

Incision and drainage is useful if there is an abscess, especially due to *S. aureus* or *S. pyogenes*. If mycobacterial or a *Bartonella henselae* infection is suspected, then FNAB is preferable to avoid fistula formation. Most cases of cat-scratch fever are managed medically. Incision and drainage is reserved for cases progressing to abscess formation. Surgical excision or curettage is effective if nontuberculous mycobacterial infection is the cause. Removal of the largest node and necrotic nodes is sufficient because the remaining adenopathy will resolve spontaneously.

◆ Complications

Infection control is essential if a postoperative complication occurs. Empiric use of broad-spectrum antibiotics is appropriate. Further care depends on the

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type of complication. Internal jugular venous thrombosis can be managed with anticoagulation. Abscess is treated with incision and drainage.

◆ Outcome and Follow-Up

The patient can return home after incision and drainage or surgical excision, but close monitoring is necessary because of the potential complications. Mediastinal abscesses, purulent pericarditis, thrombosis of the internal jugular vein, pulmonary emboli, or mycotic emboli are all rare, but serious complications.

Cervical lymphadenitis resolves completely in the majority of patients who receive appropriate antibiotic therapy. No further follow-up is necessary unless the patient's symptoms do not improve or worsen.

◆ ICD-9 Codes

683 Acute lymphadenitis
784.2 Neck mass

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6.13 Hemangiomas, Vascular Malformations, and Lymphatic Malformations of the Head and Neck

◆ Key Features

- Hemangiomas, vascular malformations, and lymphatic malformations of the head and neck are common congenital and neonatal abnormalities.
- Vascular tumors are classified into hemangiomas and vascular malformations.
- Accurate diagnosis is key to prognosis and treatment plan.
- Accurate diagnosis is based on natural history and key clinical features.

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Vascular tumors are common pediatric anomalies, and may develop throughout the head and neck. They are divided broadly into hemangiomas and vascular malformations, each with a distinct natural history. Lymphatic malformations (aka, lymphangiomas) are defects in the lymphatic system that present as swellings or nodules in the skin or mucous membranes of the head and neck. The malformation of lymphatic tissue causes an accumulation of fluid, which accounts for the clinical presentation.

◆ Epidemiology

Infantile hemangiomas occur in up to 12% of all children. They are more common in females than males, at a ratio of 3:1, and more common in Caucasians. Sixty percent of hemangiomas are located in the head and neck, including the upper aerodigestive tract, and 80% occur as single lesions.

Vascular malformations are divided into lymphatic malformations, capillary malformations or port-wine stains, venous malformations, and high-flow arteriovenous malformations (AVMs). Lymphatic malformations are further divided into microcystic lymphatic malformations and macrocystic lymphatic malformations involving the soft tissue of the neck. Vascular malformations have no gender or racial predilection, and most commonly occur in the head and neck. Lymphatic malformations represent 6% of benign cervicofacial tumors of childhood; they are seen in less than 2.8 per 1000 people. Patients with Down, Turner, or fetal alcohol syndrome have a higher incidence of lymphatic malformations.

◆ Clinical

Signs and Symptoms

Hemangiomas

Infantile hemangiomas are generally not present at birth and first appear during the first 6 weeks of life. Hemangiomas go through predictable phases of growth. The initial proliferative phase occurs over the first year, is characterized by rapid growth, and is followed by the involution phase with subsequent regression. Complete involution occurs in 50% of children by 5 years of age, 70% of children by 7 years of age, and 90% of children by 9 years of age.

Vascular Malformations

Vascular malformations are by definition present at birth but may go unrecognized. They grow proportionately with the child, and may present throughout childhood or early adulthood. Hemangiomas are firm; vascular malformations are easily compressible. They may involve the facial skeleton and abruptly increase in size with acute hemorrhage or infection, puberty, or pregnancy.

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Lymphatic Malformations

Lymphangioma circumscriptum presents as a nodular mass with a red, wart-like appearance. They are typically asymptomatic but bleeding or drainage of fluid may occur.

Cavernous lymphangioma (cystic hygroma) presents as a painless subcutaneous swelling or nodule, and can rapidly enlarge with infections.

Differential Diagnosis

The key to diagnosis is to differentiate between hemangiomas and vascular malformations. Diagnosis can generally be made on the clinical history and physical exam alone. Lymphatic malformations presenting as cystic lesions within the neck must be differentiated from other congenital cystic lesions including branchial cleft cysts (discussed in Chapter 6.14).

◆ Evaluation

History

In the patient history, the timing of the development of the lesion is important, particularly whether it was present at birth. Also note the growth rate of the lesion.

Physical Exam

A complete head and neck exam should focus on the character of the lesion. Children with hemangiomas in the chin and chest are more likely to have concurrent airway lesions, including subglottic hemangiomas. Noisy breathing in these children must be evaluated with laryngoscopy and likely bronchoscopy.

Imaging

Imaging is generally unnecessary for diagnostic purposes but may be warranted in children with multiple cutaneous hemangiomas, who are more likely to have concurrent visceral lesions. MRI may be useful, particularly in the preoperative setting, to determine the extent of soft tissue involvement. Imaging may also be helpful in the evaluation of a child with cutaneous hemangioma and noisy breathing. Flow voids are indicative of high flow lesions.

Pathology

Pathologic exam also helps to differentiate between hemangioma and vascular malformation. Hemangiomas are characterized by proliferating endothelial cells and are positive for GLUT-1, helping to differentiate them from other vascular tumors. Hemangiomas grow by cellular hyperplasia; vascular malformations grow by hypertrophy.

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◆ Treatment Options

Hemangiomas

Expectant management is recommended for most hemangiomas. Intervention is indicated for lesions threatening the airway or vision, and may be considered for bleeding and infection or progressive disfigurement. Tracheotomy is sometimes required to manage the airway of a child with a subglottic hemangioma.

Both systemic and intralesional steroids are commonly used during the proliferative phase with high success rates. Complications of systemic steroids, such as pathologic fractures, may lead to the discontinuation of this medical therapy. Interferon has been used with success, but is associated with neurologic complications. The use of various lasers may be considered. Surgery may be appropriate, but must be considered carefully.

Vascular Malformations

Vascular malformations are not expected to involute and so the decision to treat is less controversial. Tracheotomy may be required for large cystic hygromas. Sclerotherapy with OK-432 (picibanil) has shown success, though OK-432 remains an experimental treatment modality. There are other older sclerotherapy agents used widely with success, such as ethanol.

Argon lasers and dye lasers are used in the treatment of capillary malformations, or port-wine stains (see Chapter 1.8.6). Complete surgical resection, with the consideration of preoperative embolization, is required in the treatment of AVMs.

Lymphatic Malformations

Surgical management of lymphatic malformations may be considered, although the lesion may involve vital structures and complete resection may not be appropriate.

◆ Complications

Complications of enlarging vascular tumors and surgical intervention depend on the size and location of the lesion. Functional and cosmetic factors must be considered in both the decision to treat and the extent of treatment.

◆ Outcome and Follow-Up

Hemangiomas initially managed expectantly must be monitored closely, as even lesions not involving the airway or orbit may cause significant stress to parents during the proliferative phase. Untreated vascular malformations must be similarly monitored as they grow proportionately with the child. Large lymphatic malformations can require multiple surgeries, require tracheotomy, and cause significant morbidity.

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◆ ICD-9 Codes

228 Hemangioma and lymphangioma, any site
747.6 Other anomalies of peripheral vascular system

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6.14 Branchial Cleft Cysts

◆ Key Features

- Branchial cleft cysts represent the most common noninflammatory lateral neck masses in children.
- Definitive treatment is complete surgical excision.

Branchial cleft cysts are composed of remnants from any of the first five branchial arches, which give rise to head and neck structures (**Table 6.10**). Branchial cleft cysts may be associated with a fistula or sinus tract. Cysts may have different locations and characteristics depending on their branchial cleft of origin.

◆ Epidemiology

Branchial cleft cysts are common; they are responsible for 33% of congenital neck masses and 17% of pediatric neck masses. Most branchial cleft cysts are derived from the second branchial arch apparatus; 1% of cysts are derived from the first branchial arch. Third arch cysts are reported; fourth branchial cleft cysts are extremely rare.

◆ Classification

First branchial arch abnormalities can be classified as type 1 or 2.

- Type 1 abnormalities are of ectodermal origin and are lined by epidermoid elements. They lie parallel to the external auditory canal in the

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Table 6.10 Structures that Arise from Differentiation of Branchial Arches

Arch	Nerve	Structure	Muscle	Artery
First	Trigeminal	Mandible, body of incus, head and neck of malleus, major salivary glands, tympanic membrane, eustachian tube	Masticator muscles, tensor tympani, anterior belly of digastric	Facial
Second	Facial (CN VII), cochleovestibular (CN VIII)	Two thirds of long process of incus, manubrium of malleus, crura and head of stapes, styloid, lesser cornu of hyoid, upper body of hyoid, tonsil	Platysma, muscles of facial expression, stapedius, posterior belly of digastric	Stapedial
Third	Glossopharyngeal (CN IX)	Greater cornu of hyoid, thymus, inferior parathyroid, body of hyoid	Superior constrictors	Internal carotid
Fourth	Vagus (CN X)	Thyroid cartilage, epiglottis, superior parathyroid	Inferior constrictor, laryngeal musculature	Aortic arch, right subclavian
Fifth	Spinal accessory (CN XI)	Arytenoid cartilage, cricoid cartilage, lungs	Portion of laryngeal musculature	Pulmonary ductus

Source: Van de Water TR, Staecker H. Otolaryngology: Basic Science and Clinical Review. Stuttgart/New York: Thieme; 2006:208.
Abbreviation: CN, cranial nerve.

preauricular area. They may be associated with a fistula or sinus tract that may end in the middle ear or external auditory canal.

- Type 2 abnormalities are more common than type 1. They are of ectodermal and mesodermal origin and consist of squamous epithelium and adnexal structures. An external opening may be present, leading to a cyst or sinus tract that courses through the parotid gland. Patients often present with otorrhea unresponsive to typical treatment. *Note*: This tract may be closely associated with the facial nerve at the stylomastoid foramen.

Second branchial arch remnants, the most common branchial anomaly, present as a painless mass or dimple below the angle of the mandible, at the anterior border of the sternocleidomastoid muscle. They may present as a cyst, or may have a tract ending at the tonsillar fossa. The tract, if present, traverses thru the carotid bifurcation (**Fig. 6.4**).

Third and (especially) fourth and fifth branchial cleft remnants are much more rare; they may have a long tract looping deep to the carotid artery and ending in the region of the piriform.

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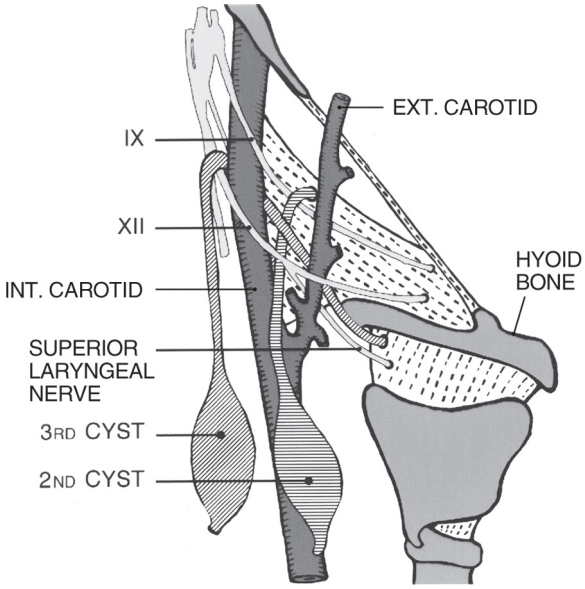

Fig. 6.4 Relationship of second and third branchial cleft anomalies to the carotid artery and cranial nerves. (From Van de Water TR, Staecker H. Otolaryngology: Basic Science and Clinical Review. Stuttgart/New York: Thieme; 2006:209.)

◆ Clinical

Signs and Symptoms

Branchial cleft cysts often present as lateral painless masses and may enlarge after an upper respiratory infection. If there is an external sinus or fistula opening, there are troublesome mucus secretions; recurrent infections are common.

Larger branchial cysts may compress the airway causing stridor, dyspnea, or dysphagia.

Differential Diagnosis

- Branchial cleft cyst or sinus
- Lymphatic malformation
- Hemangioma
- Ectopic thymus
- Lipoma
- Thymic cyst
- Parotid cyst

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- Pseudotumor of infancy
- Malignant tumor
- Laryngocele

◆ Evaluation

History

The timing of the development of the lesion is important, particularly whether it was present at birth. Also, note the growth rate, size fluctuation, and history of infection or drainage.

Physical Exam

During the head and neck exam, note the size, the precise location of the mass, the presence or absence of a dimple or opening, and quality (firm, soft, etc.). Assess for possible other congenital anomalies or syndromic features.

Imaging

Branchial abnormalities are visualized using CT with contrast; if a sinus or fistula is present, contrast fistulography and barium swallow esophagraphy may demonstrate the path of the abnormality.

Pathology

Branchial cleft cysts arise from residual embryonic tissue from the branchial clefts or pouches (**Table 6.10**).

◆ Treatment Options

Medical

If the branchial cyst is infected, treatment with IV antibiotics is indicated before surgical excision.

Surgical

The treatment of choice for branchial cleft cysts is surgical excision. Excision of a first branchial cleft cyst may include the cartilage of the external auditory canal. For first arch type 2, total parotidectomy with facial nerve dissection may also be needed.

Second, third, fourth, and fifth branchial cleft cysts are excised through horizontal neck incisions. “Step ladder” incisions may be needed to excise a tract. The relationship of the tract to carotid and cranial nerves is predictable and must be understood. If a lesion is a true third branchial cyst, it may present as a thyroid cyst, in which case hemithyroidectomy is performed.

Other treatments such as radiation or sclerosing agents do not provide a cure for branchial cysts and increase the risk of recurrence.

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◆ Complications

Bleeding or hematoma can be prevented using electrocautery on small blood vessels and silk ties on larger vessels. Recurrence of branchial cleft cysts can occur if there is incomplete excision of the mass. Injury to the facial nerve may occur during the excision of a first branchial cleft cyst. Damage to associated neurovascular structures may occur during the excision of a second or third branchial abnormality.

◆ Outcome and Follow-Up

If facial nerve dissection was performed, assess function in the early postoperative period. A Penrose or suction drain is recommended for any extensive dissection. Antibiotics are necessary if the aerodigestive tract was entered or if the cyst was infected. Postoperative care is otherwise routine.

◆ ICD-9 Code

744.42 Branchial cleft cyst

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6.15 Congenital Midline Neck Masses

◆ Key Features

- Thyroglossal duct cysts, dermoid cysts, and lipomas are common midline congenital masses.
- Thyroid cancer can develop from a thyroglossal duct cyst, rarely.
- Treatment requires complete surgical excision.

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Thyroglossal duct cysts are midline masses that may appear anywhere between the base of the tongue and the thyroid gland. Dermoid cysts typically present in the submental area of the neck but can appear in other areas of the head and neck. Lipomas are derived from adipose tissue and present as soft painless masses, which may occur anywhere in the body.

◆ Epidemiology

Thyroglossal duct cysts are the most common congenital neck mass, present in 7% of the population. Lipomas are the most common soft tissue tumor. They may occur at any age, but are common after age 40.

◆ Clinical

Signs and Symptoms

Thyroglossal duct cysts are firm, mobile typically midline masses and can appear anywhere along the path of the thyroid's descent. They can be off-midline. They will elevate upon swallowing or protrusion of the tongue and are usually asymptomatic. However, large cysts, or those in the base of the tongue, can compress the airway causing dysphagia and respiratory difficulties. Cysts can become infected and may also enlarge after an upper respiratory infection.

Dermoid cysts are attached to and move with the skin. They are usually located in the submental area. They do not move upon swallowing or protrusion of the tongue. They are typically asymptomatic unless infected.

Lipoma presents as a soft, movable lump under the skin. They occur in the neck but also in multiple tissues and locations throughout the body.

Differential Diagnosis

- Thyroglossal duct cyst
- Dermoid cyst
- Lipoma
- Enlarged thyroid isthmus
- Pyramidal lobe of thyroid
- Plunging ranula
- Thyroid nodule/cyst
- Sebaceous cyst
- Teratoma
- Cervical thymic cyst

◆ Evaluation

Physical Exam

During the full head and neck exam, note the location, the quality of the mass, its size, and its motion with skin or swallowing. Assess for possible other congenital anomalies.

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Imaging

Ultrasound of the thyroid is mandatory to document thyroid tissue in its normal location, as a thyroglossal duct cyst may rarely contain all functioning thyroid tissue. Ultrasound may be sufficient to diagnose a thyroglossal duct cyst, and differentiate it from ectopic thyroid. If the diagnosis is unclear, CT or MRI will usually differentiate thyroglossal duct cyst, dermoid, and lipoma.

Pathology

Thyroglossal duct cysts arise from remnants of the thyroid gland as it descends from the foramen cecum during embryologic development (**Fig. 6.5**). Dermoid cysts are epithelial-lined cavities containing adnexal structures. They result from epithelium becoming enclosed in tissue during embryological development. Lipomas are benign, subcutaneous masses derived from adipose tissue.

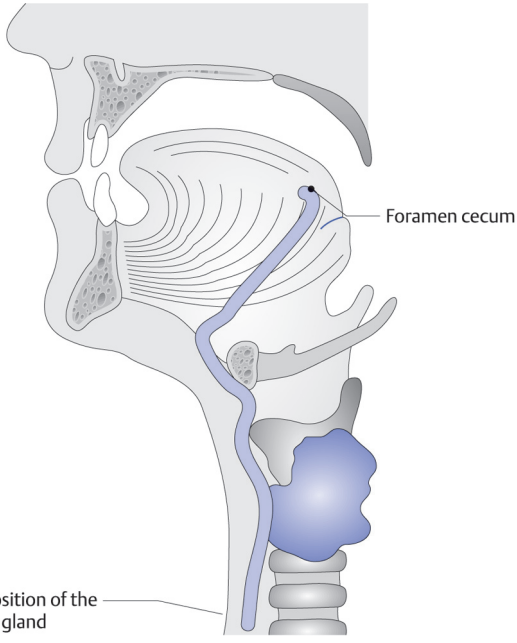

Fig. 6.5 Drawing of the lateral neck showing the path of descent of the thyroid anlage. Thyroid duct cysts, fistulae, and ectopic thyroid tissue can occur anywhere along the course of this duct. (From Mafee MF, Valvassori GE, Becker M. Imaging of the Head and Neck. 2nd ed. Stuttgart/New York: Thieme; 2005:837.)

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◆ Treatment Options

Medical

If the cyst is infected, treatment with IV antibiotics is indicated before surgical excision.

Surgical

Thyroglossal duct cysts are excised through a transverse incision at the level of the hyoid bone. The center of the hyoid bone is removed to include a tract entering the base of the tongue (Sistrunk procedure), to minimize recurrence. Avoiding lateral dissection will minimize the possibility of injury to the hypoglossal nerves.

Dermoid cysts and lipomas are excised through a simple transverse neck incision positioned in accordance with the location of the mass.

◆ Complications

Bleeding or hematoma can be prevented using electrocautery on small blood vessels and silk ties on larger vessels. Damage to superior laryngeal nerves or the hypoglossal nerve is also possible. Incomplete excision of the thyroglossal duct increases the risk of recurrence.

◆ Outcome and Follow-Up

The incision should be drained with a Penrose or suction drain. Specimen should be sent to pathology to rule out neoplasm. Prophylactic antibiotics should be given if the excised cyst was infected.

◆ ICD-9 Code

759.2 Thyroglossal duct cyst

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6.16 Congenital Midline Nasal Masses

◆ Key Features

- The most common congenital midline nasal masses are gliomas, encephaloceles, and dermoids.
- The most common of these is the dermoid, derived from ectodermal embryonic tissue.
- A glioma consists of CNS tissue at the nasal dorsum; it is congenital and not a true neoplasm.
- Meningoceles or encephaloceles do communicate with the ventricles and contain cerebrospinal fluid (CSF).
- Teratomas are true neoplasms arising from totipotent cells; half of all head and neck teratomas occur in the nose.

Gliomas, meningoceles, encephaloceles, dermoids, and teratomas exist in the differential diagnosis of the congenital midline nasal mass. A dermoid cyst is a congenital benign neoplasm containing keratinizing squamous epithelium and adnexal skin structures, may be located from the columella to the nasion, and often presents as a dimple on the nasal bridge. The dermoid may involve only skin and nasal bone, or it may have a true dural connection. Nasal gliomas lack a direct connection to the CNS and contain heterotopic glial elements in a fibrillar stroma, of varying growth rate. Encephaloceles are continuous with the CNS. Sincipital encephaloceles present in the nasal dorsum or forehead region, basal encephaloceles in the nasopharynx. Encephaloceles may include meningoceles (containing only meninges), encephalomeningoceles (containing brain also), and encephalomeningocystoceles (including part of the ventricular system). Midline nasal masses should never be biopsied in the outpatient setting or without prior imaging studies.

◆ Epidemiology

Congenital midline nasal masses occur in 1 in 20 to 40,000 live births. Ten percent of dermoids occur in the head or neck, and 10% of these occur in the nose. Encephaloceles occur in 1 in 1250 to 2000 live births; 40% have other abnormalities. Occipital encephaloceles are most common in North America (75%) although sincipital are most common in Southeast Asia.

◆ Clinical

Signs

Dermoids are typically anywhere along the midline of the nose and are firm, noncompressible, and nonpulsatile. A nasal dermoid sinus or cyst presents with a dimple containing a hair follicle. Rarely, the nasal base is

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widened. Encephaloceles are soft, bluish, compressible, pulsatile, and may transilluminate. Gliomas are smooth, firm, noncompressible, and nontransilluminating. All may be associated with a cranial defect, although this is most common with an encephalocele.

Symptoms

Masses may be intranasal or extranasal.

Differential Diagnosis

- Nasal obstruction
- Hypertelorism
- Epiphora
- Infection (local or meningitis)

◆ Evaluation

See **Table 6.11**.

Physical Exam

- Internal and external nasal exam
- There may be a positive Furstenberg test in encephaloceles (mass expands with jugular vein compression).
- Exam for other possible congenital anomalies

Imaging

CT with contrast; if there is any hint that the mass may be continuous with the CNS, an MRI is also required. Ossification of the anterior cranial base is variable in children under 5 years.

Table 6.11 Clinical Approach to Congenital Midline Nasal Mass

Evaluation
Head and neck exam
Location, size, quality of mass
Evidence of other congenital anomalies
Furstenberg test
Possible meningocele or encephalocele (enlargement of mass with jugular compression)
Imaging
CT and/or MRI
Presumptive diagnosis
Treatment planning; neurosurgical consult if evidence of CSF connection

Abbreviations: CSF, cerebrospinal fluid; CT, computed tomography; MRI, magnetic resonance imaging.

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Pathology

These masses may result from a failure of complete involution of a dural diverticulum that protrudes through the fonticulus nasofrontalis, and typically forms the foramen cecum.

- Dermoid: A cyst lined with squamous epithelium, along with hair and sebaceous glands
- Glioma: Astrocytes, neuroglial fibers, and S-100–positive
- Encephalocele: A nonneoplastic mature neuroglial tissue with meninges

◆ Treatment Options

Small superficial dermoids may be removed via a nasal dorsum incision. A sinus should be removed with an elliptical incision. Other dermoids may require an external rhinoplasty approach, and sometimes a bicoronal flap if there is extension to the cranial base. For large dermoids, calvarial bone may be needed for reconstruction of cranial base defects.

External gliomas also require an elliptical skin incision or external rhinoplasty approach. Internal gliomas are usually lateral and can be removed through a lateral rhinotomy incision. In either case, if a CSF leak is encountered, a bifrontal craniotomy or an endoscopic CSF leak repair is necessary. This may necessitate collaboration with neurosurgical colleagues for repair.

Encephaloceles require neurosurgical collaboration, and likely a bifrontal craniotomy to maximize visualization. Repair of defects requires temporalis or pericranial flaps for a water-tight closure. Extracranial repair is similar to that for gliomas.

◆ Complications

A CSF leak may require lumbar drainage and another patch procedure. Local wound infection or meningitis will require antibiotic therapy:

- Antibiotic coverage for meningitis (see also Chapter 2.2.3):
  - Cefotaxime IV every 4 hours or ceftriaxone IV every 12 hours plus ampicillin IV every 6 hours
- Alternative if there is drug resistance:
  - Vancomycin (child 15 mg/kg IV every 6 hours) plus cefotaxime or ceftriaxone plus ampicillin

Incomplete dermoid or glioma excision will lead to recurrence or fistula formation and require a wide local excision with reconstruction.

◆ Outcome and Follow-Up

Typically, patients are observed in a neurologic ICU setting, except for minor external excisions. Patients should be monitored for complete healing and infection.

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◆ ICD-9 Codes

748 Congenital deformity of nose
742 Encephalocele, meningocele

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6.17 Choanal Atresia

◆ Key Points

- Choanal atresia occurs in ~1 in 5000 to 8000 births.
- Bilateral atresia is more common and is associated with other anomalies (e.g., CHARGE association).
- Endoscopic techniques have greatly improved the safety of the transnasal approach.

Choanal atresia is a congenital condition in which one or both of the choanae are replaced with a bony or mixed bony and membranous wall. Unilateral atresia usually presents later in life, while bilateral atresia is detected at birth. Bilateral atresia can often be medically managed with the use of a McGovern nipple while allowing growth in preparation for repair. The most common repair options include the transnasal and transpalatal approaches. The most frequent complication of repair is restenosis.

◆ Epidemiology

Choanal atresia is relatively rare, only occurring in 1 in 5000 to 8000 births. It is almost twice as common in females as in males; 65 to 75% of cases are unilateral. The right choana is more commonly affected. Nearly 75% of bilateral atresias are associated with other disorders including Treacher-Collins syndrome, DiGeorge sequence, Apert syndrome, trisomy 18, and CHARGE association.

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◆ Clinical

Signs

Bilateral atresia will most often present in newborns with cyanotic spells relieved by crying and worsened with feeding. Unilateral atresia may present later in childhood with unilateral thick nasal secretions similar to sinusitis.

Symptoms

Bilateral disease will present at birth with difficulty breathing. Unilateral disease may present in older children and may be associated with history of nasal congestion.

◆ Differential Diagnosis

Bilateral

- Congenital nasal piriform aperture stenosis
- NOWCA (nasal obstruction without choanal obstruction)
- Midnasal stenosis
- Choanal stenosis

Unilateral

- Sinusitis
- Nasal foreign body
- Nasal septal deviation
- Choanal stenosis

◆ Evaluation

Physical Exam

Examination of the nares will reveal thick mucous secretions. Commonly, the diagnosis is made after failure to pass a soft suction catheter into the nasopharynx. A flexible endoscope can also be used to assess choanal patency.

Imaging

CT provides the most reliable method of diagnosis. Axial images show narrowing of the choanal orifice or widening of the posterior vomer. Approximately 30% of atresias will be purely bony in nature and 70% will be mixed bony and membranous.

Pathology

There are multiple theories, one of which is that the atresia is due to the persistence of the buccopharyngeal membrane.

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◆ Treatment Options

Medical

Initially, an oral airway or a McGovern nipple may be used to maintain an airway. Stimulating the infant to cry will also provide temporary relief. Endotracheal intubation is often appropriate.

Surgical

- *Transnasal approach:* Puncture of the atretic plate using a curved trocar or urethral sound, followed by drilling of the vomer, hard palate, and pterygoid plate. This is usually done with the use of a 120-degree endoscope in the nasopharynx to provide visualization. Blind use of a trocar is to be avoided. Stents may be placed at the completion of the procedure. Advantages include its ability to be performed in infants and its relatively short operative time. Disadvantages include difficulty in preserving mucosal flaps with the use of the drill. Some surgeons advocate the use of laser (CO_2 or Nd-YAG) with the transnasal approach.
- *Transpalatal approach:* Creation of a palatal flap followed by removal of palatine bone, atretic plate, and vomer. Stents may be placed. Advantages include better visualization, the ability to create lining flaps, and reduced duration of stenting. Disadvantages include oronasal fistula and palatal growth disturbance, with risk of crossbite or midface retrusion.
- *Transantral approach:* Primarily used in revision cases. The choana is opened widely to include the maxillary sinus.
- *Transeptal approach:* Primarily used in unilateral atresia. Removal of posterior septum creates a passage to bypass the atresia.

◆ Outcome and Follow-Up

Postoperative care should include daily suctioning of stents, and serial endoscopy and dilation. The rate of restenosis varies widely with reports between 50 and 100%. Both transnasal and transpalatal approaches seem to have similar rates of restenosis.

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6.18 Cleft Lip and Palate

◆ Key Features

- Group 1: Cleft lip
  - Unilateral or bilateral
  - Complete (extension to nasal floor)
  - Incomplete (muscle diastases: vermillion to bridge of tissue at nasal sill)
- Group II: Cleft palate
  - Unilateral or bilateral
  - Secondary palate only
- Group III: Cleft lip and cleft palate
  - Complete cleft palate (both primary and secondary palate)
- Group IV: Cleft palate
  - Unilateral or bilateral
  - Primary palate only

Cleft lip and cleft palate can be classified as syndromic (15–60%) or nonsyndromic. Syndromic associations include Apert, Stickler, Treacher-Collins, and Waardenburg syndromes. Cleft lip with or without cleft palate is believed to be genetically distinct from isolated cleft palate. Nonsyndromic clefts may be secondary to exposure to teratogens (e.g., ethanol, anticonvulsants, steroids, vitamin A excess). Likewise maternal and intrauterine factors such as gestational diabetes, smoking, or amniotic bands may play a role.

Typically cleft palate management is done by a team including surgeons, dentists, orthodontists, speech pathologists, and audiologists.

◆ Embryology of the Lip and Palate

The palate is embryologically divided into the primary (the maxillary alveolus and palate anterior to the incisive foramen), and secondary (originating posterior to the incisive foramen and terminating at the uvularis) components. Fusion of the paired median nasal prominences (MNPs) gives rise to the primary palate. This process initiates the separation of the oral from the nasal cavity. The coalescence of the MNPs forms the central maxillary alveolar arch, central and lateral incisors, anterior hard palate, premaxilla, philtrum of the upper lip, columella, and nasal tip. The genesis of the central upper lip occurs in association with the primary palate. The frequent association of these deformities is not unexpected.

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The remainder of the upper lip, lateral to the philtrum, is formed by fusion of the paired MNPs medially with the maxillary processes laterally.

Formation of the secondary palate is initiated by contact of the nasal septum to the lateral palatal shelves at the incisive foramen. The medialization of the palatal shelves then occurs in an anterior to posterior direction. The midline tongue represents a barrier to this medialization. Development of the mandible results in anterior displacement of the tongue allowing for normal growth of the palatal shelves. Failure of this process results in the Pierre Robin sequence (micrognathia, relative macroglossia, and U-shaped cleft palate); see also Chapter 6.8.

There is not only an embryologic structural division of the palate, but a temporal one as well. The primary palate forms during weeks 4 to 7 of gestation. The secondary palate begins development after completion of the primary palate and occurs during weeks 8 to 12 of gestation.

◆ Epidemiology

Cleft lip and palate is the most common congenital malformation of the head and neck. The incidence of cleft lip with or without cleft palate in the United States is 1:1000 newborns and varies according to race with the highest incidence in Native Americans and a male/female ratio of 2:1. The incidence of cleft palate is 1:2000 and is equal across ethnic groups, with a male/female ratio of 1:2.

◆ Clinical

Signs

See the classification of defects noted under Key Features, above. Signs of submucous cleft palate include:

- Bifid uvula
- Zona pellucida
- Notched hard palate

Symptoms

The symptoms are feeding difficulties with nasal regurgitation.

Differential Diagnosis

Associated syndromes:

- *Pierre Robin sequence:* Micrognathia, glossoptosis, and U-shaped cleft palate
- *Stickler syndrome:* Retinal detachment, cataracts, and early arthritis
- *Treacher-Collins syndrome:* Eyelid colobomas, middle ear ossicular abnormalities, and malformation of facial bones
- *Apert syndrome:* Acrocephaly, fused digits, and stapes fixation

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◆ Evaluation

Physical Exam

- Determine type of defect
  - Unilateral, bilateral, median
  - Complete (extension to nasal floor) or incomplete (submucosal)
  - Primary (anterior to incisive foramen) or secondary (posterior to incisive foramen)
- Look for associated defects
  - Facial defects: Telecanthus, maxillary/malar hypoplasia, nasal deformities, facial nerve paralysis
  - Otologic anomalies should be examined.
  - Synostoses
- Determine presence of associated syndrome. Apert, Stickler, Treacher-Collins, Waardenburg syndromes, and Pierre Robin sequence are associated with cleft palate.
- Cleft lip nasal deformity

Dehiscence of the orbicularis oris muscle results in its abnormal, nonanatomic insertion laterally onto the ala and medially onto the columella. The subsequent muscular tension produces a characteristic nasal deformity. The nasal tip and columella are deflected to the noncleft side. The cleft side ala is positioned laterally, inferiorly, and posteriorly. The cleft nostril is widened and horizontally oriented. The septum is bidirectionally affected. Whereas the caudal aspect deflects toward the noncleft side, the remainder of the cartilaginous and bony septum deflects toward the cleft side. This results in decreased air entry through both nasal passages.

The bilateral cleft nasal deformity is dependent on the severity of the individual sides. If both sides are equally involved the nasal tip is typically midline, poorly defined, and frequently bifid. Both sides are composed of obtuse domal angles and widened, horizontally oriented nostrils. The alae are both positioned laterally, inferiorly and posteriorly. The septum is midline. If there is asymmetry of the cleft lip, deformities of the nasal tip, columella, and septum are deviated to the less affected side; however, the deflection is less apparent than it would be with a normal side.

Imaging

Adjunctive imaging may be required for the investigation of syndromic etiology.

Other Tests

Close audiologic monitoring is warranted given the increased incidence of acute OM and serous OM due to eustachian tube dysfunction in this population.

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◆ Treatment Options

Medical

- Parental counseling
- Ensure adequate feeding and nutrition
  - Cleft lip and alveolus – often feed normally by bottle or breast
  - Complete cleft lip or palate – often have feeding problems initially
    - Inability to generate sufficient seal around the nipple
    - Increased work of feeding and swallowing of air
    - Specialty nipple may be required
      - Preemie nipple
      - Haberman feeder
      - Mead-Johnson cross-cut nipple
      - Squeeze bottle nurser
    - Palatal prosthesis for wide clefts for continued feeding difficulties

Surgical

- Lip adhesion
  - Converts a complete cleft lip into an incomplete cleft lip
  - Performed at 2 to 4 weeks of age

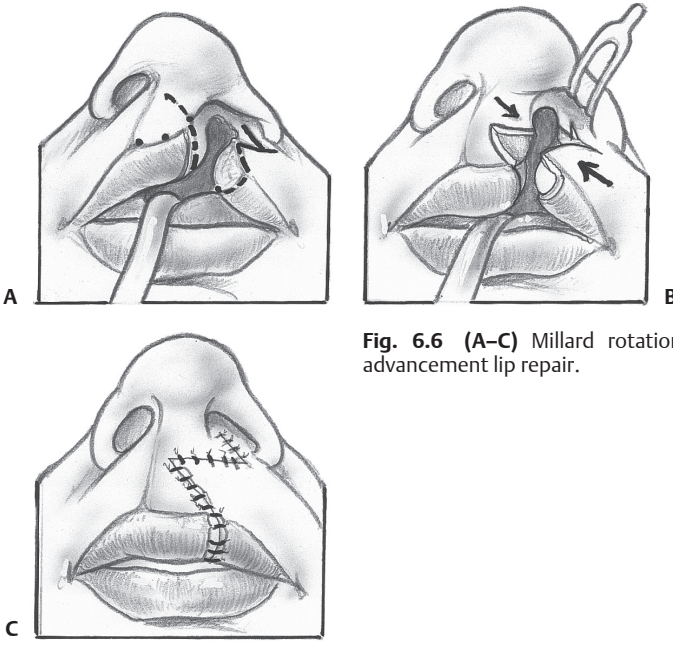

Fig. 6.6 (A–C) Millard rotation advancement lip repair.

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- Allows definitive lip repair to be performed under less tension at 4 to 6 months of age
- Criteria:
  - Wide unilateral complete cleft lip and palate where conventional lip repair would produce excessive incisional tension
  - Symmetric wide bilateral complete cleft lip with prominent premaxilla
  - Converts an asymmetric bilateral cleft lip to a symmetric cleft lip
- Disadvantage: Scar tissue that may later interfere with definitive surgical management of the lip

- Cleft lip repair
  - "Rule of tens" for timing of cleft lip repair
    - Age ≥10 weeks
    - Weight ≥10 lb
    - Hemoglobin ≥10 g
  - Surgical approaches:
    - Millard rotation advancement technique (**Fig. 6.6**)
    - Tennison-Randall: Single triangular flap interdigitation (**Fig. 6.7**)
    - Bardach: Double triangular flap interdigitation
    - Straight line closure (uncommon)

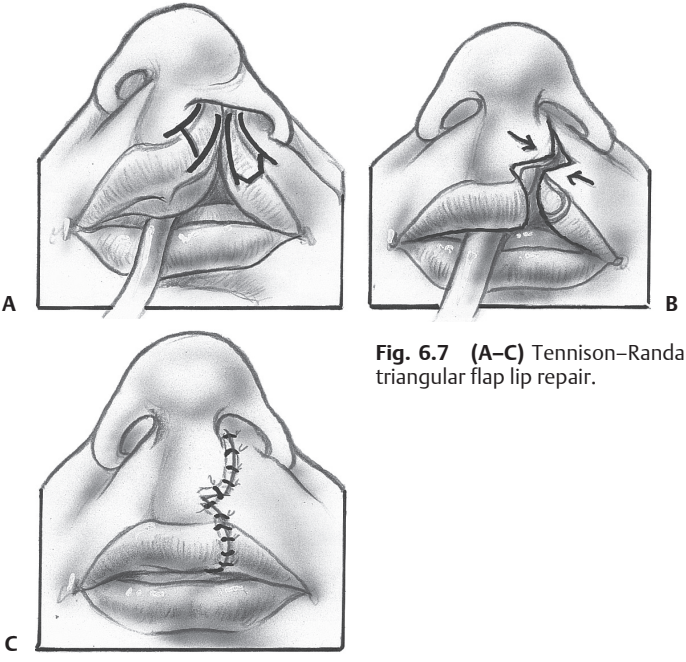

Fig. 6.7 (A–C) Tennison–Randall triangular flap lip repair.

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- Cleft palate repair
  - The exact timing of surgical closure of cleft palate is controversial.
  - There is a balance between establishing the velopharyngeal competence necessary for speech and the potential negative influence of early repair on maxillofacial growth and occlusion.
  - It is usually performed between 9 and 16 months of age.
  - Surgical approaches:
    - Schweckendiek: Closure of soft palate only
    - Von Langenbeck: Two bipedicle mucoperiosteal flaps are created by incising along the medial cleft edges and along the posterior alveolar ridge from the maxillary tuberosities to the anterior level of the cleft. They are mobilized medially and closed in layers (**Fig. 6.8**).
    - Bardach: Two flap palatoplasty for complete cleft palate repair
    - Furlow palatoplasty: Double Z-plasty for secondary cleft palate repair (**Fig. 6.9**)
    - V-Y pushback technique: For secondary cleft palate repair
  - Tympanostomy tubes are commonly inserted at the time of palate repair.

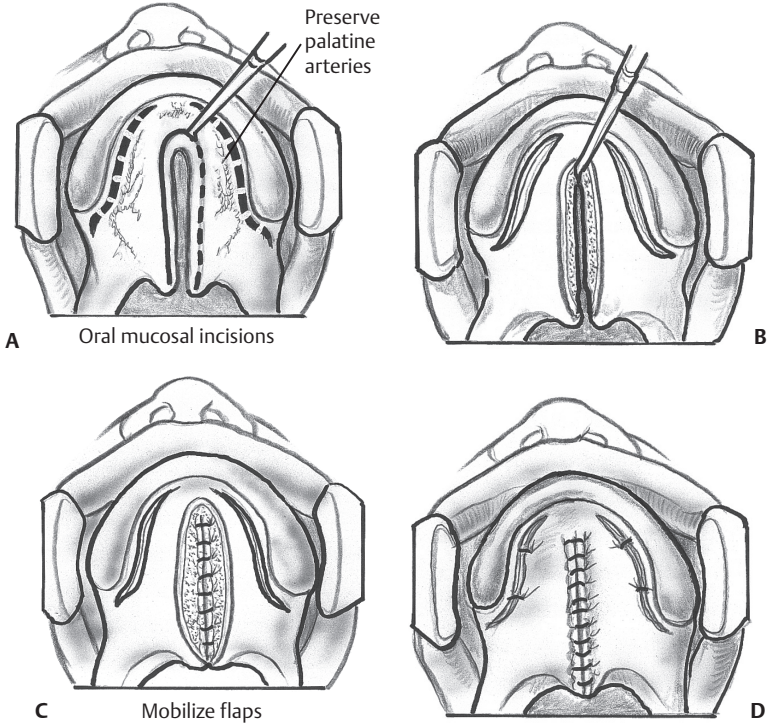

Fig. 6.8 **(A–D)** Von Langenbeck repair.

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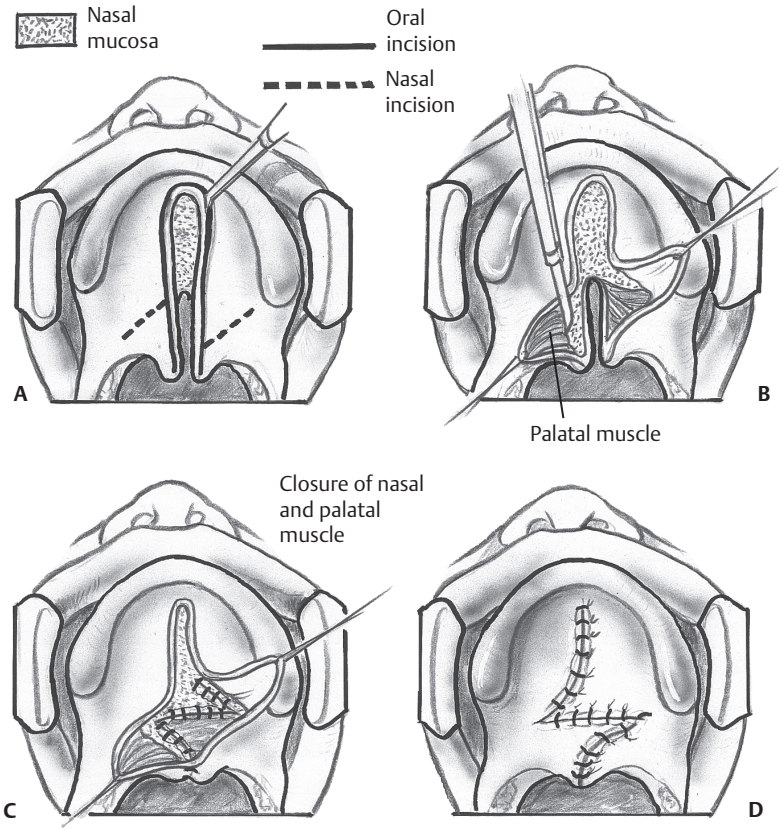

Fig. 6.9 **(A–D)** Furlow palate lengthening procedure ("double opposing Z-plasty").

◆ Complications

Primary or secondary hemorrhage is uncommon but may require a return to the OR for management. Oronasal fistula is managed with secondary repair. Velopharyngeal insufficiency is failure of closure of the velopharyngeal sphincter, resulting in incomplete separation of the nasal cavity from the oral cavity. During speech, air leaks into the nasal cavity resulting in hypernasal vocal resonance and nasal emissions. During feeding it can result in nasal regurgitation of food. Treatment options may include speech therapy, palate lengthening (**Fig. 6.9**), a palatal flap (**Fig. 6.10**), and augmentation of the posterior pharyngeal wall.

◆ Outcome and Follow-Up

Postoperative care should include pain control, the establishment of a feeding plan, and monitoring of the airway. Routine surgical follow-up is

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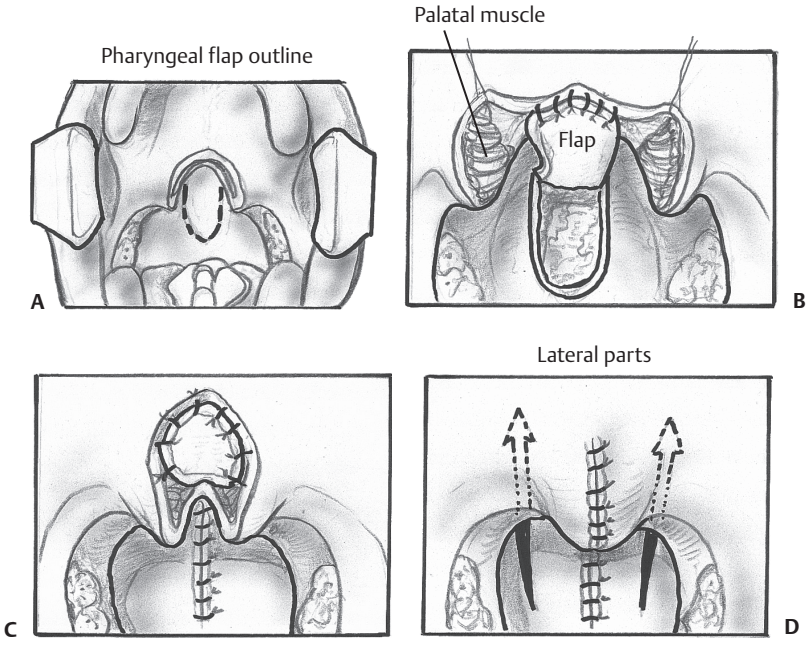

Fig. 6.10 **(A–D)** Palatal flap.

required for cleft repairs at 4 weeks with growth and feeding assessment. Ongoing evaluation and management of the middle ear in combination with audiologic assessment should be completed every 6 months. There should be a referral to a speech therapist for assessment and therapy.

◆ ICD-9 Codes

749.10 Cleft lip
749.00 Cleft palate
749.20 Cleft palate with cleft lip

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7 Facial Plastic and Reconstructive Surgery

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7.1 Craniomaxillofacial Trauma

For ear and temporal bone trauma, see Chapter 2.1.2. For laryngeal fractures, see Chapter 4.1.2. For neck trauma, see Chapter 5.1.1.4.

7.1.1 Nasal Fractures

◆ Key Features

- Nasal fractures are the most common head and neck fracture.
- They have aesthetic and functional implications.
- Closed reduction has limited application.
- Septal hematoma should be recognized early and managed.
- Define the injury precisely and anatomically before planning any intervention.
- Even if no surgical intervention is planned, reevaluate the patient after any traumatic swelling has resolved (usually 1–2 weeks)

Nasal fracture clinical presentation may include epistaxis, change in external nasal appearance, nasal airway obstruction, and infraorbital ecchymosis.

◆ Epidemiology

Nasal fractures are cited as the most common type of facial fracture, accounting for approximately half of all facial fractures. These injuries occur largely in the younger, physically active segments of the population and predominantly in males. The most common mechanism is blunt trauma; these fractures also occur via penetrating and high-energy injuries. Finally, nasal fractures occur not only in isolation but also frequently in conjunction with more extensive facial fractures.

◆ Evaluation

Physical examination is facilitated if the nose is decongested. Visual inspection, manual palpation, and anterior rhinoscopy are essential. If possible, nasal endoscopy can be performed to increase the acquisition of meaningful clinical data.

The integrity of the nasal skin should be assessed. Globally, the examiner should determine if the nose is straight, if there is a deviation, or if there is a C-shaped deformity. Via visual inspection and palpation, the integrity of dorsal support can be determined. Has the nose maintained normal midline

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support, or is there an acute saddle deformity and if so what anatomic disruption is contributing? The nasal bones should be examined. Based on visualization and palpation, are there fractures of the nasal bones?

The tip and lower two thirds of the nose should be examined. Is there an injury to the cartilaginous framework? Determining the status of the septum is frequently underemphasized; however, appropriate appreciation and management of septal injuries is essential to the restoration of optimal nasal function and appearance. And finally, the examiner should look for and document the presence or absence of a septal hematoma. If present, it should be expeditiously and appropriately managed.

Photographs should be done similar to the views obtained for rhinoplasty evaluation. Planar radiographs and computed tomography (CT) rarely add more valuable data than that obtained through the physical examination and medical history.

◆ Treatment Options

Observation

When it has been determined that there is minimal displacement of the nasal bones, minimal soft tissue injury, and minimal compromise of the nasal airway, recommend observation and undertake no active surgical or manipulative intervention.

Closed Manipulation

Under local or general anesthesia, the patient's fractured nasal bones and septum are mobilized and reduced digitally. This may be aided with the use of blunt instruments such as the Boise elevator or Ashe forceps. Traditional closed manipulation is best applied in patients who have actual subluxation or displacement of the nasal bones without significant fracture of the nasal bones themselves.

Modified Open Reduction with Osteotomies

The modified open technique is a limited version of an open technique in which intranasal incisions are made for the introduction of osteotomes. Such patients frequently benefit from manipulation of the nasal bones into position after undergoing bilateral microosteotomies. As with closed manipulation, the position of the septum may impede the success of this technique. A secondary procedure may then be necessary or other treatment offered.

Open Nasal/Septal Repair

The open nasal/septal repair refers to an aggressive approach to the acute management of complicated injuries using existing lacerations or external and intranasal incisions. With this approach the surgeon can reduce, graft, and fixate fractured anatomic components under direct visualization.

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Formal Septorhinoplasty

Formal septorhinoplasty after nasal fracture is employed in two general clinical situations: when there has been minimal displacement and treatment is delayed until swelling has resolved, and as a secondary procedure after initial acute management.

◆ Outcome and Follow-Up

Externally stabilizing dressings should be applied similarly to rhinoplasty care. When there have been significant septal injuries, internal soft silastic splints may provide stabilization and aid in the prevention of synechia. Packing is only occasionally necessary.

◆ ICD-9 Codes

471	Deviated nasal septum
802.0	Closed fracture of nasal bone
802.1	Open fracture of nasal bone

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7.1.2 Naso-Orbito-Ethmoid Fractures

◆ Key Features

- A naso-orbito-ethmoid (NOE) fracture is a severe injury involving depression of nasal bones into ethmoids with associated medial orbital wall fracture.
- It is secondary to a high-energy injury; often associated with intracranial and other injuries.
- It requires open reduction with internal fixation

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NOE fractures result from a high-energy injury, such as a motor vehicle accident. As such, multiple serious injuries are often present, requiring neurosurgery, ophthalmology, and otolaryngology, and/or facial plastic surgery care. Treatment is directed at minimizing complications and achieving adequate functional and cosmetic repair.

◆ Epidemiology

Injury results from severe trauma to the frontal and midface region. The most common mechanism is a motor vehicle accident with an unrestrained driver. Seatbelt and airbag use has reduced its incidence in recent years.

◆ Clinical

Signs and Symptoms

Often, patients with NOE fractures have life-threatening injuries and require initial trauma evaluation and stabilization. Eye and brain injuries are common. Concurrent ocular injury, such as globe rupture, lens dislocation, retinal detachment, or vitreous hemorrhage, occurs in ~30% of patients. Patients may have cerebrospinal fluid (CSF) leakage, due to disruption of the anterior skull base. Typical associated signs and symptoms include severe epistaxis, CSF rhinorrhea, diplopia, epiphora, and severe facial pain. The typical facial deformity resulting from disruption of the ethmoids and medial canthal tendon attachments includes flattening of the nasal dorsum, upward tip rotation and loss of projection, and increased interpupillary distance (traumatic telecanthus).

Differential Diagnosis

It is important to differentiate NOE fractures from isolated nasal fractures (see Chapter 7.1.1), orbital fractures (see Chapter 7.1.3), and fractures that involve only the ethmoid labyrinth. CT imaging allows rapid, definitive diagnosis.

◆ Evaluation

History

Mechanism of injury will help determine possible injuries and severity. Often, the patient may be unable to provide history.

Physical Exam

Exam, as with all traumas, begins with ABCs (airway, breathing, circulation). Care must be taken to protect the cervical spine until it has been cleared. Head and face are inspected and palpated for ecchymoses, soft tissue injuries, and bony step-offs. Telecanthus results from NOE fractures. The average intercanthal distance for women and men ranges from 25 to 35 mm; this is usually 50% of interpupillary distance. A distance of ≥40 mm is considered

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diagnostic of telecanthus. The medial canthal tendon attachment is easily evaluated with the "bowstring" test. While palpating the tendon insertion at the lacrimal crest, the lower lid is retracted laterally. If the tendon insertion is intact, the examiner will feel it tighten like a bowstring. Ideally, the nose should be decongested to allow for an intranasal exam. With use of a headlight and speculum or rigid endoscope, clots and blood are suctioned and mucosal tears, position of the septum, and possible hematomas are noted. Clear fluid may represent a CSF leak. A drop of this fluid on gauze may reveal a "halo sign," indicative of CSF.

Imaging

Thin-cut axial and coronal CT is the most useful imaging study. Fractures of the facial and nasal bones are readily visualized, with excellent detail of the medial orbital walls and lacrimal region. The integrity of the skull base may be assessed, as well as the presence of pneumocephalus or other intracranial injury. NOE fractures can be categorized according to the degree of comminution at the medial canthal tendon insertion:

- Type I: Large central fragment
- Type II: Comminution of central fragment but not involving the tendon
- Type III: Comminution involving lacrimal fossa and tendon attachment site with tendon laceration

Labs

Routine trauma and preoperative lab studies are necessary as indicated, including complete blood count (CBC), prothrombin time (PT), partial thromboplastin time (PTT), electrolytes, and possibly a toxicology screen.

◆ Treatment Options

Treatment of an NOE fracture requires open reduction with internal fixation. For relatively limited fractures, external ethmoidectomy incision may afford adequate exposure. Often, the surgeon may take advantage of large lacerations for bony exposure. However, a coronal (often termed bicoronal) incision is generally used. Stabilization of the central fracture fragment, to which the medial canthal tendon is attached, is achieved with plating to stable bone, especially in types I and II injuries. Occasionally, intercanthal wiring is necessary to achieve an adequate result, especially in type III injuries, in which severe comminution is present. Many surgeons advocate slight initial overcorrection. Bone grafting may be necessary.

◆ Outcome and Follow-Up

Admission and perioperative antibiotics are required. Frequent vision examinations are performed following repair of orbital fractures. Infection, hematoma, or vision change requires prompt attention to correct underlying issues.

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◆ ICD-9 Codes

802 Fracture of face bones
802.0 Nasal bones, closed
802.1 Nasal bones, open
802.7 Orbital floor (blowout), open
802.6 Orbital floor (blowout), close
802.9 Other facial bones, open

Further Reading

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7.1.3 Zygomaticomaxillary and Orbital Fractures

◆ Key Features

- Zygomaticomaxillary complex (ZMC) fractures involve the maxillary face, zygomatic arch, and orbital rim and floor.
- Features of an orbital floor blowout fracture may include enophthalmos, V_2 numbness, diplopia, and possibly an orbital rim fracture.
- The fractured ZMC is most often displaced posteriorly and inferiorly.

A ZMC fracture may be called a tripod fracture because there are three sites of fracture—the frontozygomatic suture, the maxillary face, and the zygomatic arch. Isolated fractures of the orbital rim, floor, maxilla, or zygomatic arch can also occur. Surgical repair is indicated in most displaced ZMC fractures, with fixation of one or more fracture sites.

◆ Epidemiology

ZMC fractures are the most common malar bone fractures and the second most common facial bone fracture after the nasal bones. Zygomatic arch fractures are most common in men (in the third decade) and are caused by sports injuries, motor vehicle accidents, or assault.

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◆ Clinical

Signs and Symptoms

Patients suffering ZMC or orbital fractures may present with epistaxis, periorbital edema, lacerations, pain, vision change, diplopia, or step-off deformities at the site of fractures. Numbness of the ipsilateral upper lip, gum, nostril, and cheek is common due to fractures through the infraorbital foramen (V_2). Orbital signs include chemosis, subconjunctival hemorrhage, immediate proptosis, and enophthalmos. Entrapment of the inferior rectus muscle in the orbital floor fracture can result in diplopia due to impaired extraocular muscle function. Malocclusion may result from either a mobile midface (LeFort) fracture or a concomitant mandible fracture. A depressed zygoma fracture may cause trismus by compressing the coronoid.

Differential Diagnosis

The spectrum of fractures in the zygoma, maxilla, and orbital bones can range from isolated to complicated, from severely displaced to greenstick, and from simple to comminuted. Classification should begin with assessing for midface instability, which would indicate a LeFort fracture (see Chapter 7.1.5). Assessment of the seven bones that constitute the orbit (lacrimal, palatine, frontal, ethmoid, zygomatic, maxillary, and sphenoid) most often reveals fractures at the weakest bones—the lamina papyracea (ethmoid) and orbital floor (maxilla).

◆ Evaluation

Physical Exam

The full head and neck examination must include cranial nerve testing. LeFort fractures have palatal mobility, which can be examined by grasping the upper teeth and pulling the maxillary arch forward and inferiorly. Finger palpation for step-off deformities of the orbital rims, zygomatic arches, nasal bones, and frontozygomatic suture can help determine the site of fractures, although edema may make this difficult. The dermatome of V_2 should be tested and documented. Extraocular movement and vision testing may demonstrate diplopia, indicating an entrapped inferior rectus muscle. Forced duction testing is performed by topically anesthetizing the conjunctiva with tetracaine drops, grasping the episcleral tissue in the fornix (near the inferior oblique insertion) with fine forceps, and testing the mobility of the globe for restriction that could indicate an impinged inferior oblique muscle in the orbital floor fracture. Facial nerve function should be assessed, especially when overlying lacerations are present. An ophthalmologic consult should be performed prior to fracture repair in all patients with periorbital fractures because findings such as retinal detachment or retrobulbar hematoma may preclude immediate surgery or obviate orbital decompression, respectively.

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Imaging

As with any trauma patient, standard ABCs and cervical spine clearance (including C-spine films) should be the first priority. The widespread availability of maxillofacial CT scans with axial and coronal cuts makes plain film obsolete. Both axial and coronal cuts should be ordered to assess the orbital floor and diagnose the exact fracture sites. The pterygoid plates and zygomatic arches are best seen on axial films; the orbital rims, floor, and cribriform plate require coronal cuts. Subcutaneous air is often seen with medial orbital and orbital floor fractures. Foreign bodies or bone fragments near the optic nerve should be identified on CT to prevent damage during fracture reduction. The teardrop sign is seen in a coronal CT scan when orbital fat protrudes through a trap door orbital floor fracture. Indications for orbital floor fracture repair include a defect >1 cm^3, muscle entrapment, and enophthalmos.

◆ Treatment Options

Mildly displaced fractures can be managed with closed reduction. Moderate to severe ZMC fractures should be repaired with open reduction through a combination of approaches that may include transoral (to address the maxillary buttress), eyelid (orbital rim and floor), and upper blepharoplasty (frontozygomatic), or, less frequently, a bicoronal approach (associated supraorbital, frontal, or comminuted zygoma fractures). Fracture fixation usually includes plating at least one or two fracture sites for stability with 1.7-mm titanium plates (often L or J shaped) on the zygomaticomaxillary buttress and a 1.2-mm plate on the orbital rim and/or frontozygomatic suture. Orbital floor fractures are repaired by reduction of the herniated orbital contents through a transconjunctival approach. An implant such as titanium mesh or porous polyethylene is often used to maintain the reduction of orbital contents. Isolated zygomatic arch fractures can be reduced via a transoral, a temporal (Gilles), percutaneous, or, rarely, a coronal approach (comminuted fractures).

◆ Complications

Increased intraocular pressure from an orbital hemorrhage can cause vision loss from the injury itself or as a complication of repair. Prompt treatment includes immediate lateral canthotomy and cantholysis, intravenous (IV) steroids (methylprednisolone), ophthalmology consult, and an urgent CT scan. Orbital decompression may be necessary. The most common complication of repair is inadequate fracture reduction with subsequent enophthalmos.

◆ Outcome and Follow-Up

Standard perioperative antibiotics and steroids are used at the surgeon's discretion. Strict precautions for no nose blowing should be enforced to prevent subcutaneous air.

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◆ ICD-9 Codes

802.4 Closed fracture of malar and maxillary bones
802.5 Open fracture of malar and maxillary bones
76.72 Open reduction of malar and zygomatic fracture

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7.1.4 Frontal Sinus Fractures

◆ Key Features

- Frontal sinus fractures represent 5–15% of all craniomaxillofacial fractures.
- A frontal sinus fracture is the third most common facial fracture.
- Its primary cause is high-velocity blunt force found in motor vehicle accidents (60–70% of frontal sinus fractures) and assault.
- These fractures encompass simple nondisplaced anterior table fractures to comminuted ones involving brain injury and CSF leak.

The two sinuses (rarely, one) are housed completely within the frontal bone, if present. Displaced frontal sinus fractures may result in forehead deformities and traumatic injury to the brain. The frontal bone forms the upper facial skeleton and is closely related to the brain. The orbit lies inferolaterally; the dura, cribriform plate, and frontal lobes of the brain lie posteriorly. The sinus outflow tracts communicate with the nose below. The sinus is made up of the anterior and posterior tables. The frontal bone has the highest tolerance of direct blunt force, with the anterior table typically able to withstand 800 to 2200 lb of force. Because of its greater thickness compared with the posterior wall, a force strong enough to fracture the

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anterior table will usually fracture the posterior table and cause damage to the outflow tracts. The complications of fractures can include deformity, mucocele, brain abscesses, and meningitis.

◆ Epidemiology

Most frontal fractures occur in young males at a higher proportion when compared with women (8:1). Although they can occur at any age, the highest incidence occurs during the third decade of life.

◆ Clinical

Signs and Symptoms

Patients with frontal sinus fractures frequently have other associated facial fractures. Depending on the degree of force, patients may or may not have been conscious during the event and may have suffered significant head trauma. Those who are conscious during the inciting event and remain so will likely complain of frontal pain. Forehead swelling, lacerations, and paresthesia may also be present. Obvious forehead deformity may be present, including significant depression, step-offs, and palpable crepitus, but typically the diagnosis is best characterized via a CT scan. CSF discharge may also be evident from the nose or wound.

Differential Diagnosis

Frontal sinus fractures should be distinguished from simple lacerations and contusions of the forehead. Frontal bone fractures can occur without involvement of the sinus, and CSF rhinorrhea is also seen in other isolated facial fractures. Adjacent facial structures must be evaluated for traumatic involvement (zygomaticomaxillary, orbital, NOE, and skull base fractures).

◆ Evaluation

Physical Exam

The trauma ABCs must be assessed first. Swelling of the oral cavity and pooling of blood in the pharynx can complicate facial trauma by causing airway obstruction. An airway should always be established immediately. Neurologic and visual status evaluation should be done as soon as potentially life-threatening injuries are addressed. Vision loss may be prevented if recognized early via elevated intraorbital pressure or evidence of optic nerve injury. If a patient is either uncooperative or combative, gross evaluation of the facial nerve is recommended when a complete exam is unobtainable. Mobility of the midface, nose, and mandible should be assessed adequately. Lacerations need to be thoroughly irrigated and probed for evidence of any foreign body. CSF rhinorrhea or otorrhea should be considered if any clear discharge is present.

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Imaging

All patients with suspected frontal sinus fractures should undergo a fine-cut (1.5–3 mm) CT scan in axial and coronal planes. Axial images will allow good visualization of the anterior and posterior tables of the frontal sinus as well as of evidence of pneumocephalus. Coronal views allow for visualization of the frontonasal tract, the cribriform plate, and the floor of the frontal sinus. In general, a displacement of the posterior table of more than one thickness of adjacent bone is considered significant.

The role of x-rays is limited, although Caldwell's view can evaluate resolution of fluid accumulation that may be found in the frontal sinus postinjury.

Labs

No specific laboratory tests are required for these patients; however, appropriate screening tests (CBC, chemistry, coagulation studies, drug screening) are often obtained during the initial trauma evaluation and may identify conditions warranting treatment.

Other Tests

When CSF rhinorrhea is suspected, β-2 transferrin testing may be performed on the nasal fluid to confirm the diagnosis. Frontonasal outflow tract obstruction is associated with development of frontal mucoceles, and, although typically best evaluated intraoperatively. Fluorescein, benzylpenicillin solution (white), or methylene blue dye is sometimes used to evaluate the integrity of the nasofrontal duct. Patency is evident when dye is seen intranasally after being introduced in the sinus.

◆ Treatment Options

Antibiotic prophylaxis is recommended for frontal sinus fractures, although some argue that closed fractures and those isolated to a nondisplaced anterior table fracture are not in need of antibiotics. The evaluation of the patients should be directed at an assessment of whether the dura needs to be repaired, whether the outflow tracts have remained sufficiently functional, and whether there is a significant deformity. The concepts of importance in frontal sinus fracture repair are prevention of intracranial infection, prevention of frontal sinus disease such as sinusitis and mucoceles, and a cosmetically acceptable outcome.

Nondisplaced fractures of the anterior table without evidence of nasofrontal duct obstruction and no forehead deformity should be treated nonoperatively. Apart from this fracture, there is no apparent consensus regarding treatment.

The management of frontal sinus injury is evolving, but the goals as listed above have been fairly constant. The management options may include sinus reconstruction and preservation, cranialization of the sinus, and obliteration. The goal is to preserve sinus physiology and consider that the patient will have to be followed closely with serial imaging. Preservation is

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usually considered in patients with more limited injuries and CT evidence of patent outflow tracts. Cranialization is performed when there has been comminution of the posterior table that necessitates dural repair, and damage to the outflow tracts. Cranialization involves removal of the posterior table of the sinus, removal of the sinus mucosa, and obliteration of the outflow tracts. Obliteration of the sinus is performed in similar situations as cranialization, but rather than removing the posterior table of the sinus, the surgeon "obliterates" the sinus cavity with fat or other biologic material.

Surgical access for frontal sinus fracture is best made with a bicoronal flap approach, supraorbital brow incision, or via an existing laceration. Minimally fragmented and nonfragmented fractures can be sufficiently reduced with miniplates. Technological advancement now allows for the use of endoscopic assistance to avoid external scars and frontal sinus fat obliteration by endoscopically opening the nasofrontal outflow tract widely and permanently.

◆ Complications

Complications may include injury to sensory or motor nerves. It is essential to document nerve function prior to surgery. Most patients with sensory change will see improvement over 3 to 12 months. Prolonged retraction can cause temporary paresis of this branch of the facial nerve, but precise surgical technique can help to avoid permanent injury.

◆ Outcome and Follow-Up

The patient is admitted after surgery and monitored for neurologic change, infection, CSF leak, visual change, hemorrhage, and pain control. The patient should be followed closely for the first several months with particular attention paid to follow-up CT to evaluate for mucocele development. The time frame for mucocele development can range from 2 months to 42 years; thus long-term follow up, though difficult, is paramount.

◆ ICD-9 Code

800 Fracture of vault of skull

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7.1.5 Midface Fractures

◆ Key Features

- Midface fractures typically result from high-energy blunt trauma, such as motor vehicle accidents and altercations.
- The mass, density, and speed of the striking object will affect the type and the severity of the facial injury.
- Suspect associated ocular, intracranial, and cervical spine injuries.

The midface transmits masticatory forces to the skull base through a series of three vertical pillars, or buttresses, of thickened bone: the zygomaticomaxillary (lateral), nasomaxillary (medial), and pterygomaxillary (posterior) buttresses (**Fig. 7.1**). These three buttresses are essential for proper facial form and function. Damage to the midface may be life-threatening.

Proper realignment of the vertical buttresses is critical in establishing premorbid dental occlusion, facial height, and projection. Proper alignment

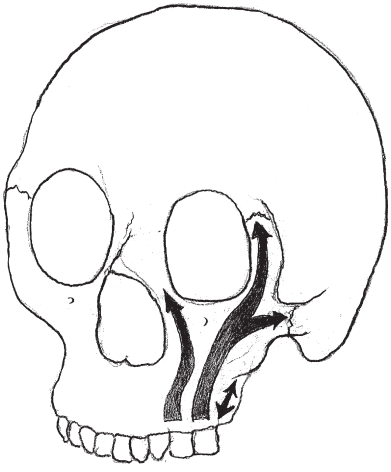

Fig. 7.1 Masticatory forces are transmitted through the zygomaticomaxillary (lateral), nasomaxillary (medial), and pterygomaxillary (posterior) buttresses to the skull base. (From Stewart MG. Head Face and Neck Trauma: Comprehensive Management. Stuttgart/New York: Thieme; 2005:78.)

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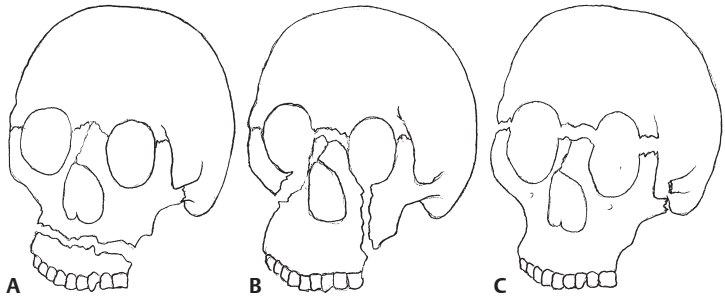

Fig. 7.2 LeFort midface fractures. **(A)** LeFort I fracture separating the inferior body of the maxilla horizontally. **(B)** LeFort II fracture causing separation of the entire maxilla and nasal complex from the cranial base. **(C)** LeFort III fracture causing a complete separation of the entire midface from the cranial base. (From Stewart MG. Head Face and Neck Trauma: Comprehensive Management. Stuttgart/New York: Thieme; 2005:78.)

of the orbital rims and maxillary alveolus (palatal fractures) is essential for establishing facial width, while the zygomatic arches play a key role in restoring both facial projection and width.

In 1901, René Le Fort described three predominant types of midface fractures (**Fig. 7.2**), which he classified as follows:

- LeFort I fractures (horizontal) extend above the dental apices from the zygomaticomaxillary buttress through the nasomaxillary buttress, pyriform aperture, and nasal septum.
- LeFort II fractures (pyramidal) extend through the zygomaticomaxillary buttress, inferior orbital rim and orbital floor, the frontal process of the maxilla, and through the nasofrontal suture.
- LeFort III fractures (aka craniofacial disjunctions) follow a fracture pattern extending from the nasofrontal suture along the medial orbital wall, then traversing the orbital floor and lateral wall before extending through the zygomaticofrontal suture and zygomatic arch.

◆ Epidemiology

Most midface fractures occur in young males and account for some 6–25% of facial fractures.

◆ Clinical

Signs and Symptoms

Patients typically present with a history of traumatic facial injury and may complain of pain, malocclusion, trismus, hypoesthesia, or diplopia. Examination often reveals periorbital ecchymosis, facial and oral mucosal

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edema, facial asymmetry, epistaxis, mobility of the midface, malocclusion (anterior open-bite deformity), or dental injury. Less commonly, CSF rhinorrhea and changes in visual acuity or restriction of extraocular motion are observed. Airway distress associated with severe injuries and decreased level of consciousness associated with intracranial injury may also be present.

Differential Diagnosis

Malocclusion is also seen in isolated dentoalveolar fractures. Adjacent facial structures must also be evaluated for traumatic involvement (mandible, zygomatico-orbital, nasoethmoid, frontal sinus, and skull base fractures).

◆ Evaluation

Physical Exam

As with all trauma patients, the initial evaluation should include a detailed systemic examination using the advanced trauma life support protocol, and then a complete head and neck examination is performed. Airway compromise secondary to mucosal trauma and edema or profuse hemorrhage is possible, and the airway must be secured. The patient's facial region should be thoroughly inspected and palpated for any bony step-offs. Midface mobility is assessed by applying traction to the maxillary central incisors and alveolus with one hand while stabilizing the forehead with the other. All teeth should be accounted for, and if any are fractured or missing, a chest radiograph should be performed to make sure the missing teeth did not enter the airway. A complete cranial nerve examination is critical. In the midface, injury to the maxillary division of the trigeminal nerve is common and facial nerve injury is also possible. Any deficits are documented prior to surgery. Visual acuity and extraocular motion are evaluated and ophthalmologic consultation obtained if these are abnormal or if ocular injury is suspected. Intranasal examination should assess for septal hematoma, septal perforation, and CSF rhinorrhea.

Imaging

All patients with suspected midfacial fractures should undergo a fine-cut (1.5–2 mm) CT scan in axial and coronal planes. High-quality reformatted coronal images are acceptable when cervical immobilization prevents direct coronal imaging. Traditional radiographs such as a Waters view are of historic interest only and should not be used for diagnosis and treatment planning. Many surgeons now routinely obtain CT images immediately postoperatively to confirm anatomic reduction and plating of the fractures. This is extremely useful in panfacial or comminuted fractures.

Labs

No specific laboratory tests are required for this patient population; however, appropriate screening tests (CBC, chemistry, coagulation studies, drug

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screening) are often obtained during the initial trauma evaluation and may identify conditions warranting treatment.

Other Tests

When CSF rhinorrhea is suspected, B-2 transferrin testing should be performed on the nasal fluid to confirm the diagnosis.

◆ Treatment Options

The goal of treatment is restoration of preinjury function and facial aesthetics. Treatment within the first 7–14 days of injury allows tissue edema to subside and lessens the likelihood of aesthetic and functional deficits that are challenging to correct after delay.

Medical

Midface fractures that are nondisplaced, stable, and accompanied by normal occlusion can be observed, but the mainstay of treatment for all other midface fractures is surgical.

Surgical

Historically, midfacial fractures were managed with a prolonged course of maxillomandibular fixation (MMF) and/or suspension wires. Continued mobility of the fracture lines with this type of treatment led to a high incidence of residual bony and soft tissue deformity. As such, the standard treatment for displaced, mobile, or comminuted midface fractures is now open reduction and internal fixation.

Operative treatment begins with exposure of all fracture lines using surgical approaches that may include gingivolabial (maxillary face and buttresses), transconjunctival, or subciliary (orbital rim and floor), upper blepharoplasty (zygomaticofrontal and zygomaticosphenoid sutures), and coronal (zygomatic arches, frontal bone, and nasoethmoid area) incisions. The maxilla is next disimpacted and proper occlusion reestablished with MMF. Inquiry should be made as to preinjury malocclusion or orthodontic/orthognathic treatment. Internal fixation with titanium miniplates is performed along the medial and lateral buttresses and along the inferior orbital rims, zygomaticofrontal sutures, zygomatic arches, and glabellar region as indicated. Consideration should be given to primary bone grafting when interfragmentary gaps >5 mm are present. Miniplates (typically 1.5–2.0 mm) are used for medial and lateral buttresses. Microplates (1.0–1.3 mm) are used on the inferior orbital rim, and the zygomaticofrontal suture is treated with 1.3- to 1.5-mm plates. Although rigid or semirigid plating has greatly improved our ability to treat these injuries, the surgeon must be meticulous in achieving correct anatomic reduction and use exacting technique when adapting the plate to the bone to avoid “fixing” the patient in the wrong position. Most patients are released from MMF at the conclusion of surgery, although arch bars may be left in place for guiding elastics (occasionally useful with severe edema).

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◆ Complications

Complications may include injury to sensory or motor nerves. It is essential to document nerve function prior to surgery. Most patients with sensory change will see improvement over the next 3–6 months. Temporary paresis of the temporal branch of the facial nerve may be seen if prolonged retraction during a coronal approach is required, but with careful exposure and surgical technique, permanent injury is rare.

Malocclusion should be noted postoperatively and surgical technique reviewed. CT may be helpful in determining the cause of the malocclusion, and if fractures malalignment is noted, reexploration should be considered with repeat reduction and plating prior to fracture union. Guiding elastics may assist in very minor dental malocclusions but cannot correct grossly malaligned fractures.

Lower eyelid malposition is largely avoided with gentle tissue handling and meticulous surgical technique. Ectropion is more common after transcutaneous than transconjunctival approaches, though entropion may be observed after transconjunctival approaches (rare). Massage is useful in the postoperative period if lower eyelid retraction is noted. More severe malpositions may require surgical intervention.

Other incisional problems include dehiscence, local infection, hypertrophy, and alopecia and are treated as indicated.

◆ Outcome and Follow-Up

The patient is admitted after surgery and monitored for airway status, visual change, hemorrhage, and pain control. Intermittent ice application is useful in decreasing edema, as is elevation of the head of the bed. The patient is instructed in oral hygiene consisting of frequent mouth rinses and gentle brushing, especially after meals. Diet should consist of soft foods for approximately 6 weeks and then a normal diet may be resumed. Cutaneous sutures are removed 5–7 days after surgery. Intraoral incisions are closed with resorbable sutures and do not require removal.

The patient should be followed closely for the first several weeks, with particular attention given to occlusal status and eyelid position. Long-term follow-up should also be scheduled, but this is sometimes challenging in this patient population.

◆ ICD-9 Codes

802	Fracture of face bones
802.4	Closed fracture of malar and maxillary bones
802.5	Open fracture of malar and maxillary bones
802.9	Open fracture of other facial bones
804	Multiple fractures involving skull or face with other bones

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7.1.6 Mandible Fractures

◆ Key Features

- Mandible fractures may be found with a history of trauma.
- Pain with or without mastication may be an indication.
- There is malocclusion.
- Evaluate concomitant intracranial, airway, and/or cervical spine injury.
- Account for missing teeth.

Mandible fractures are common after facial trauma, occurring second only to fractures of the nasal bones. Diagnosis can often be made by clinical exam, although confirmatory x-ray evaluation is required. Fractures often traverse the alveolus, creating intraoral communication with the fracture site, which leads to contamination of the fractures by oral flora. Early initiation of both systemic antibiotics as well as oral rinses should decrease the risk of infection. Repair is aimed at restoring the occlusion, reestablishing anatomic alignment of the bone fragments, and ensuring healing with minimal morbidity. Mandible fractures are classified as favorable or unfavorable for healing (**Fig. 7.3**).

◆ Epidemiology

Most mandible fractures are the result of interpersonal trauma and motor vehicle accidents; they typically occur in the third and fourth decades of life. In the elderly, falls become a more common cause, and in younger children sports activities and motor vehicle accidents are most prevalent.

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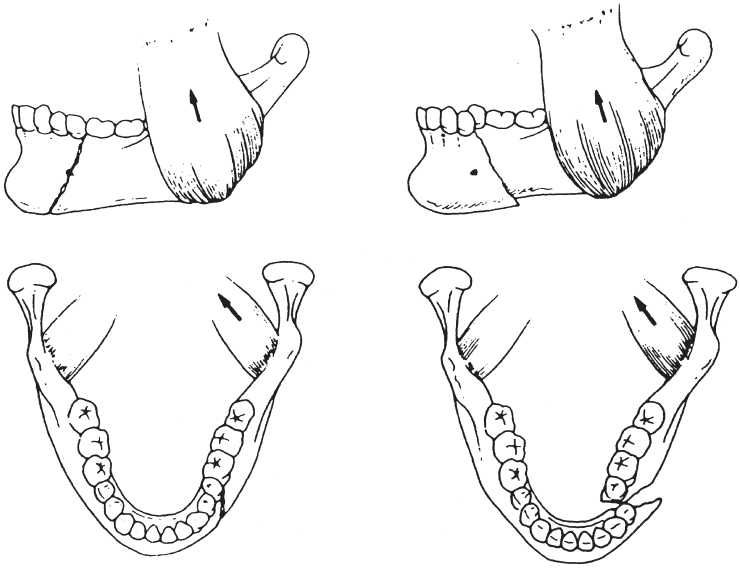

Fig. 7.3 Favorable and unfavorable mandible fractures. (From Papel ID, ed. Facial Plastic and Reconstructive Surgery. 3rd ed. Stuttgart/New York: Thieme; 2009:1002.)

Most studies indicate that the condylar/subcondylar complex is most frequently fractured, followed by the symphyseal region from canine to canine (**Fig. 7.4**). The mandibular angle region is next, followed by the body region. Fractures of the vertical ramus (excluding subcondylar fractures) occur less frequently, and coronoid process fractures are distinctly uncommon. There is some controversy regarding location frequency.

◆ Clinical

Signs

Note the presence of malocclusion, with or without trismus. Edema and ecchymosis may be seen both intraorally and extraorally. Loose or fractured teeth should be identified, and attempts should be made to account for missing teeth (intruded teeth may occasionally be mistaken for avulsions). Intraoral lacerations are common at fracture sites. Paresthesia over the chin indicates injury to one or both inferior alveolar nerves.

Symptoms

Most patients with mandible fractures present because of pain or malocclusion, both of which tend to interfere with eating. Some notice oral bleeding and facial swelling. They may also complain of loose teeth.

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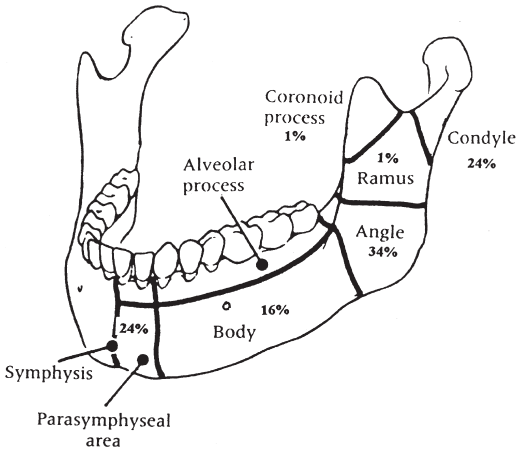

Fig. 7.4 Anatomy of the mandible, with relative incidence of fracture sites. (From Papel ID, ed. Facial Plastic and Reconstructive Surgery. 3rd ed. Stuttgart/New York: Thieme; 2009:1001.)

Differential Diagnosis

If the patient is (or was) unconscious, the nature of the trauma may be difficult to determine. Swelling and muscle injury may masquerade as a fracture. A patient with a serious dental infection, with or without a history of trauma, may have clinical findings of pain, trismus, and malocclusion, which may make it difficult to determine whether a fracture is present.

◆ Evaluation

Physical Exam

Examine for swelling, tenderness, and ecchymosis. Warmth and fever would be signs of infection, usually seen when presentation is delayed. Examination of the mandible itself might reveal mobility of fragments (to bimanual palpation), trismus, and malocclusion. Also look for loose, cracked, avulsed, and/or impacted teeth.

Imaging

Best results will be obtained if plain films, orthopantomogram (Panorex), and CT scans are obtained.

Labs

CBC is indicated to look at hemoglobin if there has been significant bleeding and to look at the white blood cell (WBC) count if presentation is

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delayed. Coagulation studies should be obtained if the patient has medical comorbidities.

Pathology

Pathology studies are generally not applicable, unless there is a concern about a pathologic fracture in a patient with another underlying disease, or in a case of delayed management or complications of prior management, where there may be concern for posttraumatic mandibular osteitis or osteomyelitis.

◆ Treatment Options

Medical

Nondisplaced, nonmobile fractures without malocclusion can be managed nonsurgically. The patient is maintained on a mechanical soft diet. The patient should be seen after 1–2 weeks to ensure patient compliance and that the fracture is healing uneventfully. Any change in occlusion status will require additional treatment. The occlusion class is based on the relationship of the retrobuccal cusp of the upper first molar to the buccal groove of the lower first molar:

- Class I occlusion (normal): The mesiobuccal cusp of the maxillary first molar occludes exactly with the mandibular first molar buccal groove.
- Class II occlusion: The mesiobuccal cusp is mesial or anterior to the mandibular first molar buccal groove.
- Class III occlusion: The mesiobuccal cusp is distal to the buccal groove.

Subcondylar fractures with minimal displacement and without malocclusion may be managed with physiotherapy and exercises.

For both of the above situations, mouth-opening exercises are important to prevent limitations in mouth opening.

Relevant Pharmacology

Antibiotic prophylaxis is recommended from the time of presentation until oral wounds have sealed after the repair. (Precise duration of antibiotic treatment after fracture repair is controversial.) An antibiotic that covers common oral flora is recommended.

Surgical

There are three basic surgical approaches to mandible fractures: (1) placement of oral appliances and application of rigid MMF (also called closed reduction, rigid fixation); (2) placement of oral appliances and the use of training elastics (nonrigid MMF; also called nonrigid closed reduction); and (3) open reduction of the fractures, generally used with rigid fixation of the fragments (also called open reduction and internal fixation or ORIF).

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The most common appliances used for MMF in the United States today are Erich Arch Bars. These are generally fixed to the teeth with wires, and small hubs on the arch bars allow the placement of wires or rubber bands for rigid and nonrigid MMF, respectively.

ORIF may be performed via either intraoral or extraoral approach. "Load-sharing" repairs are generally used when the bone being repaired is solid enough to provide a buttress, so that fixation can be placed in a biomechanically advantaged fashion that takes advantage of the naturally occurring forces of muscle function and mastication. This can be accomplished using the "Champy" technique, with miniplates placed along the "ideal line of osteosynthesis," using monocortical screws to avoid injury to tooth roots and the inferior alveolar nerve. Two plates or two lag screws are generally used in the symphyseal and parasymphyseal regions, a single plate is commonly used along the mandibular body, and one or two plates (the choice is controversial) are used for angle fractures. Compression plates can be used as well along the symphysis and body (not at the angle), but this requires bicortical screws placed along the inferior border, so tension band plates or arch bars must be applied to avoid distraction of the alveolar portion of the fracture.

"Load-bearing" repairs are used when there is inadequate bone to form a buttress and share in the load. This requires the placement of longer, stronger reconstruction plates fixed with bicortical screws along the inferior border of the mandible. At least three and preferably four screws should be placed on either side of the fracture. Load-bearing reconstruction plate repairs are indicated to span areas of mandibular deficiency, such as defects, areas of comminution, atrophic mandibles (edentulous patients), and areas involved with infection (or previous nonunion). The reconstruction plate is also a fallback technique for any mandible fracture, particularly in the angle region after loss of an impacted third molar. Fractures of the condylar neck should be opened if there is significant foreshortening of the ramus of the mandible or persistent malocclusion. The endoscopic approach allows a mostly transoral repair of selected subcondylar fractures. Open reduction of condylar head fractures remains quite controversial.

◆ Complications

A rigidly fixed malreduction should be reoperated, unless there are extenuating circumstances because MMF cannot repair this. Wound infections should be drained and managed expectantly. Loose hardware must be removed. Failure of fixation requires reoperation, and if infection has developed, a stronger, load-bearing repair will be necessary. Nerve injuries (motor and sensory) should be documented.

◆ Outcome and Follow-Up

Routine wound care is indicated. Oral hygiene must be maintained, and antiseptic oral rinses are commonly used several times daily and after meals. A liquid diet is preferred initially, and this is advanced to a mechanical soft diet as tolerated. Postoperative x-rays should be obtained to ensure

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satisfactory reduction of fractures. If satisfactory rigid fixation has been accomplished, MMF will not be necessary. After 1 week, trismus should be treated with mouth-opening exercises. Patients treated with rigid MMF are generally sent to the floor and discharged with wire cutters, in case they need to urgently release the MMF.

Patients should be followed closely for the first 6 weeks. At this point, most fractures are stable enough to allow removal of the arch bars. After this, patients should be followed until normal function is ensured.

◆ ICD-9 Codes

802.20 Fracture mandible
802.25 Angle
802.3 Open
802.30 Open
Upper – see Fracture, maxilla
802.20 Mandible (closed)
802.25 Angle
802.35 Open
802.28 Body
802.27 Alveolar border
802.37 Open
802.38 Open
802.26 Symphysis
802.36 Open
802.21 Condylar process
802.31 Open
802.23 Coronoid process
802.33 Open
802.29 Multiple sites
802.39 Open
802.30 Open
802.24 Fracture; mandible, closed; ramus unspecified
802.34 Open
802.22 Subcondylar
802.32 Open

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7.2 Facial Reanimation and Eye Care

◆ Key Features

- No one modality is universally appropriate for all afflictions of facial nerve function.
- Etiology of the paralysis, oncologic status, type of injury, and location of injury all contribute to the selection of the most appropriate reanimation methods.
- Reanimation techniques are classified into four types: neural methods, musculofascial transpositions, facial plastic procedures, and prosthetics.

For patients with facial nerve injuries, realistic expectations must be established at the initial encounter and candidly discussed between the physician and the patient and his or her family. Several points should be emphasized to the patient before the onset of treatment. First, no reanimation technique will restore the face exactly to its preparalyzed condition. The patient should understand that despite the most meticulous surgery some degree of synkinesis and residual weakness may persist. It is also important to stress that the results of these reanimation techniques may be augmented by physical therapy and rehabilitation.

◆ Location of Facial Nerve Injury

Intracranial

Intracranial nerve injuries most commonly occur during resection of acoustic neuroma or other cerebellopontine angle (CPA) tumors. The incidence of facial nerve injury following CPA tumor surgery is reported to be 2.3%. In the event of facial nerve injury during CPA tumor surgery, immediate repair by either direct anastomosis or graft is advisable. If added length is needed, careful rerouting of the tympanic and mastoid segments of the nerve may add enough length for an end-to-end anastomosis.

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Intratemporal

Intratemporal facial nerve injury is usually encountered in patients following external head trauma with skull base fractures, or iatrogenic injury during or following otologic surgery. Most temporal bone fractures result from motor vehicle accidents and violent encounters. Seven to 10% of these fractures result in facial nerve dysfunction. In temporal bone trauma, facial nerve injury most often occurs in the perigeniculate and labyrinthine sections, with axonal degeneration extending a variable distance in both directions. Management of facial nerve injury following temporal bone trauma is controversial. Most patients who present with complete paralysis at the time of injury have a poorer prognosis than those with incomplete or delayed paralysis.

Extratemporal

Extratemporal injury to the facial nerve may occur during parotid surgery, temporomandibular joint procedures, or facelift procedures, or following traumatic lacerations of the face. The incidence of facial nerve paralysis after uncomplicated parotid procedures is reported at 20% for temporary palsy and 10% for permanent paresis of either the temporal or mandibular branches. Patients at higher risk for facial nerve injury during parotid surgery include children and those undergoing a total parotidectomy. Inadvertent transection of the nerve that is recognized during parotid surgery should be repaired as soon as possible.

◆ Reanimation Options

The order of preference for restoration of function following total unilateral facial paralysis is as follows:

1. Spontaneous facial nerve regeneration (observation)
2. Facial nerve neurorrhaphy (facial nerve anastomosis)
3. Interpositional graft
4. Nerve crossovers (anastomosis to other motor nerves)
5. Muscle transfer
6. Eyelid procedures and prosthetics

Facial Nerve Neurorrhaphy

If the nerve has been completely disrupted, direct neurorrhaphy is the most effective way to reanimate the paralyzed face. The interrupted neural pathway can be reestablished either by direct anastomosis or by inserting a graft between the disrupted segments. Some of the key points in nerve repair are early identification, evaluation of nerve condition, and tension-free anastomosis. The best time to perform surgery is within the first 72 hours, before degeneration has occurred and while the distal nerve can still be stimulated. The disrupted nerve should be approximated with minimal

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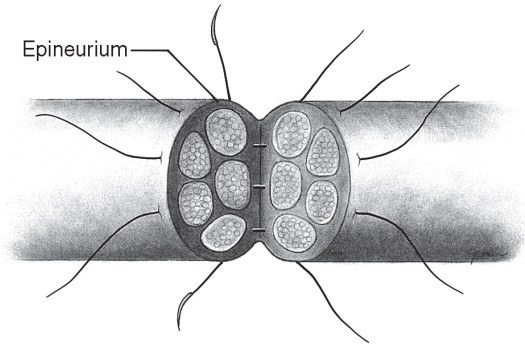
tension. It may be necessary to reroute the nerve within the temporal bone or to gain extra length by releasing the nerve. Factors that influence the success of repair include tension, the character of the wound, the presence of scar tissue, and time lag to repair.

The surgical suturing technique for nerve repair requires magnification, either with loupes or a surgical microscope. The nerve endings should be freshened with a new scalpel blade. At this point, axoplasm may be seen oozing from the proximal stump. Number 8-0 to 10-0 nylon sutures with a 75- or 100-micron needle should be used.

If possible, three or four simple sutures should be placed about the circumference of the epineural layers to achieve adequate union (**Fig. 7.5**).

Interpositional Graft

In cases where patients have undergone prior surgery or have had part of their facial nerve sacrificed or avulsed as a result of severe trauma, direct



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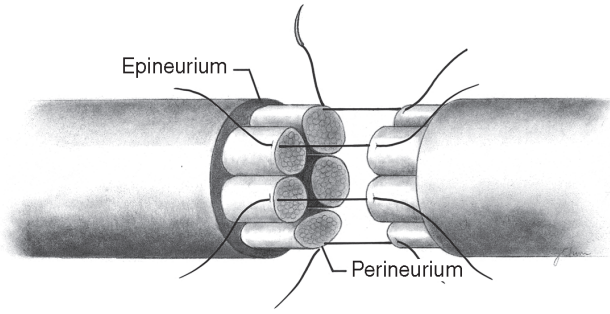

B

Fig. 7.5 **(A)** Epineural nerve repair. **(B)** Perineural (fascicular) repair. (From Burgess LPA, Goode RL. Reanimation of the Paralyzed Face. Stuttgart/New York: Thieme; 1994:13–16.)

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nerve repair is impossible and interposition of a nerve graft is required. This technique is reserved for cases in which direct nerve repair would result in excess tension or when there is loss of nerve tissue.

The greater auricular nerve is the most commonly used donor nerve especially when the nerve graft required is small. Its advantages are its proximity to the operative field and ease of exposure (**Fig. 7.6**). It should be remembered that oncological considerations mandate that the ipsilateral nerve not be used. If more nerve tissue is required, the sural nerve may be harvested.

Nerve Crossovers

This technique is used when direct suturing or cable grafting is not feasible as is often the case after removal or obliteration of proximal or intratemporal portions of the facial nerve. It is particularly useful to treat facial paralysis resulting from intracranial or intratemporal disorders or surgery. These techniques are relatively simple and require one suture line. They provide a powerful source for reinnervation although the results are not always consistent or predictable. Nerve crossovers most commonly utilize the hypoglossal nerve.

Another technique is the "babysitter graft." In this technique, the hypoglossal nerve and facial nerve are anastomosed with the interposition of a free nerve graft, end-to-end to the peripheral facial nerve stump, and end-to-side to the hypoglossal nerve. This technique is typically used in candidates for cross facial nerve grafting.

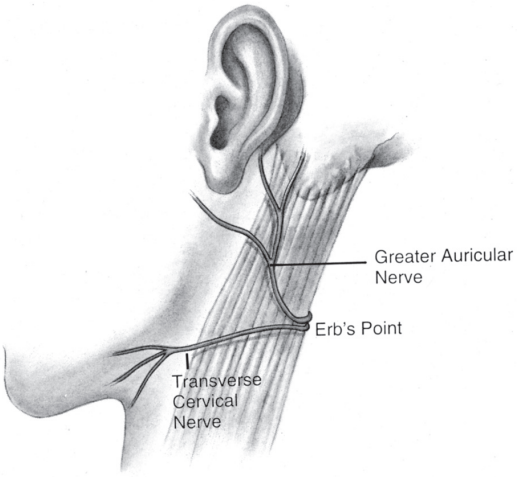

Fig. 7.6 Greater auricular and transverse cervical nerve grafts. (From Burgess LPA, Goode RL. Reanimation of the Paralyzed Face. Stuttgart/New York: Thieme; 1994:18.)

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Cross Facial Nerve Grafting

This technique uses the contralateral normal facial nerve to innervate certain facial muscles on the paralyzed side. This technique should be considered an alternative to hypoglossal or accessory nerve grafting. It should not be performed as long as spontaneous regeneration is still possible or in cases in which direct or cable grafting of the facial nerve is possible.

Muscle Transfer Techniques

Muscle transfer techniques are used when neural techniques are unsuitable. Patients with long-standing facial paralysis (>3 years) are unlikely to benefit from any of the previously discussed reanimation procedures. Severe fibrosis occurs in the distal neuromuscular unit along with atrophy of the facial musculature making reinnervation unlikely.

Regional Muscle Transfer

Muscle transfer techniques entail transplanting a new neuromuscular unit into a region of the paralyzed face. This may be done in conjunction with a nerve graft or a crossover implanted in the transferred muscle. The two basic techniques to accomplish this are regional muscle transposition and a free-muscle transfer.

Regional muscle transfer is usually used to reanimate the lower third of the paralyzed face. The new neuromuscular unit is composed of the transposed muscle with its original nerve supply. Muscles available for these procedures include the masseter, temporalis, and digastric. It should be remembered that in all muscle transfer procedures, overcorrection is desired.

Free-Muscle Transfer

The ideal situation for use of an innervated vascularized muscle free flap for facial reanimation is the large defect seen following a radical parotidectomy. This flap would be used for both reanimation and soft tissue defect reconstruction. The free-muscle flap may also be used in cases of long-term paralysis in which considerable muscle atrophy or soft tissue contracture has taken place. The most commonly used free flaps for facial reanimation are the gracilis muscle, the inferior rectus abdominus muscle, and the latissimus dorsi.

Ancillary Techniques

Facial nerve paralysis may be associated with involuntary eyelid closure and facial movements. These signs may be due to aberrant regeneration of the facial nerve. Botox induces a temporary and reversible neuromuscular blockade and thus is useful in alleviating synkinesis.

◆ Eye Care

Paralysis of the upper branches of the facial nerve results in disorders of the eyelid and lacrimal function. Sequelae include incomplete closure of the eye with corneal exposure, lower lid ectropion with epiphora,

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decreased tear production, and loss of the corneal "squeegee" effect. These factors contribute to inadequate corneal protection, which can result in exposure keratitis, corneal ulceration, and blindness. Management of the eye in a patient with facial paralysis begins with supportive care to protect the cornea. This includes mainly moisturizing the eye and preventing exposure. These measures should be adequate in cases that are temporary or partial. Artificial tears are commonly used to keep the eyes moist. Ointments should be supplemented, especially at night before sleep. Closure of the eye can be achieved by carefully taping both the upper and lower lids. A clear humidity chamber provides moisture and protects the eye from trauma and foreign bodies.

Tarsorrhaphy

Tarsorrhaphy is an effective method of eye protection in patients with facial nerve paralysis and mild lagophthalmos. Functional and cosmetic drawbacks are associated with this type of procedure. A central tarsorrhaphy completely impairs vision and is not cosmetically acceptable as a permanent procedure.

Wedge Resection and Canthoplasty

Wedge resection of the lower lid with canthoplasty is an effective and relatively simple procedure. It is particularly affective when lower lid laxity is mild. In cases with severe lid laxity or ectropion, a canthoplasty procedure is recommended.

Lid Prosthetic Insertion

The use of prosthetics, particularly gold weights and spring implants, has proved extremely useful in eye paralysis.

Gold Weights

The gold weight implant is a very simple procedure that offers consistently satisfactory results. Placement of a gold weight lid load can be performed under local anesthesia and takes advantage of the relaxation of the levator that occurs with attempted eye closure and gravity. The weight in the upper eyelid passively closes the lid. Gold is the material of choice for lid weighting because of its high density, relative inertness, and color, which blends with most skin tones.

Complications of gold weight insertion include extrusion, position shift, infection of the graft, contact allergy to gold, and overclosure of the eyelid due to insertion of the weight too low.

Palpebral Spring Implant

In cases in which there is poor levator action, a palpebral spring implant may prove more efficacious. However, this technique is technically more difficult and has a higher extrusion rate. The palpebral spring is basically a piece of wire and is therefore subject to possible complications, such as breakage, wearing out, migration, extrusion, and infection.

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◆ Conclusion

The patient with facial paralysis presents a unique challenge to the reconstructive surgeon. Options for reconstruction of the paralyzed face include both dynamic and static procedures. Dynamic procedures, nerve repair, nerve substitution, and muscle transfer provide the best functional and cosmetic results and always should be the first choice in the rehabilitation of the paralyzed face. Static procedures are essential tools in the management of the eye and provide options for patients who are not candidates for dynamic rehabilitation. The goals of reanimation are facial symmetry, eye closure, oral competence, and voluntary movement. None of the described procedures can completely restore the paralyzed face to its normal function. Some synkinesis and residual weakness will persist, yet significant improvements in both function and appearance can be accomplished if the goals of reconstruction are kept in mind.

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7.3 Facial Reconstruction

7.3.1 Skin Grafts

◆ Key Features

- Skin grafts are a relatively simple reconstructive method.
- A skin graft retains an important role in oral cavity reconstruction and cutaneous facial defects.
- The skin grafts can be either split thickness (STSG) and full thickness (FTSG).
- Complete immobilization of the graft in the early postoperative period is critical.

Skin is the largest organ of the human body, representing ~16% of the total body weight. Skin transplanted from one location to another on the same

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individual is termed an autogenous graft or autograft. Despite the development of sophisticated reconstructive methods utilized after ablative surgery, such as microvascular free flaps, much simpler approaches to reconstruction continue to be appropriate in many cases. Skin grafting in particular remains an excellent option for defects of the oral cavity, face, and scalp.

◆ Anatomy and Physiology

From superficial to deep, there are three layers of skin: the epidermis, the dermis, and the subcutaneous layer. The epidermis constitutes ~5% of the skin; the remaining 95% is dermis. The epidermis is further divided into the superficial stratum corneum (no nuclei) and the deeper basal layer. The dermis is further divided into the more superficial papillary dermis, and the deeper reticular dermis, which contains hair follicles and sebaceous glands.

STSGs are composed of epidermis and a variable portion of the dermis. FTSGs include the epidermis and the entire dermis. The thickness of STSGs typically used ranges from ~0.012 to 0.018 inches. Although thinner STSGs contract more, they "take" more consistently; thicker grafts contract less but are also more prone to fail. The thickness of FTSGs depends on the thickness of the donor site skin.

Skin graft healing is considered a three-step process. In the first stage, *imbibition*, the graft derives its nutrients from the underlying recipient bed. During the second stage, *inosculation*, preexisting blood vessels in both the graft and the recipient bed meet and form a network. Healing is completed by *neovascularization*, wherein new vessels form within the graft and grow into the underlying tissue.

Properties of the recipient bed are critical to skin graft healing. Skin grafts will "take" on most well-vascularized tissue, including granulation tissue, muscle, fat, perichondrium, and periosteum, and cancellous bone. Conversely, skin grafts will not survive on naked cortical bone or bare cartilage (i.e., tissues without their periosteum or perichondrium). Actively infected tissue should not be skin grafted. Radiated tissue is also a much less favorable recipient bed.

◆ Indications

Common settings for skin grafting in head and neck surgery include oral cavity defects after cancer resection, cutaneous defects of the face after lesion excision or trauma, closure of free flap donor sites (radial forearm, fibula, etc.), and the framework elevation step of microtia repair.

◆ Operative Technique

Split-Thickness Skin Graft

- The donor site of choice is the upper thigh (thick skin, relatively flat surface).
- A STSG is typically harvested with a pneumatic dermatome (e.g., Zimmer, Warsaw, IN).

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- The donor site is shaved, prepped, and draped, then cleaned of prep solution and lubricated (saline or mineral oil depending on model of dermatome).
- The donor site is held taut.
- The dermatome engages skin at 45-degree angle, then is dropped slightly lower.
- The graft is harvested with steady and even pressure.
- The gauze with 1/200,000 epinephrine applied to the donor site for hemostasis.
- The donor may be dressed in several ways, such as with Tegaderm (3M, St. Paul, MN) for 7 to 10 days.
- Ensure the meticulous hemostasis of the recipient bed to prevent hematoma/loss of apposition.
- The skin graft is applied to the recipient site with the epidermis facing out then sutured into place with absorbable stitches, ensuring good apposition with the recipient bed.
- The bolster (e.g., Xeroform [Kendall Company, Mansfield, MA] draped over sponge) is applied to maintain apposition of the graft and recipient bed, secured with tie-over sutures for 3–7 days.

Full-Thickness Skin Graft

- Donor sites include the postauricular area (for thin FTSGs) and the supraclavicular area.
- Grafts are often designed in fusiform fashion to facilitate primary closure.
- The borders of the graft are incised with a sharp knife.
- The edges of the graft are held with a skin hook, and the remainder of the graft is elevated from underlying fat using a knife or sharp scissors (i.e., subdermal plane).
- The graft is thoroughly defatted with scissors or a knife prior to placement.
- Hemostasis is secured with cautery.
- Primary closure.

Skin graft meshing: A skin graft may be meshed to provide coverage of a greater surface area at the recipient site, with expansion ratios generally ranging from 1:1 to 6:1.

◆ Complications

The major complication of skin grafting is partial or complete graft loss. Reasons for graft failure include hematoma, seroma, infection, and inadequate stabilization. Discoloration at the STSG donor site is to be expected.

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◆ Postoperative Care

Feeding via a nasogastric feeding tube should be considered for oral cavity skin graft placement. Bolsters may be left in place for 3–10 days.

Management of the STSG donor site is quite variable; this area may be a source of discomfort for the patient. The STSG donor site epidermis regenerates by secondary epithelialization from the wound edges and from immigration of dermal cells originating in the shafts of hair follicles as well as adnexal structures remaining in the dermis.

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7.3.2 Local Cutaneous Flaps for Facial Reconstruction

◆ Key Features

- Flap classification can be done in different ways, but the consistency is most important. *Method of transfer* is the most widely used system.
- A defect analysis should be done systematically to avoid untoward long-term results.
- Flap design must consider vectors of tension, resultant scars, and areas from which to recruit.

Cutaneous defects can arise from a host of different causes, but skin cancer remains the most common etiology in the Caucasian population. Local facial flaps are widely used for defects that are too large for primary closure or second intention healing. They remain the workhorse for facial reconstruction and should be within the comfort level of all otolaryngologists.

◆ Defect Evaluation

When analyzing a cutaneous defect of the face, there is a series of steps that one should go through to help identify the optimal flap or, more importantly,

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Table 7.1 Defect Analysis

Locate immobile landmarks
Identify areas of optimal recruitment
Orient to lines of face (relaxed skin tension lines, aesthetic units)
Anticipate resultant scars

which flaps will create significant problems, such as distortion, asymmetry, or functional issues (**Table 7.1**).

- First, recognize the "immobile landmarks" of the face. Examples of this would be the hairline, vermillion border of the lip, and nasal alar rim. These critical structures must remain undisturbed by scars as well as by flap tension.
- Second, consider the areas of optimal recruitment surrounding the defect. This issue is approached only after the first question is addressed.
- Third, evaluate the preexisting lines of the face and how are they oriented around the defect. These include the visible wrinkles, relaxed skin tension lines (RSTLs), and borders of the aesthetic units. The face is separated into distinct aesthetic units such as the forehead, nose, and cheek. When possible, it is best to place incisions along the margins of the aesthetic units and use flaps that lie within the same aesthetic unit as the defect.
- Finally, look at the resultant scars and vectors of tension of the given flap. For every flap design, one should be able to anticipate the exact orientation of the final scars and attempt to design the flap in a way that best conforms to the third step, having the scars lie within or parallel to the preexisting lines. Moreover, one must anticipate the vectors of tension for each flap with respect to the landmarks noted in the first step. The flap should not create distortion of critical adjacent landmarks. Ideally, the greatest tension from the flap will align with the lines of maximal extensibility, which generally run perpendicular to the RSTLs.

◆ Flap Nomenclature

The different systems for classification of local flaps include tissue content, proximity of the flap, blood supply, and method of tissue transfer, the last two of which are the principal methods of nomenclature. The blood supply within a flap can be *random* (based on the rich dermal plexus of the face), can have an *axial pattern* (supplied by numerous larger caliber vessels in the dermis and subcutaneous layer that are arranged in an axial pattern along the flap), or can be *pedicled* (maintained by larger, named vessels). The other system is based on the method of tissue transfer (**Table 7.2**).

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Table 7.2 Chart of Local Flaps

Advancement flaps
Simple
Unipedicled (U-plasty)
Bipedicled (H- or T-plasty)
V-Y island flap
Cheek advancement flap
Pivotal
Rotation
Transposition
Interposition
Interpolated
Hinged

Advancement flaps refer to skin paddles that are mobilized in a linear vector to resurface a given defect. They create no distortion to the adjacent tissues, although a standing cutaneous deformity will often arise. Such flaps are rarely used in their truest form. They are further subclassified based on their vascular pedicle, be it a unilateral pedicle, a bipedicle, or subcutaneous pedicle (island flap).

The remaining method of tissue transfer is the *pivotal flap*, in which the tissue transposition has a rotational element as well. A true *rotation flap* moves tissue along the circumference of a circle, around a single, fixed pivot point, such as a scalp rotation flap. Most other flaps have a combined advancement and rotational element to them. A *transposition flap* involves mobilizing tissue over an incomplete bridge of skin (e.g., rhombic and bilobed flaps). *Interposition flaps* are similar to transposition flaps but include elevation of the incomplete skin bridge to the site of the donor defect, such as a Z-plasty. Finally, *interpolated flaps* move the skin paddle and pedicle over an intact skin bridge with its pedicle base removed from the defect. These interpolated flaps are two-staged flaps that require a secondary pedicle division, usually 3 weeks later. The forehead flap is such an example.

◆ Advancement Flaps

The most simple advancement flap is the lateral undermining and mobilization along the margin of a defect with primary closure. When closing a defect primarily, the apices of the defect should be less than 30 degrees to avoid a standing cutaneous deformity. Traditional *unipedicled* (*U-plasty*) and *bipedicled* (*H-plasty*) advancement flaps without any rotational component have a narrow indication. These flaps are used when minimal tension is desired perpendicular to the direction of advancement to avoid distortion of adjacent anatomic landmarks such as the eyebrow. Secondary defects

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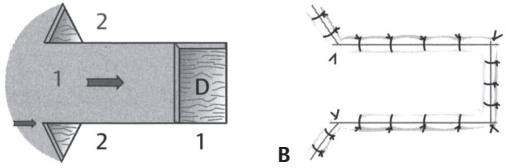

Fig. 7.7 Unipedicled advancement flap (U-plasty). **(A)** Unilateral advancement with direct excision of standing cutaneous deformity (Burow's triangle). **(B)** Closure of defect. (From Weerda H. Reconstructive Facial Plastic Surgery: A Problem-Solving Manual. Stuttgart/New York: Thieme; 2001:12.)

created along the axis of advancement can be addressed by the "halving technique," direct excision of the standing cutaneous deformity, or by advanced excision of a Burrow triangle (**Fig. 7.7**). The leading edge of any advancement flap is the point of maximum tension. One can often design the flap such that the parallel resultant scar lies within the wrinkles or RSTLs of the facial aesthetic unit. The *V-Y island* advancement flap is a unipedicled triangular flap based on a subcutaneous pedicle that is mobilized in a linear vector toward the defect. The V-Y flap creates minimal distortion around the primary defect, but its reach is limited by the subcutaneous pedicle (**Fig. 7.8**). It is well suited for small defects of the upper lip and medial cheek that are in proximity to important anatomic landmarks.

◆ Pivotal Flaps

The *rotation flap* is a pivotal flap mobilized along a curvilinear incision around a fixed point used for tissue that is not extensible, such as the scalp. Two standing cutaneous deformities are created and can be directly excised. The ratio between the peripheral arc of the flap and diameter of the defect is generally 4:1, but rotation flaps on the scalp often require a 6:1 ratio. The *cheek flap* is a combination advancement and rotation flap. The large area from which skin is recruited, together with the natural extensibility of

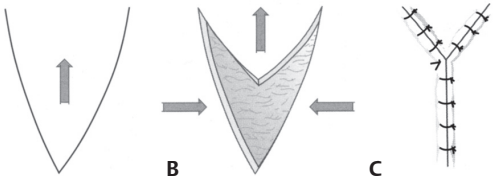

Fig. 7.8 V-Y island advancement flap. **(A)** Two unilateral advancement flaps around primary defect with minimal distortion to surrounding structures. **(B)** The triangular flap is based on a subcutaneous pedicle. **(C)** Defect closed with resultant Y-shaped scar on each side of defect. (From Weerda H. Reconstructive Facial Plastic Surgery: A Problem-Solving Manual. Stuttgart/New York: Thieme; 2001:13.)

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cheek skin, make this flap particularly adept for large defects of the medial cheek. Resultant scars can be camouflaged along the melolabial fold, lower eyelid, and preauricular crease. It is imperative to avoid inferior tension on the eyelid by placing an anchoring stitch between the periosteum of the malar eminence and the undersurface of the flap.

◆ Transposition Flaps

A *transposition flap* mobilizes a broad-based skin paddle over an incomplete bridge of skin (as opposed to the interpolated flap which crosses a complete bridge of skin). The *rhombic flap* is a precise mathematical design that leaves minimal tension or distortion around the defect. The point of greatest tension and its vector are consistent and should be oriented along a line of maximal skin extensibility. This point should be secured with a long-lasting buried suture (**Fig. 7.9**). Small modifications in design, such as a narrower arc of rotation, can reduce the amount of tension from the donor site and distribute this to the tissues surrounding the defect.

The *bilobed flap* is a double transposition flap that minimizes distortion at the primary site by distributing the soft tissue tension over the perimeter of two separate flaps. As such, it is ideal for use near immobile anatomic structures such as the alar rim. Refinement of the bilobed flap design has led to a tighter arc of rotation, reducing the standing cutaneous deformity. Each flap is usually separated by a 45-degree arc of rotation rather than 90 degrees (**Fig. 7.10**). The primary lobe should be aggressively thinned to minimize the amount of pin cushioning that typically occurs. The bilobed flap is excellent for repair of nasal tip defects up to 1.5 cm in diameter but can also be used for reconstruction of cheek defects away from the central face.

The *melolabial flap* is another transposition flap adjacent to the nose and lips, which provides a source of well-vascularized, color-matched skin for reconstruction of the nasal ala, sidewall, and lips. It can be based inferiorly

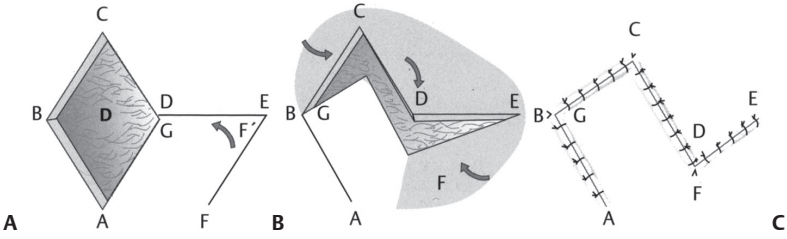

Fig. 7.9 Limberg rhombic transposition flap. **(A)** The flap is designed with 60- and 120-degree angles. **(B)** The flap is elevated in a subcutaneous plane and transposed into the defect. The arrows indicate areas of maximal tension after flap transposition. **(C)** Resultant scar. (From Weerda H. Reconstructive Facial Plastic Surgery: A Problem-Solving Manual. Stuttgart/New York: Thieme; 2001:17.)

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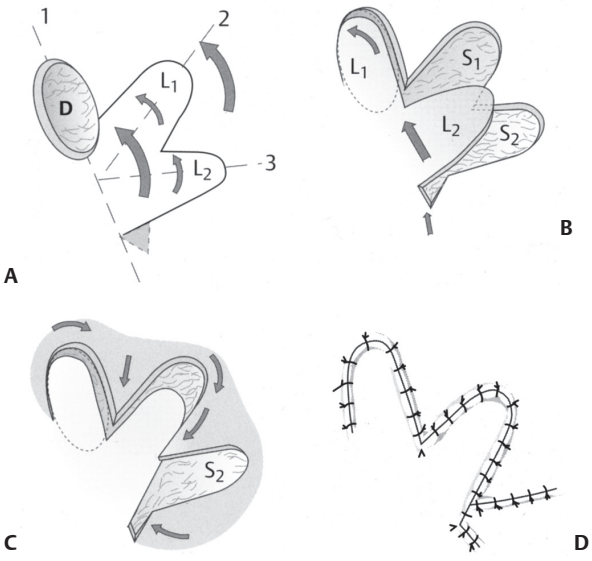

Fig. 7.10 Bilobed double transposition flap. **(A)** The flap is designed with adjacent flaps rotated 90 to 100 degrees. Two wedges are excised to avoid standing cutaneous deformities. **(B)** Inset of flaps and resultant scar. The secondary donor site is closed primarily with a linear scar that can be oriented to run along borders of an aesthetic subunit. **(C)** The surrounding skin is mobilized, and all secondary defects are closed. **(D)** Appearance after closure of all defects. (From Weerda H. Reconstructive Facial Plastic Surgery: A Problem-Solving Manual. Stuttgart/New York: Thieme; 2001:16.)

or superiorly, but the aesthetic junction between the cheek and nose is often blunted.

The two-staged *melolabial flap* and the *forehead flap* are interpolated flaps commonly used in reconstruction of larger defects of the nose. In general, nasal defects >2.5 cm in diameter, defects with denuded bone or cartilage, or wounds in irradiated fields are best reconstructed by heartier flaps such as these. With its ancient history, the forehead flap remains a robust and versatile flap and the workhorse technique for major nasal repair. The pedicle is based over the medial eyebrow area, centered on the consistent supratrochlear artery, which captures the perfusion pressure from the area rich in collaterals. The pedicle is kept narrow, i.e., <1.5 cm, to facilitate rotation. The pedicle can be based on the ipsilateral side of the nasal defect for greater inferior reach, or on the contralateral side for reduced torsion on the pedicle base and visual obstruction to the patient. The donor site defect is closed primarily, and the pedicle division and flap inset is performed after 3 weeks.

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◆ Complications

As for any surgical procedure, knowing the possible complications, ways to avoid them, and how to manage them when they arise are as important to success as the actual technique itself. Flap ischemia, infection, bleeding, and wound dehiscence are the principal complications. Flap ischemia and necrosis are dreaded complications and are related to both tension and impaired vascular perfusion. Flap tension can be attributed to insufficient undermining, flap distension from edema or hematoma, or excessive rotational or linear advancement. Flap perfusion is usually robust in the head and neck, but may be compromised with smoking. The vasoconstrictive effects of nicotine and the preferential binding of carbon monoxide to hemoglobin place local flaps at greater risk. Patient counseling for (at least) perioperative smoking cessation is critical to avoid complications. Wound infection is not common in local flaps of the head and neck, but it can be devastating to the survival of the flap when present. Complications range from prolonged wound healing, scar widening, or even complete flap necrosis. Hematomas can be destructive to a flap by several mechanisms: space-occupying tension, hemoglobin-derived free radical tissue injury, subdermal fibrosis, and an ideal medium for bacterial infection. Wound dehiscence can occur as an end-result of all the aforementioned complications. However, dehiscence may also result from local trauma or dynamic movement of a flap.

◆ Conclusion

Facial defects arise from a variety of causes, such as trauma, skin cancer, or congenital lesions. Their repair remains a challenging part of head and neck surgery as it calls on precise technical skills, attention to details, a three-dimensional perspective for planning, and a creative and artistic element. Developing an algorithm for evaluating facial defects can be useful in terms of avoiding pitfalls such as significant asymmetry, functional issues, or unfavorable scars. Most local skin flaps are versatile and predictable. Familiarity with a host of designs can prove to be a tremendous asset in facial reconstruction.

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7.3.3 Microvascular Free Tissue Transfer

◆ Key Features

- Free tissue transfer offers options to enhance surgical outcomes with improved function, appearance, and quality of life in head and neck cancer and trauma surgery.
- Free tissue transfer techniques require analysis of the defect, available donor sites, and the overall health, function, and rehabilitation potential of the patient.
- Common free tissue transfer donor sites used in head and neck surgery include myocutaneous and myofascial tissue from the radial forearm, latissimus dorsi, rectus abdominus, and lateral thigh; enteral sites such as the jejunum and osseocutaneous flaps with adequate bone supply, such as the fibula and lateral scapula and the iliac crest.

The decision to use a particular donor site for correction of a defect with a patient must account for the extent and functional sequelae of cancer or injury as well as the resultant defect itself and necessary cosmesis given available donor sites. In addition, the treatment plan, patient's prognosis, general functional status, patient and family motivation, and rehabilitation options should be considered in detail. Consideration of relevant clinical concerns supports the physical examination and defect analysis to select a donor site. Free tissue transfer techniques require specialized training, appropriate clinical resources and equipment, and a well-educated interdisciplinary team.

◆ Epidemiology

Free tissue transfer is appropriate for patients of all ages who are physiologically and functionally suited to longer single or staged surgeries and will participate in necessary rehabilitation to make full use of the microvascular reconstruction.

◆ Clinical

Patients who will incur or suffer a head and neck defect that alters anatomic form, affects function of critical structures, and negatively changes appearance and quality of life may benefit from free tissue transfer.

◆ Evaluation

Specific donor tissues are variable, and donor sites are chosen based on recipient site requirements, such as the need and types of surface to be

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reconstructed, the need for bulk, and the need for bone or lubrication. Potential donor sites are then examined for vascular supply, anatomic suitability and anomalies, and age-related changes or tissue changes from comorbid disease. Perfusion and adequacy of vascular donor sites as well as the remaining vascular anatomy in the defect are site-specific.

Imaging

Imaging for free tissue transfer may include angiograms to gauge vasculature, CT, and magnetic resonance imaging (MRI) to assess extent of defect and involvement of surrounding tissues in the field of resection. Noninvasive Doppler or MR angiography (MRA) can assess lower extremity vascularity prior to fibula flap harvest.

Labs

Laboratory assays are somewhat dependent on comorbid disease. In addition to preoperative CBC and chemistries, albumin, prealbumin, and coagulation panels may be drawn.

Other Tests

Allen's test should be performed to select radial forearm sites.

◆ Treatment Options

Medical

Medical therapeutics for free tissue transfer includes management of anticoagulation to ensure flap patency and control of comorbid disease.

Relevant Pharmacology

Unfractionated heparin is used in a continuous IV drip postoperatively to maintain flap patency. Some patients are given aspirin or similar platelet function inhibitors. Streptokinase may be used in relevant flap vessels for flap salvage in anastomotic thrombosis.

Surgical

The most commonly used flaps are of fasciocutaneous or myocutaneous origin. Myocutaneous or fasciocutaneous donor sites used for to reconstruction of soft tissue defects include the radial forearm, rectus abdominus, latissimus dorsi, and anterolateral or lateral thigh. Among these options, the radial forearm free flap offers significant advantages in tissue characteristics, including pliability, thinness, and size to meet many head and neck reconstructive needs. The pedicle of this flap is often particularly long and of large caliber. Repair of the pharynx may benefit from use of an enteral flap. The jejunum and lateral gastric border flaps offer different advantages with pliability and tubed inset; however, secretion production may be of variable benefit for lubrication. The resultant wet speech may be perceived as a significant disadvantage. Harvesting these flaps risks more complications

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with breach of the abdomen and necessitates use of two surgical teams. Bone defects, especially of the mandible, require osteocutaneous flaps from donor sites that offer good bone stock in lengths matched to the adult mandible. Both the lateral scapular border and the fibula can be successfully used. The scapula proves a more difficult site as it requires repositioning the patient and may necessitate two surgical teams. Further, the scapula in women may offer limited or thin bone supply, which will not suffice to correct a large mandibular defect. The scapula flap is associated with transient rotator cuff dysfunction at the least, and the fibula with potential foot drop or vascular sequelae.

The surgery and intraoperative care are focused on ablative procedures or preparation of the traumatic defect, flap harvesting, and final inset of the free tissue flap in the recipient site. Identification of the pedicle and recipient vessels, limiting ischemic time, and achieving anastomosis are critical elements of successful outcome. Anastomosis is performed in either end-to-end or end-to-side fashion. End-to-end using large caliber, healthy vessels is preferred. End-to-side anastomosis seems technically difficult sometimes due to pedicle geometry angularity of the flap vascular supply.

◆ Complications

Complications of free tissue transfer include ischemia and necrosis of the flap from arterial or venous thrombosis. Monitoring flap perfusion is critical to early identification of thrombosis. When recognized early and managed promptly (<6 hours), compromised flaps have a 75% salvage rate when taken back to the operating room. Reexploration of the site is often necessary to pinpoint thrombosis and perform thrombectomy and possible anastomotic revision. Some skin and soft tissue may necrose despite thrombectomy and successful salvage. Wound care to promote healing by secondary or tertiary intention is needed in these cases. Oropharyngeal and laryngeal reconstructions risk fistula formation with or without anastomotic thrombi. Early feeding in oropharyngeal reconstruction is controversial. There is limited research to guide definitive choice of postoperative day to feed, but extended delay may result in nutritional depletion. Fistula formation is often preceded by tenderness, warmth, and erythema. These signs warrant immediate attention and opening the incision to relieve compression as indicated by the physical examination. Further, oral feeding should stop with exclusive use of enteral nutritional support to circumvent use of the oropharynx. Salvage, fistula, and wound care protocols are useful adjuncts to support best practice and good patient outcomes. Donor site complications are rare, site dependent, and include foot drop in fibula free flap harvest and tendon exposure in forearm or fibula donor sites. Rehabilitation with involvement of physical and occupational therapy optimizes patient outcomes when donor site complications occur.

◆ Outcome and Follow-Up

Monitoring the free flap during the postoperative phase is critical to ensure flap survival. Techniques to monitor the free flap depend on the tissue

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composition and location of the flap. Specific monitoring techniques include evaluation of color, capillary refill, turgor, surface temperature, presence of bleeding, skin graft adherence, and auditory assessment of blood flow (Doppler). It is important to protect of the flap vascular supply (e.g., avoiding compression or flexion in the region of anastomosis).

Optimal patient outcomes are a functional, healthy free tissue transfer to restore the defect identified for reconstruction; restoration of function and appearance to the extent possible with the tissue used and the defect corrected; and patient and family satisfaction with regional and overall patient function, quality of life, and caregiver burden.

Follow-up is predicated on expected cancer and trauma protocols and includes flap healing, function, and late complications such as scarring, tethering, and ischemia.

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7.3.4 Bone and Cartilage Grafts

◆ Key Features

- Bone grafts are still commonly used primarily for structural reconstruction of the craniomaxillofacial skeleton.
- The most commonly used bone graft is from the calvarial skull followed by use of bone from the iliac crest, tibial region, and rib regions.
- Cartilage grafts are most commonly used for reconstructive rhinoplasty and in nasal reconstruction, but have also been described for use in other areas such as orbital and eyelid reconstruction.
- The most common source for cartilage grafting is the nasal septum followed by the ear concha and the costal cartilage.

Bone and cartilage grafting remain key components in reconstructive surgery of the craniomaxillofacial skeleton and nasal regions. Although alloplastic implants have supplanted the use of bone and cartilage in some regions, bone and cartilage remain the mainstay in certain reconstructive areas. Bone is essential for structural reconstruction such as in the mandible and along the buttresses of the maxilla in trauma and oncologic situations, whereas cartilage remains most commonly used in the both primary and secondary rhinoplasty as well as in reconstruction of the nose after cancer and reconstruction. It is also sometimes used as a spacer graft in areas such as the lower eyelid.

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◆ Clinical

Calvarial bone is the most common source for use of reconstruction in these areas in this situation. Although other bony defects in acute trauma may be reconstructed with alloplastic materials (e.g., absence of bone along the infraorbital rim), bone is still most commonly used in the primary setting. In acute mandibular trauma with defects, such as avulsive bony loss, the choice for bone grafting is more commonly the iliac crest with a combination of cortical and cancellous bone. Cartilage grafts are particularly useful in nasal reconstruction.

◆ Evaluation

In acute maxillofacial trauma, radiographic evaluation may show comminuting segments of bone in these regions that suggest the need to plan for bone grafting in the preoperative setting. CT scans will guide these decisions.

Cartilage grafts are commonly used in primary or secondary reconstruction of the nose, with the donor site being determined by the type of cartilage required for the reconstructive effort. The most important feature is whether structural or contour reconstruction is necessary. With structural reconstruction, the grafting material of choice is septal cartilage followed by costal cartilage, whereas contour reconstruction can be achieved with both septal and conchal cartilage.

◆ Treatment Options

Calvarial Bone

The most common bone graft used in the midface and upper maxillofacial skeleton is that of calvarial bone. Calvarial bone is harvested most commonly in the parietal skull region away from danger areas such as the coronal and temporal suture lines and away from the midline, where the underlying sagittal sinus exists. The anatomy of the skull is such that there is an outer and inner cortex of bone of varying degrees of thickness. A key feature to harvesting calvarial bone grafts is the assumption that the underlying dura is always nearby and one should be keenly aware of the fact.

A variety of techniques have been described for the harvest of calvarial bone grafts, but the basic technique involves drilling a trough to expose the presumed cancellous layer between the outer cortex and the inner cortex of bone. The graft shape is outlined as this trough is created. Then either a wide curved osteotome or a sagittal or reciprocating saw can be used to elevate the bone graft within the diploic or cancellous layer. It is essential that the surgeon is aware of being in the correct plane at all times; as a rule, an assumption can be made that one is through the inner and intercranial layers if the osteotome or saw moves more freely than expected. After the first graft is elevated, subsequent grafts are commonly much more readily elevated as the surgeon has a better idea of the depth of the outer cortex,

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and further graft harvesting is also facilitated by the greater ease of placement of the osteotome due to the widening of the trough allowed by the harvest of the first bone.

Iliac Crest

The most common graft areas of harvest for the mandible are cortical and cancellous bone grafts from the inner aspect of the iliac crest. This region is approached through an incision over the iliac crest with direct dissection down to the iliac crest. Great care should be taken to reflect the tissues in this region for later precise soft tissue repositioning. A section of the inner iliac crest is removed, followed by the harvest of cancellous bone using curettes. If only cancellous bone is used, the cortical segment is replaced and positioned usually with wires or strong sutures, and the soft tissue muscle elements are resecured to the region.

Tibial Bone Grafts

Tibial bone grafts are used by some surgeons and are harvested at the region of the lateral epicondyle just inferior and lateral to the patella.

Cartilage Grafts

Nasal septal grafts are commonly harvested as part of the routine septoplasty. The key when harvesting septal cartilage for grafting, however, is to harvest as large a piece of cartilage as possible and not to remove it in piecemeal fashion.

Conchal cartilage can be harvested either through an anterior approach (inside the antihelical rim) or by a postauricular incision just over the cavum concha and concha cymba. Through either approach, an incision is made in the conchal cartilage leaving a several millimeter rim along the antihelix to allow for persistence of the structure of the ear followed by an incision as far as the external auditory canal inferiorly and to the extent of the inferior crus of the ear superiorly in the concha cymba. A kidney bean–shaped piece of cartilage is routinely removed for this purpose. The incision is closed, and then, because of the dead space created by the harvest of this cartilage, either a through-and-through bolster or quilting-binding sutures are placed.

Costal cartilage is harvested generally through a right inframammary incision in the region of the seventh and eighth ribs. Depending on the amount of cartilage needed, after dissection through the chest and intercostal muscles, either a partial outer cortex section of cartilage is sharply removed or careful dissection around the cartilage onto the pleura is performed. The muscle layers are carefully reapproximated, as is the skin, and a pressure dressing is applied.

◆ Complications

Complications in graft harvesting generally occur at the time of harvest, such as intracranial entry. The key is the diagnosis of dural tears or

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entry into the brain itself. Neurosurgery evaluation may be required, but small tears can generally be managed by simple reapproximation of the dura and suture closure. One should vigilantly observe for any signs of postoperative subdural or extradural CSF or blood collection, however. Hematomas can occur in the subcutaneous region of the scalp and iliac crest as well as in the chest region, so pressure dressings are often applied. With respect to iliac crest bone grafting, postoperative pain may result in gait disturbance, and this is a common reason this donor site is avoided.

As with any septal surgery, septal perforation is a concern with cartilage graft harvesting and standard techniques for mucosal coverage are essential.

The most common complication with a conchal graft harvest is mild deformity because of a slight collapse of the ear along with scar deformities. Finally, with respect to rib or costal cartilage graft harvesting, the most common adverse sequela is a slight depression in the region of the graft harvest along with the scar in this region. An acute complication mentioned previously is pleural entry and subsequent pneumothorax. If pleural entry is diagnosed at the time of positive-pressure breathing intraoperatively, a drainage tube under suction is placed in the pleural cavity through the tear and suction is applied while closure is being performed; the tube is then withdrawn during a positive-pressure breath. A postoperative x-ray is always taken and will determine whether there is a postoperative pneumothorax or hemothorax. The latter situation may require chest tube drainage, but this is quite rare.

◆ Outcome and Follow-Up

The donor sites for bone grafts are generally treated as with any other wounds with adequate cleaning and moist wound care.

Outcomes with bone grafting are generally favorable, particularly when rigid fixation is used. This is based on long-term follow-up over at least several months as well as determination of the rigidity of the region as based on function of the mandible and maxilla. When complications occur in terms of poor healing, it is generally manifested by pain, mobility, and even infection, thus necessitating further intervention. Resorption of bone can occur with rigid fixation. This is relatively unusual.

Relative to cartilage grafting, none of these cartilage grafts commonly resorb, although adverse sequelae of the grafting can occur if they are improperly positioned or inadequately secured.

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7.3.5 Incision Planning and Scar Revision

◆ Key Features

- Incision planning and scar revision requires an understanding of facial anatomy and aesthetics and of advanced principles of wound healing.
- Patients who have been injured frequently bear psychologic trauma induced by the initial event or the resulting deformative scar.
- Timing of scar revision is important and depends on a variety of factors, including type and location of injury.
- The RSTLs represent the directional tendency of the skin to be more or less extensible, depending on the axis.

Incisions heal best when oriented with RSTLs and closed without tension. Scar revision is generally performed when the scar is mature (~6–12 months) and involves reorienting the scar into the RSTLs, resurfacing, or reducing the scar into shorter segments. Steroid injection for hypertrophic scars and keloids should be performed within the first month if scars are inflamed, painful, or persistently firm.

Improperly oriented incisions and wounds are more likely to be noticeable, so whenever possible they should be oriented into the RSTLs (**Fig. 7.11**). These lines are perpendicular to the pull of the underlying muscles (except around the mouth) and are best found by pinching the skin. Incisions should be placed into the hairline whenever possible except low on the forehead of males (due to male pattern baldness). Incisions in concave areas should

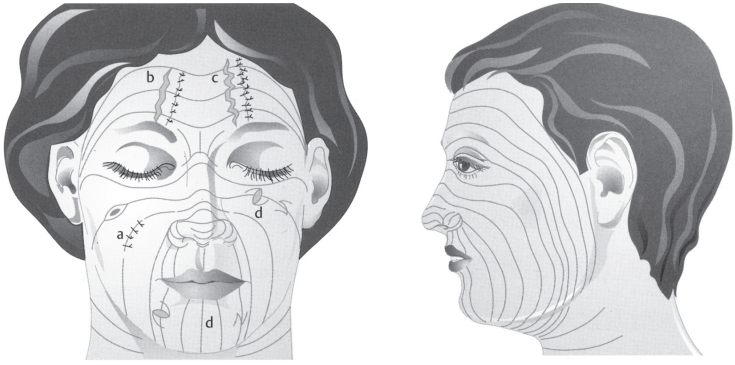

Fig. 7.11 Relaxed-skin tension lines (RSTLs) with elliptical excision in the RSTLs. (From Weerda H. Reconstructive Facial Plastic Surgery: A Problem-Solving Manual. Stuttgart/New York: Thieme; 2001:6.)

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be interrupted to avoid scar contracture. Scars >2 cm in length or >2 mm in width can usually be improved. Common revision techniques include reorientation, irregularization, resurfacing, and direct excision.

◆ Clinical

Signs and Symptoms

Most scars are physically asymptomatic. Patients may present with pruritus or limitation of movement secondary to contractures. Scarring in certain areas of the face can contribute to functional problems, especially around the eyes or mouth.

Differential Diagnosis

It is important to rule out other types of skin lesions, such as infection, manifestations of systemic disease, and cutaneous malignancy.

◆ Evaluation

Physical Exam

The etiology (**Table 7.3**), size, location, and orientation of the scar are the most important features when planning revision. Scar revision should not be performed until the scar is mature, that is, white as opposed to red and without any surrounding inflammation or induration. Maturation typically requires 6 to 12 months. Significant excision or reorientation cannot be performed if the skin is under considerable tension. Skin type also determines the propensity of the scar to pigment or heal favorably.

Table 7.3 Etiology of Unfavorable Scar Formation

Genetic
Fitzpatrick skin types III and above. Darker skin types tend to hyperpigment.
Iatrogenic
Excessive trauma to the wound edges, failure to approximate wound edges at an identical level, and failure to sufficiently evert wound edges at the time of closure
Circumstantial
Poor location or orientation of the wound, perioperative trauma, and postoperative mismanagement
Idiopathic

◆ Treatment Options

In general, the most straightforward option for a scar that is directed opposed to the RSTLs is to relocate the scar into an RSTL, into a hairline such as the brow, or into junctions of facial subunits such as the nasolabial fold. Wide scars that

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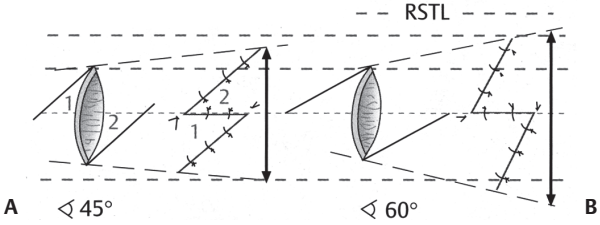

Fig. 7.12 Simple Z-plasty. **(A)** The scar, which crosses the RSTLs almost at right angles, is excised and dispersed with a 45-degree Z-plasty. Flaps 1 and 2 are transposed, causing a slight lengthening of the tissue in the direction of the arrow. **(B)** Transposing flaps 1 and 2 in a 60-degree Z-plasty produces even greater tissue lengthening (*arrow*). (From Weerda H. Reconstructive Facial Plastic Surgery: A Problem-Solving Manual. Stuttgart/New York: Thieme; 2001:8.)

are favorably located may be simply excised or serially excised into a border or RSTL. Elongated scars or wide flat surfaces, such as the forehead and cheek, can be irregularized using a W-plasty, geometric broken-line closure (GBLC), or compound Z-plasty. GBLC is often considered a technically more difficult but better technique for longer scars. The idea of these techniques is to place as many segments as possible into the RSTLs for camouflage.

Z-plasty can be used to reorient the scar 90 degrees into an RSTL or to increase the length of a contracture up to 125% (**Fig. 7.12**). The traditional Z-plasty consists of three incisions of equal length and two 60-degree angles with the central limb of the scar to be excised. Raising the resultant triangular skin flaps in the subcutaneous plane allows the tips to be transposed, resulting in a new central limb perpendicular to the original. Z-plasty works well for scars of the nasolabial fold and mentum.

In situations in which the size and elasticity of the scar preclude closure without distortion of nearby structures, tissue expansion or serial excision may be required. Resurfacing by dermabrasion or laser may be useful for uneven scars, multiple scars or scars that cover a broad, flat area where excision is not practical. Combinations of the above techniques more often lead to the best results.

◆ Outcome and Follow-Up

Scars should be followed closely to ensure that inflammation and induration improve within 1 to 2 weeks and that they gradually soften and become less red. Intralesional triamcinolone acetonide (10 mg/mL) can be injected monthly until the scar is stable. Recent literature has suggested that 5-fluorouracil may also be helpful when injected into scars. Large studies are not yet available for this finding. Patients should massage the scar several times daily and keep it covered with silicone sheeting or ointment as much as possible. Furthermore, limiting tension on the wound by keeping a new scar taped for up to 6 weeks can also be helpful.

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◆ ICD-9 Codes

709.2 Scar, disfiguring
701.4 Hypertrophic scar/keloid

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7.3.6 Implants and Fillers

◆ Key Features

- Alloplastic implants are used to achieve predictable structural volume changes for osseous cheek and chin deficiencies.
- Fat grafting provides the ideal method to restore facial volume loss for the aging face.
- A wide range of injectable fillers can provide temporary to permanent options in an office-based setting.

◆ Epidemiology

Everybody ages: fat grafting and injectable fillers provide permanent and temporary methods, respectively, for volume correction of the aging face. Alloplastic implants are used more effectively to provide structural volume changes to individuals with related anatomic deficiencies, especially alloplastic chin augmentation for microgenia.

◆ Clinical

Patients who present for correction of the aging face are focused primarily on the perceived effects of gravity and wrinkles, whereas volume deficiency, which is overlooked, is perhaps the single most important manifestation of the aging process that should be addressed. Fat grafting provides a permanent correction of volume deficiency (if properly performed) that is only contravened with further aging. Injectable fillers, which are temporary and office-based,

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can be used as an alternative to fat grafting for volume correction and may be more ideally suited for facial line correction and lip enhancement.

◆ Evaluation

When evaluating the aging face for volume deficiency, the strategic areas for correction with fat grafting (or alternative filler) include the temporal hollow, brow and upper eyelid deflation, inferior orbital rim, nasojugal groove, anterior and lateral cheeks, buccal region, precanine fossa, prejowl sulcus, anterior chin, and lateral mandible. Fat grafting is not particularly useful for lip enhancement or correction of facial lines, and injectable fillers are preferred. When considering alloplastic chin augmentation, the patient's dentition should be carefully evaluated to determine whether orthotic or orthognathic procedures are more appropriate.

◆ Treatment Options

Medical

Implanted in the perioral region materials such as expanded polytetrafluoroethylene (ePTFE) strands have limited contouring capacity and can extrude, become infected, or be palpable. For this reason, injectable fillers are preferred in highly mobile facial regions. The injectable fillers listed in **Table 7.4** are currently approved by the U.S. Food and Drug Administration (FDA) for use in the human body (although some are used off-label; e.g., injectable silicone). Surgeon experience and preference will affect material choices.

Surgical

Fat grafting is the ideal method for volume correction of the aging face or to manage complex three-dimensional defects that may arise from cancer, trauma, or other pathology that may not be as precisely addressed with standard alloplasts. As mentioned, fat grafting does not provide tremendous benefit for lip enhancement or facial lines, which are better managed with office-based injectable fillers. For the aging face, fat grafting provides ideal improvement over alloplastic implants, as implants can actually worsen the hollowness that becomes more evident above (under the eyes) and below (submalar) the implant, whereas fat grafting can more effectively contour the extensive and subtle panfacial volume loss elaborated in the physical examination section of this chapter.

◆ Complications

Complications following alloplastic implant (malposition, infection, extrusion) usually must be managed with removal and subsequent reinsertion. Contour problems that arise with fat grafting are difficult to manage. A discrete bulge or lump should be physically excised if microliposuction fails. Undercorrection or partial resorption can be easily managed with injection of additional fat.

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Table 7.4 Injectable Fillers

Filler	Benefits/Uses	Limitations	Longevity
Hyaluronic acid (Restylane*/ Juvéderm Ultra†)	Good for emptiness around the eyes, fine lines, lips and folds, also entirely reversible	Not as good for deeper folds or for volume correction	Generally between 6 to 12 months
Hyaluronic acid (Juvéderm Ultra Plus†)	Good for deeper folds and lines, also entirely reversible	Not good for fine lines and lips	Between 6 to 12 months
Hyaluronic acid (Captique‡)	Almost no swelling, reversible, good for lip enhancement in someone that cannot handle even a day of swelling, very smooth result, also entirely reversible	Very short longevity (low hyaluronic acid content), can last as short as 6 weeks depending on the individual	Generally between 2 to 3 months
Calcium hydroxylapatite (Radiesse§)	For deep lines, folds, and for volume correction, very smooth and even results in these areas	Irreversible (but temporary), not good for lips, fine lines, and around the eyes	Generally 6 months to a year and a half
Liquid silicone (Silikon‖)	Permanent correction, excellent for lips (smoothest, softest product available), acne scarring, contour irregularities, certain types of nasolabial folds, nasal contour irregularities	Irreversible (and permanent), not good for fine lines, around the eyes, or for larger volume correction. Must be undertaken with small amounts over many months to achieve a safe result.	Permanent (minus the effects of aging)
Poly-L-lactic acid (Sculptra¶)	Panfacial volume loss (except around the upper face), smooth long-term results for volume correction. Ideal for HIV-related lipoatrophy	Not good for lips, nose contour correction, around the eyes/upper face, also not a great primary method for correction of facial lines if that is main concern of the patient. Expensive and must wait many months to see the change, risk of nodule formation	1 to 2 years

Abbreviation: HIV, human immunodeficiency virus.
*Restylane, Medicis Aesthetics, Inc., Scottsdale, AZ
†Juvéderm Ultra, Allergan Inc., Irvine, CA
‡Captique, Genzyme Pharmaceuticals, Cambridge, MA
§Radiesse, BioForm Medical, Inc., San Mateo, CA
‖Silikon, Alcon Labs, Fort Worth, TX
¶Sculptra, Sanofi Aventis U.S., Bridgewater, NJ

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Hyaluronic acid is the only reversible injectable filler; it can be eliminated in less than a day with injectable hyaluronidase (Vitrase, ISTA Pharmaceuticals, Inc., Irvine, CA). Vascular compromise, especially in the glabellar region, following use of injectable fillers is an emergency that can lead to tissue loss and skin necrosis and should be managed with hyaluronidase (for hyaluronic acid products), warm soaks, nitropaste application, and possible subcutaneous heparin until improvement and resolution of the condition.

◆ Outcome and Follow-Up

There is really no significant postoperative care that is needed for injectable fillers, fat grafting, or alloplastic implant placement.

Unlike traditional otolaryngologic procedures, the endpoint is simply aesthetic improvement that meets the rigorous standards of the surgeon and patient alike. Follow-up is tailored to surgeon preference and patient desire for visitation and further intervention.

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7.4 Cosmetic Surgery

7.4.1 Rhytidectomy

◆ Key Features

- Reversing the undesirable signs of the aging face has become a trend in societies the world over.
- Although many techniques have been described to lift sagging facial tissues, some are more effective and long-lasting than others.
- Recommendations should be modified to address the problems specific to each patient.
- Postoperative results of a rhytidectomy (aka rhytidoplasty or facelift procedure) should be designed to produce a “natural” and “unoperated” appearance.

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◆ Epidemiology

All faces sag with aging. The skull gets smaller and fat is redistributed from the cheeks into the jawline and neck. As facial skin loses its elasticity, it responds to the downward forces of gravity. Prolonged stress, sun exposure, and illness seem to speed up the aging process, making the person appear older than he or she actually is.

◆ Clinical

Signs and Symptoms

Patients in their late forties may present with early sagging of the cheeks and deepening of the melolabial creases. With each decade, the conditions worsen, resulting in drooping of the forehead, lateral brows, cheeks, and neck. Facial rhytides become more pronounced with each passing year, especially in the areas of facial animation.

In some patients, the platysmal muscles in the midline of the neck become separated and migrate laterally, producing vertical banding from the clavicle to the submental region. Many patients develop submental fat pads. Sagging and bulging tissues of the upper and lower lids are generally seen as well.

Differential Diagnosis

It is important to remember that surgery can reposition drooping tissues and remove loose skin; for facial rhytides, however, a resurfacing procedure is generally required. Dermabrasion, chemical peeling, or laser resurfacing can provide more permanent results in wrinkled skin. In general, unless it takes 2 weeks for a resurfaced area to heal, minimal long-term improvement is expected.

Botox and injectable fillers provide only temporary improvement and need to be repeated several times each year. Injectables are not recommended for pronounced rhytides. Fat or fascial grafting may provide more permanent improvement in deep folds and creases.

◆ Evaluation

Physical Exam

Five facial regions should be examined for ptosis and loss of elasticity: forehead, temporal, cheek, neck, and submental areas. Special attention needs to be paid to the upper and lower lid areas as the forehead, brows, temporal, and cheek tissues are lifted. Some of the loose tissues in the upper lid may be improved by forehead and brow lifting; lifting the cheeks may accentuate loose skin in the lower lid regions.

The lower cheeks and neck should be examined to determine whether liposuction should be included in the treatment plan. If sagging muscles are present, they must be lifted and secured with several sutures that produce fascia to fascia closures.

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All branches of the facial nerve should be examined preoperatively to ensure full function. Photographs taken during facial expression maneuvers are helpful for documentation.

Imaging

Unless an underlying bony deformity is suspected, no imaging examinations are recommended for rhytidectomy.

Labs

Metabolic profiles, bleeding studies, and urinalysis are standard preoperative tests. For patients over the age of 40 (or high-risk patients), electrocardiography (ECG) is recommended. Medical clearance from the patient's personal physician is also recommended.

Other Tests

The value of quality photographic documentation before elective facial plastic surgery cannot be overemphasized. Photographs should also be taken from frontal, oblique, and lateral positions, bilaterally.

◆ Treatment Options

The "facelift procedure" is not a single operation but *a series of procedures*, each designed to lift, reposition, and tighten redundant skin, muscles, and fat in the face and neck. A variety of approaches have been described, and new ones seem to arise almost daily. Surgical approaches to the aging face vary from surgeon to surgeon. Some surgeons advocate ultraconservative techniques, meaning limited incisions in front of (and behind) the ear, minimal undermining of the skin and no imbrication suturing of facial muscles and their enveloping fascia. In many cases, lifting is achieved only by lifting skin.

Other surgeons advocate more radical techniques, involving incisions from the occipital region, around the ear, into the hairline and across the head to join with those of a similar nature on the opposite side of the face and neck. In some cases extensive submental muscle and fat work is performed under the chin as a "routine" part of facelifting. The face and neck skin may be freed from ear to ear, connecting the flaps under the chin, and deep dissection may extend beneath the superficial musculoaponeurotic fascia, identifying the various branches of the facial nerve. Liposuction of the lower face and neck may also be added if indicated (see Chapter 7.4.8).

In recent years, minimally invasive techniques have been on the rise. Many of those, however, have been abandoned rather quickly because results appear to be short-lived.

◆ Complications

Complications of rhytidectomy include the following:

- Hematoma: Most common complication after rhytidectomy

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- Nerve injury: Permanent motor nerve paralysis occurs at a rate of 0.5 to 2.6%. The marginal branch is most commonly injured.
- Infection (rarely severe)
- Skin flap necrosis: More common in smokers and in patients with longer and thinner flaps
- Hypertrophic scarring: Predisposing factors for hypertrophic scarring include race, ethnicity, and skin type or family history.
- Alopecia and hairline/earlobe deformities: May be caused by excessive tension on suture lines and is often transient parotid gland pseudocyst; may occur after trauma to the parotid gland when raising the superficial musculoaponeurotic system flap.

◆ Outcome and Follow-Up

Postoperative care for rhytidectomy is similar to that for managing major flap reconstructions following head and neck cancer or trauma. Subcutaneous drains, negative-pressure vacuum systems, subcutaneous tissue sealants, or pressure dressings may be used to minimize the occurrence of postoperative bleeding.

Flaps should be monitored for vascularity and possible accumulation of body fluids between skin and underlying tissues. Hematomas and seromas should be evacuated as soon as possible.

Avoidance of undue tension on suture lines is important to minimize scarring. Suture or staple removal should be performed within ~7 days. Preoperative and perioperative antibiotics are optional.

Patients should be counseled on avoiding nicotine and vessel-constricting agents during at least the first 2 postoperative weeks. Time-release niacin and topical nitroglycerin paste are often helpful should flap vascularity appear to be compromised.

A rhytidectomy lasts for life, in that the skin removed at surgery never returns. Patients should always appear younger than their chronologic age. However, the aging process is ongoing and brings with it *additional* sags and bulges to the skin and tissues that were left behind with the first operation.

Secondary lifting or "tuck-ups" are often beneficial to help maintain a more youthful appearance. Timing of secondary surgery varies from surgeon to surgeon and from patient to patient. Some techniques require earlier secondary intervention than others. Some patients age more rapidly than others, causing the appearance of new sags and bulges sooner than in their peers. Stress and chronic illness seem to play a part in premature aging.

Skin resurfacing (chemical peeling, dermabrasion, and laser surgery) several months after rhytidectomy produces new collagen and elastic fibers, creating a more youthful look that seems to last for years.

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7.4.2 Brow and Forehead Lifting

◆ Key Features

- Brow ptosis not only creates aesthetic issues but may be associated with a functional visual field deficit.
- The initial consultation should be used to evaluate the orbital complex, the upper third of the face, and the position of the hairline.

Over time, the aging face bears the cumulative effects of sun exposure, loss of soft tissue elasticity, and dermal atrophy in a predictable manner. The resultant brow ptosis not only creates aesthetic issues but may be associated with a functional visual field deficit. The muscular elevators of the forehead become hypertonic in an effort to combat brow ptosis. This results in prominent horizontal forehead wrinkling. These interdependent processes produce a tired facial appearance. The aging face surgeon has multiple brow-lifting techniques and surgical approaches which can be tailored to the individual patient.

◆ Anatomy

Forehead and Scalp

The forehead is the region from the superior brow to the anterior hairline (trichion). The layers of the scalp, from superficial to deep, include the skin, subcutaneous fat, galea fascia, a loose areolar layer, and the periosteum. Galea fascia envelops the frontalis and connects it to the occipitalis muscle. The galea aponeurosis is contiguous with the superficial musculoaponeurotic system of the face below and the temporoparietal fascia (TPF) laterally. The periosteum of the frontal bone merges with the arcus marginalis of the orbit inferiorly. Laterally, at the temporal line, the periosteum fuses with the galea, TPF, and deep temporal fascia to form the conjoined tendon.

Paired frontalis muscles are the principal elevator of the forehead. They originate from the galea and insert onto the overlying skin. They are responsible for the transverse frontal rhytides. The corrugator supercilii muscles originate from the periosteum along the medial supraorbital rim. They insert laterally onto the skin along with the frontalis and orbicularis oculi. They are primarily responsible for the vertical rhytides of the glabella. The procerus muscles originate from the periosteum over the nasal bones and insert onto the skin between the eyebrows. They are principally responsible for the transverse rhytides of the glabella.

The sensory innervation of the forehead is supplied by the supratrochlear and supraorbital nerves. These represent terminal branches of the first division of the trigeminal nerve. In most skulls, the supraorbital nerve exits from a supraorbital notch along the medial supraorbital rim. However, 10%

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of nerves will exit from a true foramen located 1–2 cm superior to the orbit. In either case, these nerves should be identified and preserved during the browlift dissection.

Temporal

The layers of the temporal area include the skin, subcutaneous fat, superficial temporal fascia (also known as the TPF), and deep temporal fascia, which splits and envelops the temporalis muscle. The temporal branch of the facial nerve travels with the temporal artery and vein within the TPF.

◆ Aesthetic Evaluation

The initial consultation should be used to evaluate the orbital complex, the upper third of the face, and the position of the hairline. The patient's wishes and expectations are addressed. Any asymmetries, including those of brow position, should be documented and discussed with the patient. The classic brow is described in its relationship to other structures of the face. The medial limit is positioned through a vertical line originating at the alar–facial groove. The lateral limit is through an oblique line from the alar–facial groove through the lateral canthus of the eye. The medial and lateral brow should lie at the same horizontal position. The ideal female brow possesses an arc over the supraorbital rim. Classically, the maximal peak of the arc is over the lateral limbus of the eye, but many believe that a more natural peak is located above the lateral canthus. The ideal male brow is more horizontal than arced. It should rest along the orbital rim rather then extending above it.

◆ Surgical Approaches and Techniques

Elevation of the brow occurs within a few main approaches. Although many surgeons of the aging face currently favor the endoscopic approach, many techniques are available that can be tailored to the individual patient.

Coronal Approach

This technique employs a coronal incision placed 4–6 cm posterior to the anterior hairline. The incision is beveled parallel to the hair shafts to minimize trauma and alopecia. Dissection proceeds inferiorly to the level of the supraorbital rims. It occurs within a subgaleal, supraperiosteal plane. The supraorbital neurovascular bundles are identified and preserved. The procerus, frontalis, and corrugator supercilii muscles can be scored or incised. This helps to address prominent forehead and glabellar rhytides.

The lateral dissection occurs in a plane between the TPF and deep temporal fascia. This protects the temporal branch of the facial nerve as well as the temporal artery and vein, which are superficial to the dissection. Once sufficient elevation is achieved, 15–25 mm of skin and soft tissue is excised from the length of the incision.

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Candidates for this procedure include patients with a low frontal hairline. This approach should not be used on anyone who is losing their frontal hair or who is expected to do so. Advantages of the coronal approach are camouflaging of scar, ability to perform myoplasty, and excellent exposure. Limitations include elevation of the hairline, possible alopecia and hypesthesia along the incision, and requirement of the most extensive dissection.

The *pretrichial lift* is a modification using a coronal incision anterior or just within the frontal border of the hairline. This approach is favored for those with a high hairline or a long forehead. During a *trichophytic lift,* a coronal incision is placed just posterior the border of the hairline. This has the advantage of superior camouflaging when compared with the pretrichial lift.

Midforehead Approach

A transverse incision is placed within a prominent rhytide of the central forehead. The initial dissection is supragaleal and later deepened to a subgaleal plane as the supraorbital margins are approached. This allows for myoplasty of the procerus, corrugator supercilii and the inferior aspect of the frontalis muscles, while minimizing the risk of hypesthesia to the forehead.

Candidates for this approach are men with thinning hair and prominent forehead rhytides. Advantages of the midforehead approach are that it does not alter the level of the hairline, it allows for myoplasty, and a limited dissection is required. Disadvantages include a potentially unsatisfactory scar, limited lateral elevation, and that it does not address the upper forehead.

Direct Brow Approach

The direct brow approach is a transverse excision of skin and subcutaneous tissue parallel, and immediately superior to each brow. The orbicularis oculi is suspended to the periosteum above. This approach is seldom employed. Its application is limited to those with a functional brow ptosis who place little emphasis on aesthetic results. Advantages are that it is applicable for the very elderly and otherwise poor surgical candidates. It employs a limited dissection, and has excellent control of brow position. The limitations of this approach are an unsatisfactory scar, inability to perform myoplasty, and that it does not address the upper or lateral forehead.

Endoscopic Approach

The most recently described and currently favored approach. It employs four to six 2-cm incisions placed posterior and perpendicular to the hairline. Dissection is carried inferiorly in a subperiosteal plane. Blind dissection may be used until a level 2 cm above the supraorbital rim. A 30-degree endoscope is then employed to visualize the supraorbital neurovascular bundles. Gentle elevation is used to release the periosteum of the forehead from the arcus marginalis of the orbit. The corrugator supercilii, procerus, and frontalis muscles may be scored if necessary. The lateral dissection occurs in a plane between the TPF and deep temporal fascia.

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At the temporal line the conjoined tendon is released with sharp and blunt instrumentation joining the central and lateral pockets. The inferior limit is the zygoma which approximates the level of the lateral canthus. Following sufficient elevation the flap is suspended to the calvarium using a variety of techniques. Though many advocate suture fixation through cortical bone tunnels, miniplates, microscrews, and other methods are also commonly used.

This is the preferred approach for most patients. Advantages of the endoscopic approach are that it is the least invasive, allows for excellent scar camouflaging, ability to perform myoplasty, and it can address the entire upper one third of the face. It is limited by the need for special training, and a lack of long-term results.

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7.4.3 Chemical Peels and Laser Skin Resurfacing

◆ Key Features

- Chemexfoliation is useful for improving the quality and texture of the skin.
- Hypopigmentation is a side effect of peeling, but careful technique may make lines of demarcation less obvious.
- Appropriate measures should be taken to reduce toxic systemic and local side effects.
- Options for ablative facial resurfacing include chemical peeling, dermabrasion, and laser surgery.

For many years, various methods of chemical face peeling have been used to improve the quality and texture of the skin. Numerous exfoliative agents and formulas have been used (salicylic acid, trichloroacetic acid, and β-naphthol), which primarily affect the superficial layers of the skin. Phenol, which is the main component of the Baker-Gordon solution, usually peels to a greater depth and seems to produce more consistent results.

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Although superficial peeling agents remain popular, these do not penetrate the dermis. Laser skin resurfacing has become an important component of rejuvenation surgery. Laser skin resurfacing is used in treating severely photodamaged skin, facial rhytides, dyschromias, and atrophic scars.

◆ Clinical

Patients with wrinkled, sun-damaged, weathered skin are the best candidates for an exfoliative procedure. Chemical peeling will also address fine wrinkles that may be present after other rejuvenation procedures (i.e., blepharoplasty). Pigmented lesions (lentigo, freckles, etc.) and diffuse keratotic lesions (seborrheic and actinic keratosis) may also show improvement after chemexfoliation. Limited improvement may sometimes be seen when peeling is used for superficial acne scars; however, deeper lesions may require other modalities.

◆ Evaluation

Patients must be in appropriate physical and mental condition, must be compliant with posttreatment care, and must have realistic expectations. Female patients with a fair complexion are ideal candidates. Pigment change may be more obvious in dark-skinned patients. Obvious color differences can usually be camouflaged with make-up. Because of the risk of cardiotoxicity, nephrotoxicity and hepatotoxicity, a preoperative workup of ECG, liver function panel, and blood urea nitrogen (BUN)/creatinine assay may be considered.

◆ Chemical Peel Procedure

When performing a deep peel: phenol USP 88% (3 mL), croton oil (3 drops), septisol soap (8 drops), and distilled water (2 mL) or similar solution may be used. More-dilute solutions of phenol may penetrate more deeply. This solution is an emulsion and it is wise to agitate the emulsion constantly during the procedure to ensure that the proper concentration is applied to each region treated.

After adequate preparation (for full facial peels) the patient is usually given light sedation while the local anesthesia is administered. Peels can be full face or regional, and can be used in combination with other rejuvenation surgery. If more than 20 to 25% of the face is to be peeled in the same session, a 15-minute waiting period is recommended between each aesthetic region to ensure the blood level of phenol remains safe.

After sedation and administration of local anesthesia, surface oils are removed with acetone. The removal of the surface oils will allow for a deeper, more evenly distributed peel. If a full face peel is anticipated, the patient should receive generous perioperative IV fluids to increase clearance and decrease serum phenol concentrations. Cardiac monitoring is maintained during the procedure and the postoperative observation

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period. After applying a light coating of solution with a cotton-tipped applicator, a light frost should appear immediately and will usually fade within a few minutes. Feathering of the peel at the margins of the peel area helps avoid obvious demarcation lines at the edge of a treated area. If hair is adjacent to the peeled area, feathering should be performed into hair-bearing areas.

When deep wrinkles extend onto the lips, the application of additional peel may be needed to improve these creases. This is done by dipping the broken end of a wooden cotton-tipped applicator into the peel, then applying a small amount directly onto the crease. When peeling the eyelids, the peel should be applied within 2 to 3 mm of the lid margin. Care should be taken in the canthal regions to dry any tearing that occurs. Tears can draw the peeling fluid into the eye, which could result in ocular damage. Tears may also dilute the peeling formula, which could result in deeper penetration and scarring.

◆ Laser Skin Resurfacing Procedure

Currently, two lasers are in common use for facial skin resurfacing (i.e., photorejuvenation): the carbon dioxide (CO_2) laser, and the erbium:yttrium-aluminum-garnet (Er:YAG) laser. These may also be used in combination. For information on these and other lasers, see Chapter 1.8.6.

Contraindications for laser skin resurfacing include active acne or infection, deep acne pits, and isotretinoin use in the past 2 years.

The use of prophylactic antibiotics has been suggested to decrease secondary bacterial contamination, but is not uniformly used. A nerve block and 1 mg of lorazepam may be given to patients. No intravenous or intramuscular medications are typically necessary. Regional blocks may be used.

Skin lesions and actinic regions are removed by sequential ablative passes. The endpoint is reached when the lesion base has been removed or when a depth to the midreticular dermis has been achieved. Wrinkles are treated by direct lasering into the furrows. Next, the wrinkle shoulders are flattened using the laser as a planing tool. If a regional procedure is to be performed, the aesthetic unit should be blended within its boundaries for optimal camouflage. Following laser skin resurfacing, patients may experience erythema and edema for a week while reepithelialization occurs. Head elevation and ice application and local wound care will help.

◆ Complications

Systemic complications associated with phenol use include hepatotoxicity, nephrotoxicity, and cardiotoxicity. Adequate hydration and judicious time spacing between peeling the aesthetic regions are the keys to keeping blood levels at a tolerable level and avoiding toxicity. Caution should be exercised when using the phenol-containing formulas in patients with abnormal ECGs or with patients with elevated liver or kidney function studies. It may be wise to obtain medical clearance in these patients before proceeding.

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One of the more alarming local complications associated with chemical peeling is wound infection. This can occur in the form of a cutaneous herpes simplex outbreak. The incidence of herpes outbreak can be decreased in carriers with perioperative systemic antiviral therapy and avoidance of peeling in areas of active outbreak. The risk of postherpetic scarring can be decreased with topical antiviral therapy and an increase in oral antiviral dose. Bacterial infections of peeled areas (such as with *Staphylococcus* or *Pseudomonas*) are rare when patients follow the postoperative instructions faithfully. Should they develop, vigorous cleaning and antibiotic therapy is important to prevent any long-term sequelae.

Other local complications of chemical peels include hyperpigmentation and hypertrophic scarring. The risk of hyperpigmentation is increased in patients with high postprocedure estrogen states (such as hormone replacements or pregnancy). Patients should be made aware of this risk and it is recommended holding estrogen-containing medications immediately prior to and for 6 months after a chemical peel; however, this is not an absolute contraindication. Hypertrophic scarring has been known to occur and can be treated with intralesional and topical steroids. Scar revision might be needed in some situations. Milia may be a problem in the first 4 to 6 weeks after chemexfoliation, but usually subside with the proper cleaning regimen. If they persist, an 18-gauge needle can be used to uncap the cyst.

Complications of laser skin resurfacing include hyperpigmentation, erythema, infection, and scarring.

◆ Outcome and Follow-Up

Postoperatively, the patient is allowed to apply cool saline sponges to help reduce discomfort. The patient will usually have some discomfort in the peeled areas for ~6 to 8 hours after the procedure is completed. Patients are instructed to rinse their face in the shower six times daily. After each shower, the patient applies a thick layer of oil-based cream usually for the first 10 to 14 days. Swelling usually begins to subside by the fourth day, whereas the associated erythema may take 8 to 12 weeks, or sometimes longer, for complete resolution. The desquamation usually begins at 24 to 48 hours after surgery, and most is completed by 10 to 14 days postoperatively. Ideally, sunlight should be avoided for 6 months after peeling to reduce any undesirable pigmentary changes. Sunscreen should also be used for added protection.

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7.4.4 Blepharoplasty

◆ Key Features

Blepharoplasty is used for

- Elimination and correction of laxity and redundancy of skin
- Removal of pseudoherniated fat
- Correction of abnormal eyelid position and ectropion

The aging face often reflects changes in the periorbital region, and blepharoplasty can address many changes associated with aging with a restoration of a more youthful appearance. The goals of this surgery are to provide restoration and rejuvenation of the eyelids and may involve resolving the following problems:

1. *Blepharochalasia* occurs in young women with eyelid edema leading to progressive tissue breakdown and may be associated with a relatively uncommon variant of angioneurotic edema.
2. *Dermatochalasis* refers to relaxation of skin and is indicative of the aging process.
3. *Blepharoptosis* occurs when the inferior portion of the upper eyelid margin sits over the iris and is due to levator muscle dysfunction.

◆ Anatomy

Eyelids

The eyelid is a trilamellar structure with thin skin (average thickness 0.13 inches) that is adherent to underlying orbicularis muscle, especially in the region of the tarsal plates, with progressive loosening in the area of the orbital rims.

- The anterior lamella consists of skin and orbicularis muscles.
- The middle lamella consists of orbital septum.
- Posterior lamella
  - Upper eyelid composed of levator aponeurosis, tarsus, Müller's muscle, and the conjunctival lining
  - Lower eyelid composed of lower eyelid inferior retractor muscles, conjunctiva and associated capsulopalpebral fascia, superior portion tarsal plate 4 to 5 mm in width (**Fig. 7.13**)

Orbicularis Muscles

Three distinct bands of striated muscle encircle the orbit and act to close the eyes and facilitate tear flow.

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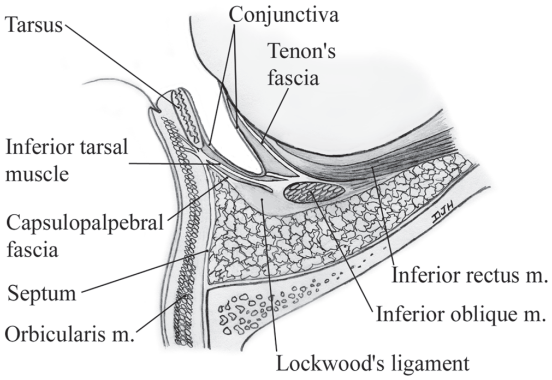

Fig. 7.13 Cross-section of the lower eyelid demonstrating connective tissue expansion of inferior rectus into its terminal insertions. (From Papel ID, ed. Facial Plastic and Reconstructive Surgery. 3rd ed. Stuttgart/New York: Thieme; 2009:273.

- The pretarsal muscle lies directly over tarsal plates.
- The preseptal muscle covers peripheral orbital septum.
- The orbital muscle covers orbital bony margin and blends with frontalis muscle.
- The medial canthal tendon is formed via attachment of superficial heads of pretarsal muscle and attaches to the lacrimal crest.
- The lateral canthal tendon is formed by the upper and lower pretarsal muscles joining laterally and inserts on the orbital tubercle (Whitnall's tubercle).

Orbital Septum

The orbital septum attaches to the bony orbital cavity and is anatomically continuous with the periosteum. It acts to support the orbital contents including orbital fat and serves as a physical barrier to the spread of infection and tumor. The upper orbital septum fuses with the levator aponeurosis, whereas the lower orbital septum fuses with the capsulopalpebral fascia.

Levator Muscle

The levator palpebral superioris muscle is the principal elevator of upper eyelid, originating in the superior orbital apex and inserting in the upper eyelid. As it courses anteriorly, it thins to form the levator aponeurosis.

Capsulopalpebral Fascia

The capsulopalpebral fascia is a fibroelastic structure in the lower eyelid, similar to the levator aponeurosis, which fuses with the orbital septum ~5 mm below the inferior boundary of tarsus. This functions, in combination with inferior palpebral muscles, to retract the conjunctiva and tarsus on downward gaze.

Tarsus

The tarsus is a dense connective tissue providing support for the eyelid that attaches to trochlear fascia medially and fascia of orbital lobe of lacrimal

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gland laterally. Note that the height of the superior tarsal plate is 8 to 9 mm and the height of the inferior tarsal plate is 4 to 5 mm.

Orbital Fat Compartments

- Lower eyelid: Three compartments with lateral and medial compartments separated by a fascial barrier. The medial fat compartment is separated from the central compartment by the inferior oblique muscle.
- Upper eyelid: The lateral compartment consists of the lacrimal gland; the central compartment is separated by the medial compartment and the superior oblique muscle. Contains two fat pads.

◆ Evaluation

- The ophthalmologic examination should include visual acuity, ocular motility, and ocular tension by palpation. In complex cases, an ophthalmologic consultation may be necessary.
- Lid distraction test: A "snap test" test for lower lid laxity is performed by outwardly displacing the lower lid and observing for a normal snap (lid settling quickly back in place with less than 10-mm displacement). If the test is positive, the patient may need a lid-tightening procedure.
- Lid retraction test: This also tests the laxity of the lower eyelid via inferior displacement; if the puncta moves >3 mm, it indicates a lax canthal tendon and the possible need for a tendon plication to avoid ectropion or scleral show.
- Schirmer's test: A strip is folded at the notch and placed in the lower eyelid at the lateral edge of the limbus; more than 10 to 15 mm of moisture on the paper in 5 minutes is normal.
- Bell's phenomenon: Eyes normally rotate upward on attempted closure of the eyelids and this rotation ensures corneal protection if blepharoplasty results in suboptimal lid closure.
- The brow position should be assessed and considered as a separate entity. The brow should be at the orbital rim in males or just above the orbital rim in females and if ptosis is present, a brow repositioning procedure may be needed as well.

◆ Contraindications

Contraindications for a cosmetic blepharoplasty include severe heart or lung disease and psychological factors. Blepharoplasty is cautioned in patients with any of the following:

- Bleeding disorder or recent use of anticoagulants
- Previous facial palsy as it may lead to persistent weakness of the periorbital musculature with inadequate corneal lubrication and recurrent periorbital edema

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- Chronic renal disease and diabetes as they may lead to wound-healing problems
- Thyroid disease including dry eyes syndrome and myxedema; eyelid manifestations may be eliminated by treating hypothyroidism
- Dry eyes may be worsened by blepharoplasty.
- Blindness or vision impairment is possible.

◆ Surgical Goals

Upper Eyelid Blepharoplasty

Goals include addressing fat herniation, skin redundancy, and muscle hypertrophy. Note the asymmetry of the upper eyelids and the position of the superior orbital sulcus: the "tarsal crease" should be <10 mm from lid margin and below the bony margin of the orbital rim.

The incision is marked with the patient in an upright position. The medial point of incision is 4 mm medial and 4 mm cephalad to the medial canthal tendon taking care to avoid the concavity of the medial orbital rim as this leads to webbing. The lower incision is marked along the tarsal crease ~9 to 10 mm from the lid margin. Incisions may be carried laterally 3 to 4 mm past the lateral canthal tendon. An elliptical-shaped skin specimen is incised followed by blunt dissection with scissors and elevation off the orbicularis muscle. An orbicularis strip may then be removed, followed by fat removal if necessary. The skin is closed with a single layer of running 5–0 Prolene suture.

Lower Eyelid Blepharoplasty

Goals include a sharp, well-defined canthus, maintenance of a good lower-eyelid position with an absence of scleral show. Surgical incisions are designed to avoid scar contracture in the vertical dimension, lower eyelid retraction, and ectropion.

Two possible surgical approaches with either subciliary or transconjunctival approaches are described.

Subciliary Approach

The transcutaneous subciliary approach employs an external incision just below the eyelashes (high, immediately subciliary, or relatively lower to preserve the pretarsal orbicularis muscle).

A skin–muscle flap technique is the preferred method when resection of the orbicularis muscle and skin is indicated – the incision is through the skin followed by elevation, and possible fat removal. Fat removal requires the discrete separation of muscle fibers over each fat compartment and incising through the orbital septum. Closure involves lateral and superior elevation with resuspension of the orbicularis muscle.

Fat may be removed from the lateral compartment first, followed by the central and then medial compartments. Fat may be infiltrated with

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additional non–epinephrine containing local anesthetic prior to cautery and removal.

Advantages of the subciliary approach include a relatively avascular plane with a minimal risk of skin penetration, and additional tightening via skin muscle suspension using sutures from the lateral orbicularis muscle to the lateral orbital region.

Limitations of the subciliary approach include a possible increased risk of ectropion. The surgeon may use surgical tape to counter the gravitational effect of postoperative edema, external scar, hematoma, or bruising as a result of orbicularis muscle dissection. There may be scar contracture with rounding of the eyelid.

Transconjunctival Approach

Lower eyelid blepharoplasty is centered on the removal of redundant pseudoherniated fat with incision on the inner aspect of the eyelid. The ideal candidate is 20 to 30 years of age with significant pseudoherniation of fat, minimal skin excess, and minimal orbicularis hypertrophy. This approach is especially helpful to use in patients with tight, inelastic lower eyelids exhibiting scleral show, as this approach transects and releases inferior retractor muscles.

The incision is in the lower eyelid conjunctiva with avoidance of disruption of orbicularis muscle. The preseptal approach involves placing the incision high along the inner eyelid conjunctiva with dissection anterior to orbital septum and under the orbicularis muscle. It is important to protect the cornea while dissecting behind the orbicularis muscle. Exposure of the surgical site and globe protection is facilitated with the use of nonconducting retractors. The dissection is continued downward and forward until all the pseudoherniated fat is exposed. Fat is removed to a depth 1 mm below the surface of the orbital rim with gentle pressure placed on the globe to assess for irregularity and asymmetry. Skin may be resected as necessary using the "pinch" technique or may be combined with chemical peel or laser resurfacing to address superficial fine-line rhytides. Transection of lower lid retractors may have the lower lid margin appear elevated for a few weeks.

Advantages of the transconjunctival approach include avoidance of external scar, and potentially less risk of ectropion. Limitations include lack of addressing skin excess or hypertrophy of the orbicularis muscle.

◆ Surgical Pearls

- Meticulous hemostasis is a must. Lower eyelid blepharoplasty is associated with a higher rate of hematoma formation.
- Avoid braided sutures for closure as they may lead to inflammation and rejection.
- Avoid a retracted or "hollow" look in patients due to excessive fat removal.

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◆ Complications

Acute

- Milia
- Hematomas: More common in the lower lid—fat removal from the lower eyelid may predispose to a retrobulbar hematoma with increased intraocular pressure (IOP >80 means need for urgent management). It may occur up to a few days postoperatively and is treated with a lateral canthotomy and orbital decompression. An ophthalmologic consultation should be sought.
- Blindness

Chronic

- Ectropion
- Lagophthalmos
- Scleral show
- Ptosis
- Epiphora
- Inadequate excision of skin and fat
- Dry eyes

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7.4.5 Otoplasty

◆ Key Features

- An otoplasty entails the changing or reshaping of the auricle, usually applied to congenitally prominent or protruding ears; it should be differentiated from the operations used to repair the congenital condition of microtia.
- Techniques include cartilage scoring, removal, and varied sutures.

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Otoplasty can aesthetically improve the shape, position, or proportion of the auricle (external ear). For the embryology and anatomy of the normal ear, see Chapter 2.0. Figure 2.2 (see page 97) illustrates the anatomical landmarks of the auricle. The auricle is composed of fibroelastic cartilage with overlying skin. The lateral skin is tightly adherent to the cartilage, whereas the medial or postauricular skin has loose connective tissue subcutaneously and thus can be easily separated and peeled from the underlying concha and scapha. The lobule has no cartilage and can have several anatomic configurations and positions.

The abnormal development that results in deformities of the auricle usually originates from the second branchial arch. These abnormalities usually manifest themselves before the end of the first trimester of pregnancy; the frequency of variants is from 3 to 5% of the Western population. Auricular deformities are often inherited in an autosomal dominant fashion.

Also, aging makes the auricle appear larger, in part due to elongation of the lobule.

◆ Evaluation of Aesthetic Deformities of the Auricle

The helix, scapha/antihelix, posterior conchal wall, and conchal floor make up the four planes of the auricle. The angles between these planes and the auricle or scalp determine the degree of protrusion of the ear. The degree of protrusion or malformation is described as a variant from the normal concha–scapha angle. Normal ears have a concha–scapha angle of 75–105 degrees, with 90 degrees most common. The scalp–concha angle also is typically ~90 degrees. This sets the helix approximately parallel to and 2 cm from the scalp. The typical angle from the scalp to the helix is ~30 degrees. An ear is classified as "protruding" when the concha–scapha angle >110 degrees, the angle of the ear to scalp >40 degrees, or the helical rim protrudes >3 cm.

◆ Surgical Techniques

Treatment of abnormally shaped ears commonly addresses two concerns: the lack of development of the antihelical fold and the deep concha cavum, respectively. Treatment of the underdeveloped antihelical fold is divided into two concepts. The Mustarde-type approach utilizes permanent sutures to recreate the antihelical fold. The second approach utilizes scoring incisions, abrading, or filing the cartilage to alter its shape thus reestablishing a fold. A combination of the techniques may be utilized particularly if the scapha is resistant to reshaping via the suture placement. The conchal bowl is likewise treated with two different approaches. One is the Furnas-type approach of suturing posterior conchal cartilage to the mastoid periosteum. The other techniques involve excisions of conchal cartilage usually performed through the postauricular incision. The excisions can be elliptical or crescent-shaped with reapproximation of the cartilage or they can be disk shaped when combined with the conchal setback techniques. The goal is to reduce the height of the posterior conchal wall, thus reducing the prominence of the ear.

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In the majority of patients, the permanent suture technique is utilized with or without scapha weakening. Deep conchal bowls are reduced by elliptical posterior cartilage excisions of 3 to 5 mm and usually followed by a conchal setback procedure. The procedure is done under general anesthesia in children and local anesthesia with sedation in adults. The procedure is performed with vasoconstricting local anesthesia. The incision is placed above the postauricular sulcus in an intermediate location between the mastoid and postauricular skin and the edge of the auricle. When reducing the conchal bowl, perform this first by excision of 3 to 5 mm and reapproximating the edges with 5-0 clear Prolene. The edges are undermined to avoid bunching of the skin, but not so extensively as to create conditions for hematoma formation. The creation of the antihelical fold is done by folding the auricle and noting the location for suture placement necessary to create the fold.

The locations are then marked externally and internally prior to suture placement. The sutures are placed (usually two or three) in horizontal mattress fashion and are tied after all are placed. The knots are triply placed so that the suture can be "cinched" into its ideal position. The ear to scalp angle is measured during this to approximate 2.0 cm so the ear is not bound to the scalp nor will it be inadequately repositioned. After completing the conchal bowl and antihelical fold portions of the procedure, excess skin may be trimmed and additional maneuvers to reposition the lobule by postauricular skin excision are completed if deemed necessary. A cotton ball impregnated with mineral oil or topical ointment is then sutured fixated to the conchal bowl by placing 3–0 nylon on a long Keith needle to secure the bolster. A mastoid dressing is applied. The patient is discharged with a pain medication and a broad spectrum prophylactic antibiotic.

◆ Complications

Complications of otoplasty are rare, but include the possibility of hematoma formation, incision dehiscence, and a permanent suture dehiscence that would require a secondary procedure to replace the suture. Delayed extrusion of permanent suture can occur. If this should develop after 6 months, the suture can be removed without a significant change to the auricular contour; thus it is usually not necessary to replace an extruded suture at this point of the recovery. Wound infection is uncommon, but should be treated aggressively to avoid the long-term complication associated with chondritis. Long-term management of the auricular incision with Scarguard (Red Rock Laboratories, Stamford, CT) or other scar management products can reduce the visibility of the surgical incision (though it is already placed so as to remain inconspicuous). Although not commonly recognized as a complication, under- or overcorrection of the auricular deformity may occur. This should be managed individually to promote patient and family satisfaction with the surgical outcome with consideration of the risk-benefit ratio of further intervention.

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Revision surgery should not be considered until long-term healing has occurred, with the exception of short term suture failure, which should be addressed by suture replacement.

◆ Outcome and Follow-Up

After discharging the patient, a postoperative visit is scheduled 48 hours after the procedure to evaluate the surgical site specifically for a conchal bowl hematoma. The appropriate use of the cotton bolster combined with conservative elevation of the overlying skin of the conchal bowl should make this complication rare. Any hematoma should be promptly evacuated by aspiration or evacuation. It is common at this point to see significant swelling as well as some bruising of the auricle. The dressing is reapplied and left in place for another 48 hours. Upon the patient's revisit, the patient is advised to apply an elastic bandage over the ears at bedtime for one month. Suture removal is done at 7 to 10 days.

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7.4.6 Rhinoplasty

◆ Key Features

- Careful preoperative nasal analysis and clear patient communication are important to success.
- Open or closed approaches can be utilized, depending on the exposure required and preference.
- Meticulous surgical technique is required.

Rhinoplasty is surgery to reshape the nose, the most prominent and central facial feature. Common requests include making a nose smaller, reducing the bridge of the nose, narrowing the nose, making changes to the nasal tip, lifting a droopy nose, revising a previous rhinoplasty, and others. What bothers one person about their nose may not bother another person. Still, most rhinoplasty patients know what they do not like about their nose. In addition to cosmetic concerns, deformities may contribute to problems with nasal function, such as an obstruction from valve collapse, requiring repair. The great majority of patients benefit emotionally and psychologically from rhinoplasty.

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◆ Anatomy

Although the anatomy of the nose has been fundamentally understood for many years, only relatively recently has there been an increased understanding of the long-term effects of surgical changes upon the function and appearance of the nose. A detailed understanding of nasal anatomy is critical for successful rhinoplasty (**Fig. 7.14**).

The accurate assessment of the anatomic variations presented by a patient allows the surgeon to develop a rational and realistic surgical plan. Furthermore, recognizing variant or aberrant anatomy is critical to preventing functional compromise or untoward aesthetic results. It is critical to consider the soft tissue and skin of the nose, which is thickest usually at the nasal tip, thinnest at the rhinion, and thick also at the nasion. The main underlying structures are the paired nasal bones, the upper lateral cartilages, the lower lateral (alar) cartilages, which include a medial crus and a lateral crus, and the nasal septum.

Nasal Analysis

It is critically important that the rhinoplasty surgeon develop skills of facial and nasal analysis. This requires an understanding of the "aesthetic ideal." A nose that is considered "ideal" is one that is harmonious with a patient's other favorable facial features. Our perception of beauty helps define what makes an ideal shape for a female or male nose, so there is also always a bit of an artistic element to this concept. Although the "aesthetic ideal" cannot be completely boiled down to simple lines and numbers alone, guidelines

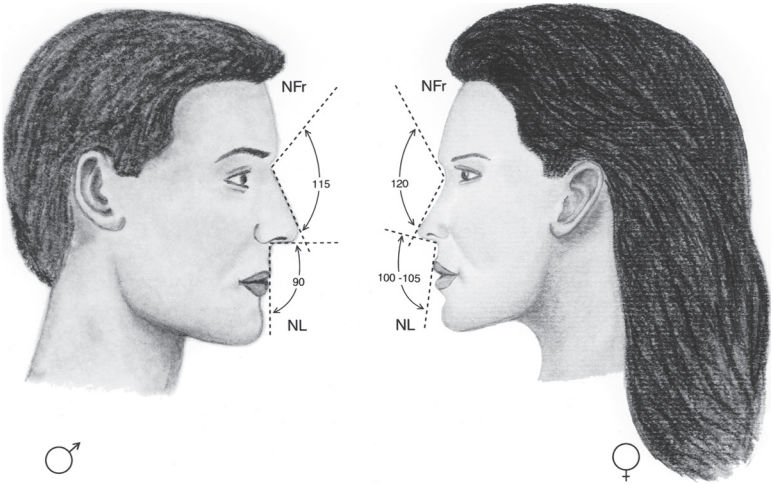

Fig. 7.14 Differences between male and female ideals for the nasofrontal (NFr) and nasolabial (NL) angles. (From Papel ID, ed. Facial Plastic and Reconstructive Surgery, 3rd ed. Stuttgart/New York: Thieme; 2009:122.)

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or proportions do exist that represent the aesthetic ideal. Examples include the nasolabial angle (ideally ~90–105 degrees) and the nasofrontal angle (ideally ~115–120 degrees), and others. Artists have long made studies of beauty and aesthetic proportions and, today, facial plastic surgeons must similarly understand beauty to make changes that can enhance their patients' beauty. Preoperative photographic documentation is important, in frontal, right and left oblique, right and left lateral and basal views. Again, good communication regarding surgical goals is key, bearing in mind these contraindications to rhinoplasty:

- Continued intranasal cocaine use
- Psychiatric or mental instability
- Unrealistic patient expectations
- History of too many previous rhinoplasties

◆ Incisions and Approaches

Incisions are methods of gaining access to the bony and cartilaginous structures of the nose, and include transcartilaginous, intercartilaginous, marginal, and transcolumellar incisions. Approaches provide surgical exposure of the nasal structures including the nasal tip and include cartilage-splitting (transcartilaginous incision), retrograde (intercartilaginous incision with retrograde dissection), delivery approach (intercartilaginous + marginal incisions), and external (transcolumellar and marginal incisions). Based on an analysis of the individual patient's anatomy, appropriate incisions, approaches, and tip-sculpturing techniques may be selected.

An operative algorithm may provide a helpful starting point in selecting the incisions, approaches, and techniques used in nasal surgery. In every case, the patient's anatomy directs the selection of appropriate technique. As the anatomic deformity becomes more abnormal, a graduated, stepwise approach is taken. However, other factors, such as the need for spreader grafts, complex nasal deviation, surgeon preference, and other factors may also appropriately affect the ultimate selection of approach.

The endonasal approaches may be generally preferred for patients requiring conservative profile reduction, conservative tip modification, selected revision rhinoplasty patients, and other situations in which conservative changes are being undertaken. Advantages of less invasive approaches include less dissection, less edema, less "healing." However, less invasive approaches provide by their very nature less exposure, which in some cases may be a disadvantage.

Indications for external rhinoplasty approach generally include asymmetric nasal tip, crooked nose deformity (lower two thirds of nose), saddle nose deformity, cleft-lip nasal deformity, secondary rhinoplasty requiring complex structural grafting, and septal perforation repair. Other indications may include complex nasal tip deformity, middle nasal vault deformity, selected nasal tumors. Some surgeons prefer the open approach for less

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complex nasal tip deformities due to the precision that they feel it offers them, in their hands, compared with the endonasal approach.

Advantages of the external approach include the maximal surgical exposure available, potentially allowing more accurate anatomic diagnosis. The external approach also provides the opportunity for precise tissue manipulation, suturing, and grafting. Disadvantages include the transcolumellar incision, wide field dissection resulting in loss of support, and nasal tip edema.

Regardless of approach, one must be mindful of the need to maintain appropriate structural support. When the approach is disruptive of tip support, countermeasures, such as the placement of a columellar strut, are warranted. When the support to the upper lateral cartilages has been disrupted, spreader grafts may be appropriate.

◆ Surgical Techniques in Rhinoplasty

Techniques in surgery can be seen as tools to help achieve a specific task. These tools on their own are not necessarily enough, but rather the right combination of techniques must be well applied in a particular situation.

There are a variety of techniques available for various rhinoplasty goals, including hump reduction, profile augmentation, osteotomies, tip modification, and others. There are a wide variety of grafts—spreader grafts, tip grafts, columellar struts, alar batten grafts, radix grafts, and others—that may be used for a variety of different clinical situations. Graft material may vary from the septum, to the ear, to the rib. Some surgeons favor alloplastic materials, although many experienced surgeons believe that the nose fulfills few of the criteria needed for the safe use of alloplastic grafts.

Although a surgeon must master several individual techniques for rhinoplasty, he must also have the judgment, skill, and ability to choose the right techniques for each individual situation. A detailed discussion of the different techniques and approaches used in rhinoplasty are beyond the scope of this book.

Septoplasty may be performed at the same time as rhinoplasty (known as septorhinoplasty); see Chapter 7.4.7.

Surgical considerations include the following:

- Informed consent: Should include risks of pain, infection, bleeding, nasal airway obstruction, intranasal scarring, irregularities or asymmetries, failure to meet expectations, need for revision
- Anesthesia: Most surgeons prefer general anesthesia; however, a local with IV sedation can be used.
- Approach: Closed versus open
- Profile surgery: Hump reduction via rasp, powered rasp, or scalpel and Rubin osteotome; dorsal augmentation
- Tip surgery: May address projection, rotation, dome refinement, grafting; tripod concept.
- Osteotomies: May correct the twisted nose, narrow the nose, close open roof deformity; medial oblique osteotomy, low curved lateral osteotomy versus perforated.

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- Septoplasty: Cartilage harvest for grafting if needed. Consider auricular cartilage, the rib.
- Grafts: Middle vault spreader grafts, alar batten, tip shield, columellar strut, other.
- Closure: Precise closure of columellar incision with 5–0 or 6–0 nylon; marginal incisions closed with 4–0 chromic; external tape and splinting. Intranasal packing may be required if septoplasty was performed.

◆ Complications

Infection is uncommon. Alloplastic grafts, however, can become infected and require removal.

◆ Outcome and Follow-Up

Any nasal packing should be removed as soon as possible. Columellar sutures are removed at 5 to 7 days. External splint is also removed at 5 to 7 days. Tissue remodeling is gradual; long-term follow-up is required to monitor final results.

◆ ICD-9 Code

738.0 Acquired deformity of the nose

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7.4.7 Deviated Septum and Septoplasty

◆ Key Features

- Nasal obstruction is the most common complaint.
- Deviated nasal septum is a common cause of nasal obstruction.

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The nasal septum is a central support structure for the nose. When significantly deformed, the septum may cause dysfunction and cosmetic deformity, potentially having an impact on the many functions of the nasal cavity. As many as one-third of people have some nasal obstruction, and up to one-quarter of these patients pursue surgical treatment.

◆ Anatomy

The nasal septum is composed of cartilaginous and bony parts. The bones that make up the septum are the perpendicular plate of the ethmoid bone posterosuperiorly and the vomer, together with the crests of the maxillary and palatine bones, posteroinferiorly. The perpendicular plate of the ethmoid unites superiorly with the cribriform plate and anterosuperiorly with the frontal and nasal bones.

◆ Epidemiology

The patient may provide a history of trauma to the nose; however, often there is no clear history of an inciting event. The initial insult to the nasal septum may have been caused by birth trauma or by microfractures occurring early in life that have led to asymmetric growth of the septal cartilage.

◆ Evaluation

History

The diagnosis of septal deviation begins with taking an adequate history from the patient. This history should include a history of trauma to the nose, nasal airway problems, and prior nasal surgeries; also consider other causes of obstruction such as allergy or infection.

Physical Exam

- Inspect the nasal cavity before and after nasal decongestion.
- Anterior and posterior rhinoscopy is helpful in diagnosing the location, type, and severity of septal deformity.
- Both the anterior and posterior septum should be evaluated. A 0- or 30-degree 4-mm rigid nasal endoscope (e.g., Karl Storz 7200A, Tuttlingen, Germany) will facilitate inspection of the posterior septum. Note the presence of polyps; the severity and extent of septal deviation and bony spurs; assess for septal perforation; and note the presence of mucopus.
- The size and position of the inferior turbinates should be noted during the inspection of the nasal mucosa both before and after the decongestant spray.
- Assess the nasal valve areas for cartilage derangement or dynamic collapse.

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◆ Treatment Options

Indications for septoplasty include nasal obstruction, epistaxis, sinus ostium obstruction, trauma, cosmetic deformity, and surgical access (transseptal–transsphenoidal approach).

There are various techniques, individualized to the specific problem. If performing open rhinoplasty, the columella and marginal incisions enable one to separate the domes and separate the upper lateral cartilages from the septum, permitting open direct exposure to the entire septal cartilage and bone. Another approach involves a hemitransfixion incision thru the lining of the caudal septum, with elevation of mucoperichondrium and mucoperiosteum. The caudal septum can be exposed followed by elevation of the contralateral mucoperichondrial flap. Deviated cartilage or bone is removed and replaced. It is important to reconstruct the septum by replacing tissue for support. Tip and septal support must be maintained. Mucosal flaps are replaced and hematoma is prevented by using a mattress absorbable suture and/or silastic (Doyle) septal splints. Splints are removed in 4 to 6 days.

For more limited deformities, an incision can be made internally in the septal mucosa to expose cartilaginous or bony deformity. The flap is elevated, cartilage is incised, and a contralateral flap is dissected. The deformity is then removed; cartilage is morcellized and replaced. If endoscopic sinus surgery is being performed, the limited septoplasty can be done using endoscopic instruments. A suction Freer elevator is very useful in this technique. Powered instrumentation using a septoplasty bur can be useful for the reduction of bony spurs.

Regardless of approach, care must be taken to minimize mucosal tears. Recognized tears should be repaired with absorbable suture. Replacement of cartilage is also important to help minimize the chance of development of septal perforation. Dissection posteriorly and superiorly should be done carefully to avoid transmission of force to the ethmoid roof to prevent CSF leakage. If turbinate surgery is done along with septoplasty, the use of silastic splints will help prevent intranasal synechiae.

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7.4.8 Liposuction of the Face and Neck

◆ Key Features

- Liposuction of the face and neck involves the use of negative pressure to remove subcutaneous fat.
- The goal of liposuction is improved facial contouring.
- Small incisional punctures in the skin are used to gain access to the subcutaneous plane and then the cannula is inserted back and forth in different directions (usually radially) and some of the fat is extracted.

The use of negative pressure to remove subcutaneous fat has for several years been the most popular cosmetic surgical procedure in the United States. This has evolved over the past couple of decades, and various hollow cannulas combined with aspiration machines had been used in many parts of the body to remove unwanted subcutaneous fat. The primary purpose is to help "sculpt" the neck, jowl, and sometimes the face. Liposuction may be performed under local anesthesia or it may be added to another surgery as an adjunct. The purpose is never to remove all the fat in an area; rather to thin and partially remove. The fat that is aspirated will be removed permanently, additional fat will be traumatized, and later some of this will necrose and shrink.

◆ Indications

Patients with diet-resistant cervicofacial fat deposits are candidates for liposuction (as long as they have realistic expectations of the outcome).

◆ Contraindications

- Absolute: None
- Relative: Prior trauma or surgery in the area. Scar tissue and fibrosis, heavy smokers, and those who have dermatologic, collagen, vascular, or other systemic diseases, psychiatric instability or those who have unrealistic expectations

◆ Procedure

It is important to leave a layer of normal subcutaneous fat, which acts somewhat as a "carpet pad" between the skin "carpet" and the underlying deeper anatomy. When fat is suctioned as part of a rhytidectomy, the technique might be "open." With a small puncture incision, then the technique is "closed."

Over the past few years the size of cannulas has decreased. Some early cannulas were 10 to 12 mm in diameter or even larger. Although aspiration machines may generate negative pressure close to 1 atmosphere (960 mm Hg), handheld syringes are thought to generate pressures of ~600 mm

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and empirically work well. The negative pressure produced by a handheld syringe combined with a small 2-mm cannula attached to a 3-cc syringe (or 10 cc if preferred) is sufficient for removal of most subcutaneous fat in the submental plane. Additional negative pressure can be utilized for more aggressive or speedier fat removal if necessary. Skin elasticity must be sufficient to redrape in a superior direction (unless it is supported with an additional procedure, such as a facelift). Simply removing fat from a face with poor skin turgor will result in worsening of the appearance (submental "turkey gobbler" deformity, for example). Younger patients (20s–30s) are ideal. Patients in their 40s sometimes retain sufficient elasticity. Patients in their 50s will be risky and older patients will very rarely be good candidates for liposuction alone.

◆ Complications

- Postoperative edema
- Hematoma
- Infection
- Scarring
- Poor skin draping
- Necrosis of the skin flap
- Unsatisfactory final contour
- Nerve injury resulting in paresis or paresthesias

◆ Outcome and Follow-Up

Following liposuction, a light compression dressing can be worn by the patient for a week or so, and worn at night for perhaps 2 weeks to facilitate redraping of skin. There is some mild discomfort once local anesthesia wears off, and may last a few days, but pain is not usually great. In fair-skinned patients, those who bruise easily, or those taking aspirin or nonsteroidal antiinflammatory drugs (NSAIDs), there often will be some bruising that may last a week or so.

Healing is gradual and effects may not be fully appreciated for several months as the remaining fat shrinks, edema resolves, and the skin continues to contract. In properly selected patients, judicious liposuction can be a valuable adjunct to improving the contoured appearance of the face and neck. It is not indicated for facial weight loss and should be used conservatively. It is usually easy to remove additional fat later, if need be. It is more difficult to replace it.

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7.4.9 Hair Restoration

◆ Key Features

- Androgenetic alopecia or male pattern alopecia (MPA) is due to 5-α-dihydrotestosterone (DHT) binding to susceptible hair follicles. The mechanism of female pattern alopecia (FPA) is not well delineated.
- Minoxidil and finasteride are the only proven medical treatments for alopecia.
- Modern hair restoration focuses less on scalp flap/reduction surgery and more on rearranging existing hair through follicular unit hair transplant techniques.
- Following transplantation, hair follicle typically enters telogen phase and does not grow for the first 3 to 4 months.

Alopecia affects 60 to 80% of men and is due to DHT binding to susceptible hair follicles. Hair loss can be staged according to the Norwood scale (I–VII), and treatment involves topical minoxidil, oral finasteride, hair pieces, and surgery. Today, scalp flaps (e.g., Juri, bitemporal, occipitoparietal) are less commonly performed, and follicular unit hair transplant techniques are the mainstay of surgical therapy.

◆ Epidemiology

By age 60, androgenetic alopecia affects 60 to 80% of males and ~10% of women. Whites are more affected than Blacks who are more affected than Asians. Transmission is thought to be polygenic, sex-linked, autosomal dominant with variable penetrance.

◆ Anatomy

The hair follicle is divided into three parts: the infundibulum, isthmus, and inferior portion. The infundibulum is the most superficial part and joins the isthmus where the duct of the associated sebaceous gland enters the follicle. The isthmus is separated from the inferior portion by the insertion of the arrector pili muscle. Hair grows naturally in groupings or units of 1, 2, 3, or 4 hairs associated with a sebaceous gland and an arrector pili muscle. This is referred to as a follicular unit.

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Hair growth occurs in cycles:

- Anagen (lasts 2–6 years): Hair spends 90% of time in this phase. The inferior portion of the hair elongates and forms the matrix containing bulb and papilla. Hair is growing.
- Catagen (lasts 2–3 weeks): The inferior portion of the hair ascends to the level of the arrector pili muscle. No hair growth occurs.
- Telogen (lasts 3 months): The inferior portion of the hair is absent. No hair growth occurs. The hair can be shed, but often remains until anagen phase begins again.

◆ Clinical

Signs and Symptoms

Progressive hair loss, usually with family history, initially affects the frontotemporal region and the crown in MPA. FPA is often diffuse.

Differential Diagnosis

Other rare causes of alopecia include alopecia areata (autoimmune), telogen effluvium (possibly stress related), secondary syphilis, trichotillomania, traction alopecia, and hair breakage due to aggressive grooming or chemical treatments.

◆ Evaluation

Physical Exam

The Norwood scale quantifies MPA from class I to class VII: class I = no hair loss, class VII = only occipital hair remains. The Ludwig scale grades FPA from grade I to grade III: grade I = crown thinning, grade III = severe diffuse thinning. Hair density at occipital donor site ideally should be greater than 8 hairs per 4 mm diameter if the patient is to undergo transplant. Darker skin with curly, coarse dark hair or lighter skin with curly, coarse salt and pepper hair are the best transplant combinations. Fine, dark hair on light skin is the worst combination.

Imaging

Pre- and posttreatment imaging should include a frontal view, brow, and anterior hairline close-up view, oblique views, oblique close-ups, posterior view, and anterior and posterior bird's eye views.

◆ Treatment Options

Medical

Minoxidil helps about one-third of men and women. Finasteride helps about two-thirds of men.

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Relevant Pharmacology

Minoxidil (Rogaine): Opens K+ channels and directly vasodilates peripheral vessels. Men: Use 5% topical application, 1 cc to scalp BID. Women: Use 2% formula due to risk of facial hair growth.

Finasteride (Propecia): Inhibits type II 5-α-reductase, thus preventing conversion of testosterone to 5-α-dihydrotestosterone. Male dosage = 1 mg/day. Women cannot use finasteride because of the risk of birth defects.

Surgery

Scalp reduction (removal of nonhair-bearing scalp) and scalp flaps for hair rearrangement are falling out of favor except in selected cases. The mainstay of therapy is hair transplantation with (1) harvest of occipital hair, (2) primary closure of the donor site and microscopic dissection of donor hair into follicular unit grafts, and (3) placement of 800 to 1600 follicular unit grafts (1–4 hairs per graft) via stab incisions. Hair must be placed at the appropriate site and angle to the skin to recreate a natural look. Only micrografts (1–2 hairs per graft) should be placed along the anterior hairline, which should be ~7 to 8 cm above the brows.

◆ Complications

Complications for hair transplantation are rare (less than 5%) and include cobblestoning, folliculitis, graft failure, and donor site scarring. Treatment for folliculitis is improved scalp hygiene and antibiotics (e.g., cephalexin, tetracycline).

◆ Outcome and Follow-Up

For hair transplantation, patients can shower postoperative day 1 and wash hair postoperative day 2. Transplanted hairs will fall out in 2 to 3 weeks and begin new growth at 3 to 4 months postprocedure.

Hair transplantation is reliable procedure with a consistent outcome. Typically, 3 to 5 sessions (at least 6 months apart), each with placement of 800 to 1600 grafts are necessary for complete treatment. Patient must continue minoxidil and finasteride to see continued results from these medicines.

Further Reading

Hamilton MM. Hair replacement surgery. In: Truswell WH, ed. Surgical Facial Rejuvenation: A Roadmap to Safe and Reliable Outcomes. Stuttgart/New York: Thieme; 2009:160–168

Rousso DE, Sule S, Stough D, Whitworth JM. Hair replacement techniques. In: Papel ID, ed. Facial Plastic and Reconstructive Surgery. 3rd ed. Stuttgart/New York: Thieme; 2009:409–420

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Appendix A Basic Procedures and Methods of Investigation

◆ A1 Bronchoscopy

Two methods of bronchoscopy are available – rigid and flexible.

Rigid Bronchoscopy

Historically, rigid bronchoscopy is the older method. Rigid bronchoscopes are tubes of different calibers with a proximal cold light source. Rigid bronchoscopy is performed under general anesthesia. The bronchoscope has direct connection to the anesthetic and respiratory apparatus, so it is called the *respiratory bronchoscope*. A rigid bronchoscope can be combined with other instrumentation, including aspiration lavage, cytologic diagnosis, and swabs for culture. Rigid bronchoscope may be used in conjunction with a laser.

Indications

Rigid bronchoscopy as a therapeutic measure:

- Emergency bronchoscopy done to bypass sudden obstructive respiratory insufficiency
- Removal of tracheal or bronchial foreign body; arrest of bleeding of the trachea or bronchi

Rigid bronchoscopy as a diagnostic procedure:

- To treat tracheal or bronchial stenosis
- To biopsy a tracheal tumor
- To investigate hemoptysis
- To assess upper airway trauma

Advantages

- Versatile procedure
- Can be used on a bleeding patient
- Extraction of foreign body

Disadvantages

- Technically more difficult with abnormal cervical anatomy
- Limitations on neck extension
- Must be done under general anesthetic

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Flexible Bronchoscopy

Flexible bronchoscopes usually have a diameter of between 4 to 5 mm and are thinner than rigid bronchoscopes. Their distal end is controlled externally so they can be introduced into the low bronchi or segmental bronchi. The instrument may be introduced via the nose, the mouth, or a tracheotomy. Flexible bronchoscopy may be performed under local or general anesthetic with the patient sitting or lying. When using general anesthetic, at intubation the bronchoscope may be introduced through the endotracheal tube.

Indications

- Bronchial or upper airway tumors
- Hemoptysis
- Undiagnosed disorders such as unresolved pneumonia
- Middle lobe syndrome

Advantages

- It can be introduced far into the periphery as far as the fifth generation bronchi; therefore, it complements the rigid endoscope.
- It can also be done under local anesthetic with an awake patient.

Disadvantages

- It has a relatively narrow working radius; therefore, it cannot be used for large foreign bodies or in the presence of profuse bleeding.

Complications

Complications of rigid and flexible bronchoscopy include:

- Damage to vocal folds
- Perforation of tracheobronchial tree
- Pneumothorax
- Laryngospasm
- Death

◆ A2 Esophagoscopy

Esophagoscopy can be performed with either a rigid or flexible esophagoscope. The rigid esophagoscope is a rigid tube that is usually used under general anesthesia. It has a high-powered cold light source at the proximal or distal end. Extraction, excision, and coagulation instruments can be used in conjunction with the rigid esophagoscope. Lasers may also be used. Flexible esophagoscopy has a narrow caliber, is suitable for foreign body extraction, and can be used in conjunction with air insufflation and be attached to air insufflation and suction. It also typically provides good photographic documentation for permanent record keeping. Percutaneous endoscopic

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gastroscopy (PEG) feeding tube placement may also be done via flexible esophagoscopy. Seven PEG kits are available.

Indications

Rigid esophagoscopy as a therapeutic measure:

- Removal of foreign bodies
- Removal of polyps and fibromas
- Division of hypopharyngeal rings and diverticulum
- Dilation stenosis
- Injection of esophageal varices

Rigid esophagoscopy as a diagnostic procedure:

- To diagnose diseases of the esophagus
- To diagnose tumors of the hypopharynx and esophagus
- To evaluate dysphagia

Flexible esophagoscopy as a diagnostic procedure:

- In cases where rigid esophagoscopy is contraindicated or impossible due to an ability to flex or extend the neck because of cervical spine disease, panendoscopy is indicated.
- General indications are otherwise similar to rigid esophagoscopy.

Advantages

Rigid esophagoscopy:

- Versatility and superior ability to remove large foreign bodies from the esophagus. It is very efficient for both diagnostic and therapeutic usage.

Flexible esophagoscopy:

- Simultaneous panendoscopy of the stomach and duodenum may be performed.
- Good screening instrument
- Less traumatic for the patient

Complications

- Esophageal perforation
- False passage
- Mediastinitis
- Pneumomediastinum
- Oral and dental injury, especially with use of rigid instrumentation
- Death

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◆ A3 Rigid Direct Microscopic Laryngoscopy with or without Biopsy

This is used for larynx and hypopharynx evaluation and biopsy.

Indications

- Suspected or known malignancy
- Treatment of cancer through endoscopic resection
- Together with esophageal endoscopy, bronchoscopy (collectively, panendoscopy)
- Evaluation and treatment of hoarseness
- Endotracheal intubation for difficult airway

Contraindications

- Unstable cervical spine
- Unable to obtain exposure of the larynx

Laryngoscope Types

- Dedo laryngoscope–widely used for laryngeal biopsy procedures including working diameter
- Holinger anterior commissure scope–used for better exposure anteriorly
- Lindholm laryngoscope
- Weerda laryngoscope–bivalve design; useful for endoscopic management of Zenker diverticulum
- Jackson "sliding" laryngoscope

Steps

1. Patients are placed in the supine position with the head extended and with the eyes protected.
2. General anesthesia is used.
3. A tooth or mouth guard is in place.
4. A rigid laryngoscope is placed through the mouth and with the use of an operating microscope or fiberoptic telescope the entire throat and affected area is magnified and evaluated.
5. Suspension laryngoscopy—suspending the laryngoscope allows the surgeon to use both hands for procedures within the larynx
6. Lasers, a microdébrider, a monopolar cautery, and cold microdissection or biopsy instrument tools can be introduced through the laryngoscope.

Complications

- Loss of airway and obstruction
- Damage to teeth, mouth, and gums
- Numb tongue, altered taste, temporomandibular joint disorders

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- Hoarseness
- Perforation
- Airway fire; if using laser or cautery

◆ A4 Tonsillectomy

Indications

Absolute:

- Enlarged tonsils with an upper airway obstruction
- Severe dysphagia
- Sleep disorders thought to be related to obstructive tonsil hypertrophy
- Peritonsillar abscess unresponsive to medical management
- Tonsillitis resulting in febrile convulsions

Relative:

- Three or more tonsil infections per year despite adequate medical therapy
- Persistent foul taste or breath
- Chronic tonsillitis in a streptococcal carrier
- Unilateral tonsil hypertrophy presumed to be neoplastic

Contraindications

- Bleeding diathesis, unless managed with appropriated perioperative medical therapy
- Poor anesthetic risk or uncontrolled medical illness
- Acute infection

Steps

1. Shoulder roll
2. General anesthesia and intubation in most cases
3. Insert a mouth prop, open, and suspend
4. Apply a tonsil clamp to the tonsil to allow for medial traction during dissection
5. Dissection of tonsil and removal, taking care to fully preserve the posterior pillar and stay in the capsular plane

Dissection Instruments

- Cold steel instruments
- Monopolar cautery
- Bipolar cautery with or without a microscope
- Radiofrequency ablation or coblation
- Harmonic scalpel
- Microdébrider

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Complications

- Hemorrhage
- Pain
- Dehydration
- Weight loss
- Fever
- Postoperative airway obstruction
- Pulmonary edema
- Local trauma to oral tissues
- Tonsillar remnants regrowth
- Vocal changes
- Temporomandibular joint dysfunction
- Death

◆ A5 Adenoidectomy

Indications

- Adenoid enlargement with nasal airway obstruction
- Obstructive sleep apnea symptoms
- Chronic mouth breathing
- Recurrent or persistent otitis media in children ≥3 years old
- Recurrent and/or chronic sinusitis

Contraindications

- Severe bleeding disorder (relative)
- True cleft palate
- Muscle weakness or hypotonia (relative)
- Atlantoaxial joint laxity (relative)

Steps

1. Use a mouth appliance to open the mouth and retract the palate.
2. A mirror can be used to see the adenoids because they are behind the nasal cavity.
3. The adenoid is removed through the mouth.

Dissection Instruments

- Adenoid curette
- Adenoid punch
- Electrocautery with a suction Bovie
- Microdebrider

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Complications

- Hemorrhage
- Velopharyngeal insufficiency
- Torticollis
- Nasopharyngeal stenosis
- Atlantoaxial subluxation from infection (Grisel syndrome)
- Eustachian tube injury
- Death

◆ A6 Open Surgical Tracheotomy

Indications

- Prolonged intubation with mechanical intubation
- To bypass upper airway obstruction
- To provide pulmonary toilet
- Prophylaxis for anticipated need for ventilator support
- Sleep apnea

Steps

1. Place patient supine, with neck extended on a shoulder roll if possible.
2. Palpate the neck landmarks.
3. Infiltrate lidocaine epinephrine.
4. Incise the skin between the cricoid and sternal notch (horizontal or vertical).
5. Separate strap muscles and retract laterally.
6. Divide or retract thyroid isthmus.
7. Verify hemostasis.
8. Alert the anesthesiologist of impending airway entry.
9. Tracheal opening (window, trap door flap, slit).
10. Have anesthesiologist withdraw endotracheal tube under direct visualization.
11. Insert the tracheotomy tube into the trachea.
12. Connect circuit and inflate balloon.
13. Verify end tidal CO_2.
14. Secure the tracheotomy tube to the skin with four sutures and a tracheotomy collar.

Complications

- Hemorrhage
- Pneumothorax
- False Passage

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- Obstruction or decannulation
- Infection
- Tracheoesophageal fistula
- Tracheocutaneous fistula
- Tracheoinnominate artery fistula
- Death

◆ A7 Cricothyroidotomy

Steps

- Palpate the cricoid in the midline with a neck extension; the thyroid cartilage is stabilized superiorly with a nondominant hand. Move your index finger down until you palpate the cricoid cartilage.
- The space between the thyroid and cricoid cartilages is the cricothyroid membrane. Use the scalpel to make a 2.5-cm vertical incision through the skin and subcutaneous tissue.
- Use the curved hemostat to make a blunt dissection in the subcutaneous tissue.
- Next, use the scalpel to make a horizontal incision through the cricothyroid membrane.
- You may feel a pop as the trachea is entered.
- Extend the incision laterally, turn the blade, and extend it in the opposite direction.
- Once the trachea has been entered, make sure the blade stays within the incision, so that communication with the trachea is never lost.
- Insert a tracheal hook, and pull superiorly on the upper portion of the incision, elevating the larynx. Once the tracheal hook is in place, you may remove the blade.
- Insert a Trousseau dilator and open the membrane vertically, then insert the tracheotomy tube.

Because the cricothyroid membrane is situated between two rigid bodies (the thyroid cartilage above and the cricoid cartilage below), there is little flexibility in the size of the opening that can be made in the membrane. A tracheotomy tube or endotracheal tube with an internal diameter of 6 mm should be used. A tube with an internal diameter >7 mm would be difficult to insert into the cricothyroid membrane. Cricothyroidotomy is a *temporizing technique* only, and in these cases the patient should have a formal tracheotomy performed as soon he or she is stabilized.

Complications

- Esophageal perforation
- Subcutaneous emphysema
- Hemorrhage
- False route
- Injury to larynx

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Appendix B The Cranial Nerves

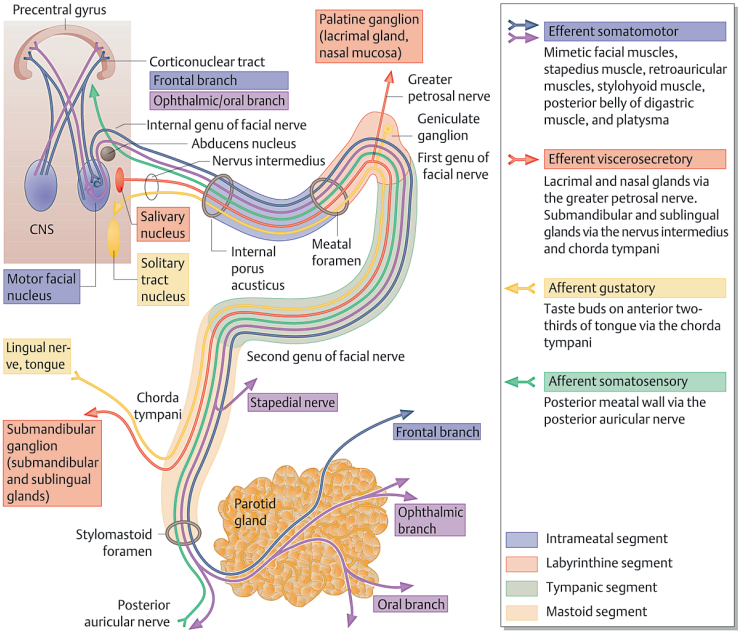

Fig. B1 Course, segments, and functions of the facial nerve (cranial nerve VII). (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:291.)

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Cranial Nerve	Name	Function				Comments
I	Olfactory	Special sensory	Smell			
II	Optic	Special sensory	Sight			
III	Oculomotor	Motor	Skeletal motor to 4 extrinsic eye muscles and levator palpebrae superioris muscle	Visceral motor parasympathetic Preganglionic fibers synapse in ciliary ganglion Innervate ciliary muscle and sphincter pupillae muscle in the eye		
IV	Trochlear	Motor	Innervates superior oblique muscle (an extrinsic eye muscle)			
V	Trigeminal	Somatic sensory – skin of the face	Visceral sensory – mucous membranes of the nose and mouth	Skeletal motor – mastication (chewing) muscles		Three divisions: Ophthalmic (V_1) Maxillary (V_2) Mandibular (V_3)
VI	Abducens	Motor	Innervates lateral rectus muscle (an extrinsic eye muscle)			
VII	Facial	Somatic sensory – skin of external ear (small contribution)	Visceral motor (parasympathetic) – salivary and lacrimal glands	Visceral sensory – taste buds on anterior ⅔ of tongue	Skeletal motor – muscles of facial expression	Nervus intermedius carries general sensory fibers, taste fibers, and visceral motor (parasympathetic) fibers

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Cranial Nerve	Name	Function				Comments
VIII	Vestibulocochlear	Special sensory	Hearing and balance	Vestibular nerve – receptors in semicircular ducts, utricle, and saccule		Cochlear nerve: Receptors in spiral organ (organ of Corti)
IX	Glossopharyngeal	Visceral sensory – mucous membranes of middle ear, posterior tongue, and throat	Visceral sensory – taste posterior ⅓ of tongue	Visceral motor (parasympathetic) – parotid gland	Skeletal motor – stylopharyngeus muscle	
X	Vagus	Somatic sensory – skin of the external ear and eardrum	Visceral sensory – mucous membranes of lower throat and larynx	Visceral motor (parasympathetic) – cardiac and smooth muscle in organs of thorax and abdomen	Skeletal motor – palate, pharynx, & larynx muscles	
XI	Spinal accessory	Motor	Innervates all muscles of the tongue – both intrinsic and extrinsic – except palatoglossus muscle			C1 motor fibers "hitch a ride" on CN XII
XII	Hypoglossal	Skeletal motor	Innervates all muscles of the tongue – both intrinsic and extrinsic palatoglossus muscle			

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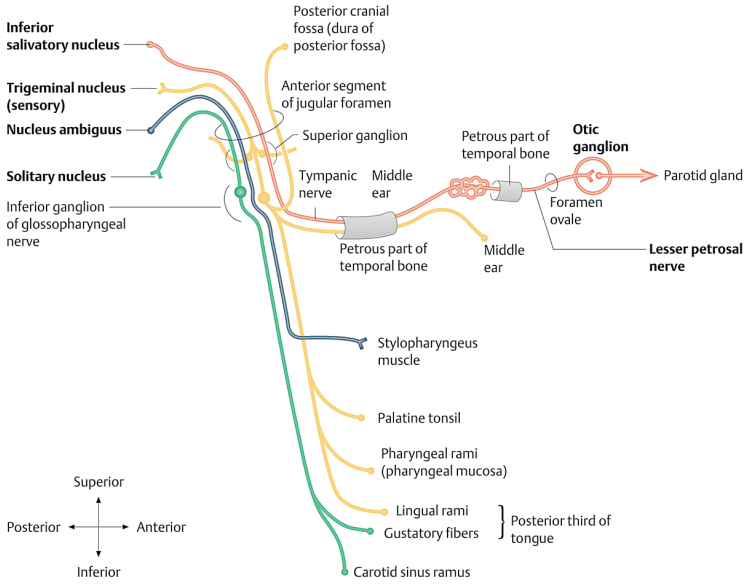

Fig. B2 Course of the glossopharyngeal nerve (cranial nerve IX). (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:315.)

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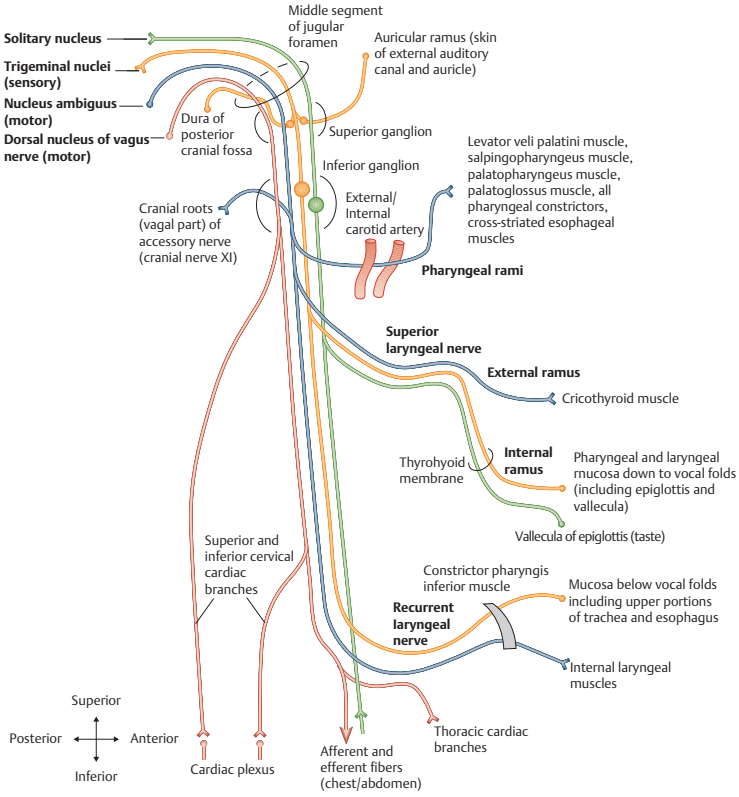

Fig. B3 Course of the vagus nerve (cranial nerve X). (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:316.)

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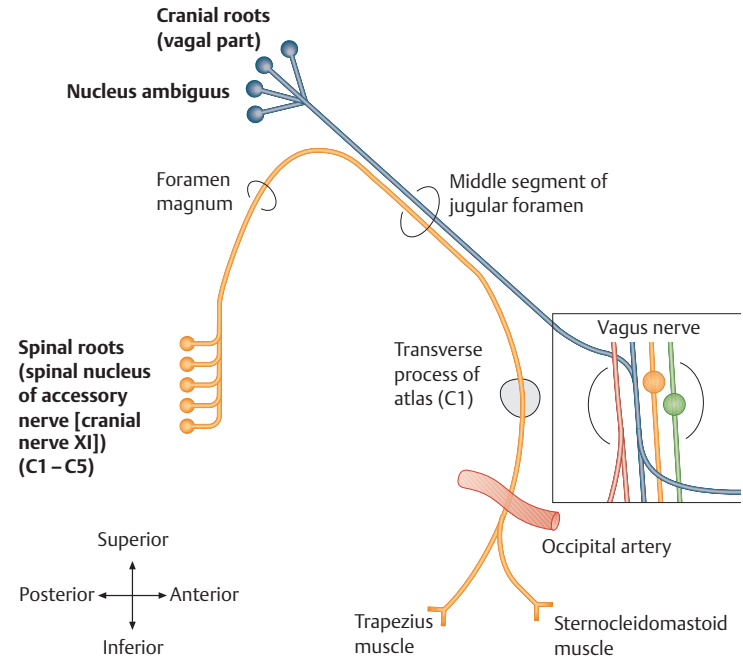

Fig. B4 Course of the accessory nerve (cranial nerve XI). (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:317.)

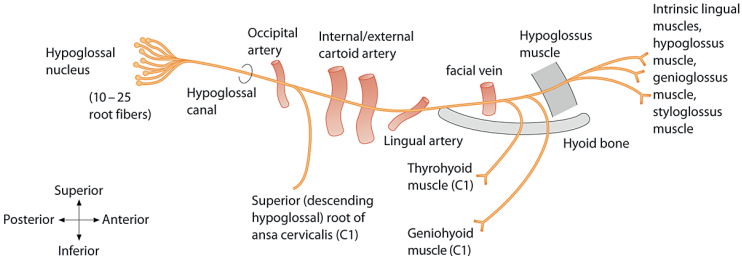

Fig. B5 Course of the hypoglossal nerve (cranial nerve XII). (From Probst R, Grevers G, Iro H. Basic Otorhinolaryngology: A Step-by-Step Learning Guide. Stuttgart/New York: Thieme; 2006:317.)

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Appendix C Otolaryngologic Emergencies Requiring Immediate Diagnostic and Therapeutic Intervention

Emergency	See page(s)
Airway fire	47
Airway obstruction	16–20
Airway obstruction, pediatric	507–510
Anesthetic emergency	47–48
Aspiration	311–316
Blood transfusion complication	52–55
Carotid artery blowout	63–64
Caustic ingestion	278–280
Cerebrospinal fluid rhinorrhea	225–228
Choanal atresia	584–586
Confusion (mental status change)	59–61
Delirium tremens	61
Ear foreign body	114–116
Ear trauma	104–108
Epistaxis	229–233
Facial nerve paralysis	109–113, 620–624
Fever, high	58–59
Frontal sinus fracture	605–608
Fungal rhinosinusitis, acute invasive	215–218
Hearing loss, sudden sensorineural	102–104
Hematoma following thyroid surgery	62
Laryngeal fracture	275–277
Laryngeal infection	280–284
Laryngomalacia	511–513
Ludwig angina	331–333
Lymph nodes, enlarged or inflamed	565–569
Malignant hyperthermia	47–48
Mandible fracture	614–619

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Emergency	See page(s)
Midface (LeFort) fracture	609–613
Nasal fracture	597–599
Naso-orbito-ethmoid fracture	599–601
Neck infection, deep	333–336
Neck trauma	337–341
Necrotizing soft tissue infection	328–330
Orbital fracture	602–604
Otitis externa, malignant	145–148
Otitis media complication	127–135
Pulmonary edema, acute postobstructive	60
Pulmonary embolism	60
Sinusitis complication, intracranial	222–224
Sinusitis complication, orbital	219–221
Stridor	272–275
Subglottic stenosis	529–533
Temporal bone trauma	104–108
Thyroid storm	478–479
Vestibular neuritis	186-189
Vocal fold motion impairment	297–300
Zygomaticomaxillary complex fracture	602–604

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Index

Note: Page numbers followed by *f* and *t* indicate figures and tables, respectively.

A

ABCDE, of melanoma, 417
ABCs, of neck examination, 338
Abducens nerve (cranial nerve VI), 327, 540
  function, 690
ABI. *See* Auditory brainstem implant (ABI)
ABR. *See* Auditory brainstem response (ABR)
Abscess
  Bezold
    etiology, 128
    imaging, 128
    physical examination of patient with, 128
    signs and symptoms, 128
    treatment, 128
  brain, 220
    clinical course, 131
    imaging, 132
    otitis media–related, 131–132
    parenchymal
      signs and symptoms, 222
      sinusitis and, 222
    pathology, 132
    signs and symptoms, 131
    treatment, 132
  carotid space, drainage, 336*t*
  epidural, 133
    imaging, 133
    signs and symptoms, 133, 222
    sinusitis and, 222
    treatment, 133
  frontal lobe, signs and symptoms, 222
  intracranial
    deep neck infection and, 335
    ICD-9 code, 224
    treatment, 224
  lung, deep neck infection and, 335
  masticator space, drainage, 336*t*
  neck, in children, 508, 567–568
  orbital, 219*t*
    treatment, 221
  parapharyngeal
    drainage, 336*t*
    ICD-9 code, 336
  parotid, drainage, 336*t*
  peritonsillar, 329
    drainage, 336*t*
    ICD-9 code, 336
  pharyngeal, 329
    in children, 508
  prevertebral, drainage, 336*t*
  retropharyngeal
    drainage, 336*t*
    ICD-9 code, 336
  salivary gland, ICD-9 code, 447
  subdural
    signs and symptoms, 222
    sinusitis and, 222
  submandibular, drainage, 336*t*
  subperiosteal, 219, 219*t*, 220
    etiology, 127
    imaging, 127
    otitis media and, 127
    physical examination of patient with, 127
    signs and symptoms, 127
    treatment, 127
  thyroid, 481
  wound, treatment, 59
ACE. *See* Angiotensin-converting enzyme (ACE)
Acetaminophen, preoperative, 10*t*
Acetazolamide, for otitic hydrocephalus, 135
Acetic acid
  for chronic otitis media, 122, 123, 124*t*
  for otitis externa, 143*t*, 144
Acetic acid/hydrocortisone, for otitis externa, 143*t*
Achondroplasia, 538
Acid ingestion, 278–280
Acid-reflux disorders, 317–319
  See also Gastroesophageal reflux disease (GERD), and Laryngopharyngeal reflux (LPR)
  after tracheoesophageal fistula/esophageal atresia repair, 524
Acinic cell carcinoma, salivary gland, 451

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Ackerman tumor, 372
Acoustic neuroma. *See* Vestibular schwannoma
Acoustic reflex test, 153–154
Acoustic rhinometry, 214*t*
Acquired immunodeficiency syndrome (AIDS). *See* Human immunodeficiency virus (HIV), infection
ACT. *See* Activated clotting time (ACT)
Actinomycosis, laryngitis caused by, 282–283
Activated clotting time (ACT), 56
Acupuncture, 91
  for allergic rhinitis, 91
  for asthma, 91
  for tinnitus, 90
Acute invasive fungal rhinosinusitis, 215–218
  clinical course, 215
  clinical features, 215–216
  diagnosis, 216
    algorithm for, 217*f*
  differential diagnosis, 216
  epidemiology, 215
  ICD-9 codes, 218
  imaging in, 216
  microbiology, 215
  outcomes with, 218
  pathology, 218
  physical findings in, 216
  prognosis for, 218
  risk factors for, 215
    laboratory evaluation, 217
  signs and symptoms, 215–216
  treatment, 218
    algorithm for, 217*f*
Acute otitis media (AOM), 116–120
  clinical features, 116, 117
  complications, 117, 118, 120, 127–135
    epidemiology, 127
    extratemporal, 127–128
    intracranial, 131–135
    intratemporal, 128–131
  differential diagnosis, 117
  epidemiology, 116, 117, 127
  evaluation of patient with, 118
  and facial paralysis, 110, 130
  ICD-9 code, 120
  laboratory investigation in, 118
  microbiology, 119
  pathology, 119
  physical examination in, 118
  recurrent, 118
    treatment, 120
  risk factors for, 117, 119
  signs and symptoms, 117
  treatment, 116, 119–120
    medical, 119
    surgical, 120
  unilateral, in adults, 117, 119
Acute postobstructive pulmonary edema (APOPE)
  causes, 60
  signs and symptoms, 60
  treatment, 60
  type I, 60
  type II, 60
Acyclovir
  for papillomatosis, 293
  for sudden sensorineural hearing loss, 103
  for vestibular neuritis, 188, 188*t*
Adenitis. *See also* Lymphadenitis
  cervical, in children, 566
Adenocarcinoma
  ceruminous, of ear and temporal bone, 425–426
  intranasal, differential diagnosis, 238
  of temporal bone, 425
Adenoid(s), hypertrophy, 508
  ICD-9 code, 553
Adenoid cystic carcinoma
  of ear and temporal bone, 425–426
  laryngeal, 390–391
  salivary gland, 451
    cribriform, 451
    solid (basaloid), 451
    tubular, 451
Adenoidectomy
  complications, 687
  contraindications to, 686
  dissection instruments for, 686
  indications for, 686
  procedure for, 686
Adenoid facies, 549
Adenotonsillar hypertrophy, 549–553
  clinical features, 549–550
  differential diagnosis, 550
  epidemiology, 549
  evaluation, 550
  history taking in, 550
  ICD-9 codes, 553
  imaging, 550
  laboratory investigation, 550
  physical examination of patient with, 550
  signs and symptoms, 549
  treatment, 551

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Adenotonsillectomy, 547
for adenotonsillar hypertrophy, 551
contraindications to, 548*t*
indications for, 548*t*
Adenotonsillitis, 545–548
complications, 545
differential diagnosis, 546
epidemiology, 545
evaluation, 546
follow-up, 547
ICD-9 codes, 547
imaging, 546
laboratory investigation, 546
microbiology, 545
outcomes with, 547
physical examination of patient with, 546
signs and symptoms, 545–546
treatment, 546–547
Adenovirus, subacute thyroiditis caused by, 479
Adjuvant therapy
chemotherapy as, 352
definition, 352
Advanced Trauma Life Support (ATLS), 338
Advancement flap(s). *See* Cutaneous flap(s), advancement
Aerobes, deep neck infection, 334
Aerodigestive tract, upper. *See* Upper aerodigestive tract
Afrin. *See* Oxymetazoline
AGA. *See* Androgenetic alopecia (AGA)
Agar nasi cells, 212
Ageusia, definition, 257
Aging face
aesthetic evaluation, 654
clinical findings in, 650
evaluation, 646–647
examination, 650–651
implants and fillers for, 646–649
rhytidectomy for, 649–652
Agitation, postoperative, 61
AHI. *See* Apnea–hypopnea index (AHI)
AI. *See* Arousal index (AI)
AIDS (acquired immunodeficiency syndrome). *See* Human immunodeficiency virus (HIV), infection
Air–bone gap, 160
Airway(s). *See also* Upper airway
anatomy, 7–8
assessment, 8–10
difficult, 16–22, 274–275
approach to, 17
ASA algorithm for, 17, 18*f*
conditions associated with, 16*t*
identification, 8–9, 17
management, 17
and preoperative planning, 17
dimensions, by age, 530*t*
emergency surgical, 17–19, 19*f*
in children, 19
foreign body in
in children, 509, 510
and difficult airway, 16*t*
injury, traumatic, signs, 337
innervation, 7–8
management, 10–16. *See also* Intubation
in deep neck infection, 334–335
emergency, 274
in trauma patient, 339–340
obstruction
in children, 510, 513
Pierre Robin sequence and, 533–537
pediatric, anatomy, 507
stable but compromised, 19–22
causes, 19
management, 19
subglottic, topical anesthesia, 35
Airway(s) (artificial)
fire in, 47, 552
nasal, 8
contraindications to, 8
oral, 8
Airway equipment, 8
Airway fire(s), 47, 552
ALA. *See* 5-Aminolevulinic acid (ALA)
Albumin, serum, in hypocalcemia, 503
Alcohol
abuse, and epistaxis, 231
and head and neck cancer, 346
and hypopharyngeal cancer, 383–384
and laryngeal cancer, 389
and nasopharyngeal cancer, 366
and oral cavity cancer, 370
and oropharyngeal cancer, 378–379
Alcohol abuse, preoperative assessment, 6*t*
Alcohol withdrawal
mental status change caused by, 61
postoperative, 61
treatment, 61
Alexander aplasia, 555–556
Alkaline ingestion, 278–280
Alkaloids, mechanism of action, 354
Alkylating agents, mechanism of action, 353
Allen's test, 637
Allergic reaction(s), 247
Gell and Coombs classification, 247, 247*t*
types, 247, 247*t*

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Allergic rhinitis (AR), 243
  acupuncture for, 91
  complementary and alternative medicine (CAM) for, 90
  exacerbation, differential diagnosis, 234
  treatment, 246
Allergic salute, 248
Allergy(ies), 247–250
  and acute otitis media, 119
  chronic, management, 249
  clinical features, 247–248
  differential diagnosis, 248
  emergency, 248
  epidemiology, 247
  follow-up, 249
  ICD-9 codes, 249
  laboratory investigation, 248
  laryngeal manifestations, 322
  outcomes with, 249
  physical examination of patient with, 248
  signs and symptoms, 248
  treatment, 249
  work-up for, 248–249
Alopecia. *See also* Androgenetic alopecia (AGA)
  after rhytidectomy, 652
  epidemiology, 677
  female pattern, 677
    Ludwig scale for, 678
  male pattern, 677
    Norwood scale for, 678
  traction, 678
Alopecia areata, 678
Alpha-2 agonist(s), 40. *See also* Dexmedetomidine
Alphanate, 58
Alport syndrome, 559
  hearing loss in, 165*t*
  laboratory investigation, 562
ALS. *See* Amyotrophic lateral sclerosis (ALS)
Alveolar ridge, malignant neoplasm, ICD-9 code, 375
Amerge. *See* Naratriptan
American Academy of Otolaryngology–Head and Neck Surgery, diagnostic guidelines for Ménière disease, 183, 184*t*
American Society of Anesthesiologists (ASA)
  difficult airway algorithm, 17, 18*f*
  NPO guidelines, 6*t*
  physical status classification system, 5, 7*t*
American Society of Geriatric Otolaryngology (ASGO), 81
Amicar. *See* Aminocaproic acid
Amifostine, 358
Aminocaproic acid, 58
Aminoglycoside
  for malignant otitis externa, 147–148, 148*t*
  ototoxicity, 164, 164*t*
5-Aminolevulinic acid (ALA), in diagnosis of oral cancer, 371
Amiodarone
  hypothyroidism caused by, 475
  thyroiditis caused by, 482
Amitriptyline, for migraine, 192*t*
Amnesia, perioperative, 30
Amoxicillin
  for acute otitis media, 119
  for acute rhinosinusitis
    adult dosage and administration, 235, 236*t*
    pediatric dosage and administration, 236
  for adenotonsillitis, 547
Amoxicillin/clavulanate
  for acute otitis media, 119
  for acute rhinosinusitis
    adult dosage and administration, 235, 236*t*
    pediatric dosage and administration, 236
  for adenotonsillitis, 547
  for bacterial lymphadenitis, in children, 567–568
  for deep neck infection, 335
  dosage and administration, pediatric, 567
Ampicillin, for meningitis prophylaxis, with repair of congenital nasal mass, 583
Ampicillin/sulbactam, for orbital complications of sinusitis, 221
Amyloidosis
  ICD-9 code, 322
  laryngeal manifestations, 321
Amyotrophic lateral sclerosis (ALS)
  ICD-9 code, 290, 322
  laryngeal manifestations, 322
  voice changes caused by, 286–290
ANA. *See* Antinuclear antibody(ies)
Anaerobes
  chronic rhinosinusitis caused by, 240
  deep neck infection, 334
  lymphadenitis caused by, in children, 566
  rhinosinusitis caused by, 235

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Anagen, 678
Anagen effluvium, 678
Analgesia
  perioperative, 30
  preoperative, 10*t*
Analgesics, for migraine, 191
Anaphylaxis, 248
  treatment, 249
Anatomic variation(s), and difficult airway, 16*t*
Androgenetic alopecia (AGA), 677–679
Anemia, thyroid function testing in, 476
Anesthesia. *See also* General anesthesia; Regional anesthesia
  dissociative, 41–42
  drugs for, 36–46. *See also* Anesthetic(s); *specific drug*
  emergence, 31, 31*t*
  factors affecting, 30
  induction, 30, 31*t*
    medications for, 40–43
  maintenance, 31, 31*t*
  modes, 29–30
  for nasolaryngoscopy, 4
  phases, 30–31
  preinduction, 30, 31*t*
  stages, 31–32
  topical
    for awake intubation, 14
    for subglottic airway, 35
Anesthetic(s), 36–46. *See also specific drug*
  ideal, properties, 30
  inhaled, 43–44. *See also specific drug*
    lipid solubility, 43
    minimum alveolar concentration, 43
  volatile, 43–44
Anesthetic emergencies, 47–48
Angioedema, 248
  treatment, 249
Angiofibroma, 251
  differential diagnosis, 238
Angiography
  indications for, 106
  with neck mass, 344
  in neck trauma, 339, 339*t*
  in trauma patient, 340
Angioneurotic edema, laryngeal manifestations, 321–322
Angiotensin-converting enzyme (ACE), 214*t*
Angiotensin-converting enzyme inhibitors, rhinitis caused by, 244
Anorexia, radiation-related, 358
Anosmia, 254–257
  causes, 255
  clinical features, 254–255
  conductive, 254
    treatment, 256
  congenital, 255
  definition, 254
  differential diagnosis, 255
  follow-up, 256
  ICD-9 codes, 256
  mixed, 254
  outcomes with, 256
  posttraumatic, 255, 256
  postviral, 255, 256
  sensorineural, 254, 255
    treatment, 256
  signs and symptoms, 254–255
Ansa cervicalis, roots, 328*f*
Anterior commissure, laryngeal cancer spread to, 388
Anterior compartment (of neck), 405
Antibiotic(s)
  for acute otitis media, 119
  for acute rhinosinusitis, 235–236, 236*t*
  for acute suppurative labyrinthitis, 131
  for adenotonsillitis, 547
  antitumor, mechanism of action, 353
  for bacterial lymphadenitis, in children, 567–568
  for Bezold abscess, 128
  for brain abscess, 132
  for chronic rhinosinusitis, 241, 241*t*
  and *Clostridium difficile* colitis, 64
  for coalescent mastoiditis, 129
  for deep neck infection, 335
  for epidural abscess, 133
  for facial paralysis due to acute otitis media, 130
  for intracranial complications of sinusitis, 223–224
  intravenous, for meningitis, 131, 132*t*
  for lateral sinus thrombosis, 133
  for malignant otitis externa, 147–148, 148*t*
  for meningitis, 131, 132*t*
  for meningitis prophylaxis, with repair of congenital nasal mass, 583
  for necrotizing soft tissue infection, 330
  for orbital complications of sinusitis, 221
  perioperative
    with cholesteatoma removal, 140
    indications for, 140
  for petrous apicitis, 130

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Antibiotic(s) *(continued)*
postoperative, 59
for ear surgery, 126
for subdural empyema, 134
for subperiosteal abscess, 128
topical
for chronic otitis media, 123, 124*t*
for otitis externa, 143, 143*t,* 144
for tympanostomy tube recipient, 120
for upper respiratory infection, in children, 510
for wound infection, 62
Antibiotic resistance, 119
in *Clostridium difficile,* 64
in *Pseudomonas aeruginosa,* 147–148
Antibiotics, ototoxicity, 164, 164*t*
Antibody(ies) (Ab), 68-kD, testing, indications for, 103
Anticholinergics, for nonallergic rhinitis, 246
Anticoagulation, with cavernous sinus thrombophlebitis, 224
Antidepressants
for migraine, 191, 192*t*
for tinnitus, 195
Antiemetic(s), 30
Antifungal agent(s), topical, for otitis externa, 143, 143*t*
Antihelix, auricular, 97*f*
crura, 97*f*
Antihistamines, oral
for acute rhinosinusitis, 236
for chronic rhinosinusitis, 241
Antiinflammatory agent(s), for temporomandibular joint disorders, 75
Antimetabolites, mechanism of action, 353
Antineutrophil cytoplasmic antibody (c-ANCA), 214*t,* 261
testing, indications for, 103
in Wegener granulomatosis, 206
Antinuclear antibody(ies)
testing, indications for, 103
tests for, 214*t*
Antipsychotics, rhinitis caused by, 244
Antisialagogue, for awake intubation, 14
Antithyroid drugs, for hyperthyroidism, 473
Antitragus, 97*f*
Antiviral(s)
for Bell palsy, 113
for sudden sensorineural hearing loss, 103
for vestibular neuritis, 188, 188*t*
Anxiety, postoperative, 61
Anxiolysis, preoperative, 10*t,* 30
AOM. *See* Acute otitis media (AOM)
Aortic arch
congenital anomalies, ICD-9 code, 529
double, 525
treatment, 527–528
left, anomalies, 525
right
with aberrant left subclavian artery, 525
with mirror image branching and left ductus arteriosus, 525
variants, treatment, 527–528
Aortic body, malignant neoplasms, ICD-9 code, 439
Apert syndrome, 558, 588
and choanal atresia, 584
and cleft lip/palate, 587
Aphonia
definition, 398
postlaryngectomy, 398–401
evaluation, 399
signs and symptoms, 399
Apnea, definition, 66
Apnea–hypopnea index (AHI), 67
APOPE. *See* Acute postobstructive pulmonary edema (APOPE)
AR. *See* Allergic rhinitis (AR)
Areca nut. *See* Paan
Argon laser, 84
Arnica montana, 91
Arnold's nerve, 98
ear innervation by, 402
Arnold's reflex, 98
Arousal index (AI), 67
Arrhythmia(s), cardiac, postoperative, 60–61
Arteriovenous malformation (AVM), in children, epidemiology, 570
Arthrocentesis, for temporomandibular joint disorders, 76
Arthroplasty, for temporomandibular joint disorders, 76
Arthroscopic surgery, for temporomandibular joint disorders, 76
Arytenoidectomy, for bilateral vocal fold paralysis, 516
Arytenoidopexy, for bilateral vocal fold paralysis, 516
ASA. *See* American Society of Anesthesiologists (ASA)
ASGO. *See* American Society of Geriatric Otolaryngology (ASGO)

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Aspergillosis. *See also* Acute invasive fungal rhinosinusitis
ICD-9 code, 218
laryngitis caused by, 282
Aspergillus
identification, 218
malignant otitis externa caused by, 147
otitis externa caused by, 143
Aspiration, 311–317
causes, 312–313
complications, 316
differential diagnosis, 312–313
in elderly, 81
evaluation, 313–314
foreign body, 507
imaging, 509
ICD-9 codes, 316
imaging, 313
primary, 312
prophylaxis, preoperative, 10*t*
secondary, 312
signs and symptoms, 312
in sleep, 312
treatment, 314, 315*t*–316*t*
Aspiration pneumonia. *See also* Aspiration
deep neck infection and, 335
ICD-9 code, 316
Aspirin
and epistaxis, 230
rhinitis caused by, 244
Assessment
airway, 8–10
preoperative, 5, 6*t*, 7*t*
Assistive devices, for hearing loss, 170
Association, definition, 537
ASSRs. *See* Auditory steady-state responses (ASSRs)
Asthma
acupuncture for, 91
complementary and alternative medicine (CAM) for, 90
Atlantoaxial joint, mobility, assessment, 9
Atlantooccipital joint, mobility, assessment, 9
ATLS. *See* Advanced Trauma Life Support (ATLS)
Atresia, and congenital conductive hearing loss, 560
Atticotomy, 124
Audiogram, 149, 150*f*
with cerebellopontine angle tumor, 197
with cholesteatoma, 137
in chronic otitis media, 123
of cochlear implant candidate, 173
indications for, 187
in Ménière disease, 184–185
of new patient, reviewing, 5
Audiologic testing, in balance disorders, 178
Audiology
basic assessments, 149–155
follow-up, 155
objective/electrophysiologic assessments, 157–159
outcomes with, 155
pediatric assessments, 155–157
Audiometry
acute otitis media and, 118
behavioral, 152
behavioral observation, in children, 156
in children, 156, 563, 564*t*
indications for, 160
play, in children, 156
pure tone, indications for, 167
visual reinforcement, in children, 156
Auditory brainstem implant (ABI), 174–175, 564
complications, 175
follow-up, 175
indications for, 175
outcomes with, 175
principles, 175
use rate, 175
Auditory brainstem response (ABR), 158
assessment procedure, 158
with cerebellopontine angle tumor, 197
clinical applications, 158
factors affecting, 158
norms, 158
testing
indications for, 167
pediatric, 156
waves seen on, ECOLI mnemonic for, 158
Auditory neuropathy/dyssynchrony, pediatric, hearing loss caused by, 561–562
Auditory steady-state response (ASSRs)
assessment procedure, 158
clinical applications, 158
factors affecting, 158
Auricle
aesthetic deformities, evaluation, 666
age-related changes in, 666
anatomy, 97, 97*f*, 666
avulsion
epidemiology, 104
treatment, 107

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Auricle *(continued)*
basal cell carcinoma, 424–427
bite wound, treatment, 107
embryology, 95, 95*t*
frostbite injury
signs and symptoms, 104
treatment, 107
hematoma, 105
epidemiology, 104
treatment, 107
innervation of, 98
laceration
epidemiology, 104
treatment, 107
thermal injury (burn), 104
epidemiology, 104
treatment, 107
trauma
epidemiology, 104
signs and symptoms, 105
Auricular abnormalities, 666
Autoimmune disease
hearing loss caused by, 165
hypoparathyroidism in, 500
otologic manifestations, 206
and vertigo, 183
Autophony, 202
AVM. *See* Arteriovenous malformation (AVM)
Azelastine, 246
Azithromycin, for adenotonsillitis, 547
Aztreonam, for orbital complications of sinusitis, 221

B

Bacteria, gram-negative, chronic rhinosinusitis caused by, 240
Bacterial infection
acute otitis media caused by, 119
chronic otitis media caused by, 123
chronic rhinosinusitis caused by, 240
deep neck, 334
laryngitis caused by, 281–282
lymphadenitis caused by, in children, 566
rhinosinusitis caused by, 235
acute, 233–237. *See also* Rhinosinusitis, acute
sialadenitis caused by, 444–445
transfusion-related, 54
Bacteroides fragilis, deep neck infection, 334
BAER (brainstem auditory evoked responses). *See* Auditory brainstem response (ABR)
BAHA. *See* Bone-anchored hearing aid (BAHA)
Balance
assessment, 176–179
physical examination in, 177
physiologic components, 176
Balance disorders
pathology, 178
treatment, 178
Balance dysfunction, in elderly, 80
Balloon sinus dilation, 242
Barbiturates
adverse effects and side effects, 43
for anesthesia induction, 43
contraindications to, 43
dosage and administration, 43
mechanism of action, 43
pharmacology, 43
Bardach repair
for cleft lip, 591
for cleft palate, 592, 592*f*
Barium esophagram, 27
indications for, 27, 318
with vascular ring, 527
Barium swallow, 27
indications for, 318, 384
modified, 313
modified (MBS), 27
Barotrauma, hearing loss caused by, 165
Barrett esophagus, 318, 319
Bartonella henselae, lymphadenitis caused by, in children, 566
Basal cell carcinoma (BCC)
auricular, 424
epidemiology, 407
follow-up, 409
genetic disorders associated with, 407
of head and neck, 407
ICD-9 codes, 410
of lip, 373
of middle ear, 424–427
outcomes with, 409
prevention, 407, 409
recurrence, 409
risk factors for, 407
of skin (head, face, and neck), 407–411
clinical features, 408
differential diagnosis, 408
evaluation, 408
histopathologic types, 408–409
morpheaform, 408
nodular, 408
pathology, 408–409

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physical examination of patient with, 408
pigmented, 408–409
signs and symptoms, 408
superficial, 409
treatment, 414*t*
medical, 409
surgical, 409, 410*f*
of temporal bone, 424–427
treatment, 407
Basal cell nevus syndrome, and basal cell carcinoma, 407
Basilar membrane, 101*f*
Battery(ies), disc, in ear, 115
Battle's sign, 105
BCC. *See* Basal cell carcinoma (BCC)
Beckwith-Wiedemann syndrome, and Pierre Robin sequence, 534
Bell palsy, 109–113
clinical features, 109–110
differential diagnosis, 109–110, 110*f*
epidemiology, 109
onset, 109
outcomes with, 113
recurrent, evaluation of patient with, 113
signs and symptoms, 109
treatment, 109
medical, 112–113
surgical, 113
Benign paroxysmal positional vertigo (BPPV), 179–182
after vestibular neuritis, 187, 189
clinical features, 179–180
differential diagnosis, 180, 183
epidemiology, 176, 179
evaluation, 180
follow-up, 182
ICD-9 code, 182
outcomes with, 182
pathology, 180
physical findings in, 180
signs and symptoms, 179–180
temporal bone injury and, 108
treatment, 178, 181–182, 181*f*
Benzodiazepine(s), 38–40. *See also specific drug*
cardiovascular effects, 39*t*
drug interactions, 39*t*
mechanism of action, 38
neurologic effects, 39*t*
pharmacology, 38
physiologic effects, 39*t*
respiratory effects, 39*t*
reversal, 40
for vestibular neuritis, 188, 188*t*
Beta-blockers
for hyperthyroidism, 473
for migraine, 191, 192*t*
rhinitis caused by, 244
Beta-lactam allergy, and antibiotic therapy for acute rhinosinusitis, 235, 236, 236*t*
Betel leaf. *See* Paan
Bezold abscess, 128
Bicitra, preoperative, 10*t*
Bilateral vocal fold paralysis (BVFP), 297
acquired, 514, 515
congenital, 514, 515
differential diagnosis, 515
epidemiology, 514
evaluation, 515
ICD-9 code, 516
imaging, 515
pathology, 515
physical examination of patient with, 515
signs and symptoms, 514
treatment, 515–516
Bill's bar, 101
Bing-Siebenmann dysplasia, 556
Biofeedback, for tinnitus, 90
Biopsy, sinonasal, 216
Bisphosphonates, for hypercalcemia, 504
Blastomycosis, laryngitis caused by, 282
Bleeding, oronasopharyngeal, trauma-related, 338
Bleomycin
for head and neck cancer, 349
mechanism of action, 353
Blepharochalasia, 660
Blepharoplasty, 660–665
complications, 665
contraindications to, 662–663
evaluation for, 662
indications for, 660
lower eyelid, 663
subciliary approach, 663–664
surgical goals for, 663–664
surgical pearls, 664
transconjunctival approach, 664
upper eyelid, 663
Blepharoptosis, 660
Blood
compatibility testing, 51
donor, screening, 51
stored, coagulation factor depletion in, 55

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Blood *(continued)*
universal donor, 52
whole
stored, microaggregates in, 54
therapy with, 51
Blood component therapy, 51–52
complications, 52–55
immunosuppressive, 53–54
infectious, 54
group O Rh-negative, 52
massive transfusion, 52
and coagulopathy, 54
and dilutional thrombocytopenia, 55
Blood loss
allowable, 51
intraoperative, replacement, 50
management, 51–52. *See also* Blood component therapy
Blood type and crossmatch, 51
Blood type and screen, 51
Blood typing, 51
Blood volume, estimated, 51
Blunt trauma, to neck, 276
BOF syndrome. *See* Branchio-oculo-facial syndrome
Bone-anchored hearing aid (BAHA), 108, 167, 170, 174–175, 199, 564
follow-up, 175
indications for, 167, 175
outcomes with, 175
principles, 175
use rate, 175
Bone disease, otologic manifestations, 207–208
Bone graft(s), for facial reconstruction, 639–642
complications, 641–642
evaluation for, 640
indications for, 640
outcomes with, 642
sources for, 640–641
Bone marrow transplantation (BMT), and acute invasive fungal rhinosinusitis, 215, 218
Bones, stones, and groans, of hyperparathyroidism, 496–497
Bony labyrinth, anatomy, 100
Bony spiral lamina, 101*f*
Borrelia burgdorferi. *See* Lyme disease
Botox, 113, 289, 307, 624, 650
injection into sphenopalatine ganglion, 246
for temporomandibular joint disorders, 76
Bowen disease, 413
BPPV. *See* Benign paroxysmal positional vertigo (BPPV)
Brachial plexus, 327
injury to, in neck dissection, 406
Brachytherapy, 356–357
Brain, abscess, otitis media–related, 131–132
Brainstem, compression
signs and symptoms, 196
tumors causing, 196
Brainstem auditory evoked responses (BAER). *See* Auditory brainstem response (ABR)
Brainstem evoked responses (BSER). *See* Auditory brainstem response (ABR)
Branchial arch(es), 402
in auricle formation, 95, 95*t*
fifth, anomalies, 574
first, abnormalities
type 1, 573–574
type 2, 573–574
fourth, anomalies, 574
in middle ear formation, 95
second, anomalies, 574, 575*f*
structures formed from, 573, 574*t*
third, anomalies, 574, 575*f*
Branchial cleft cyst
in children, 573–577
classification, 573–574
differential diagnosis, 575–576
epidemiology, 573
evaluation, 576
and fistula, 573
ICD-9 code, 577
imaging, 576
pathology, 576
signs and symptoms, 575
and sinus tract, 573
treatment, 576
complications, 577
follow-up, 577
outcomes with, 577
Branchio-oculo-facial syndrome, 538
Branchio-oto-renal syndrome, 558
evaluation, 563
hearing loss in, 165*t*
laboratory investigation, 562
Brandt-Daroff exercises, 182
Bronchial anomalies, ICD-9 code, 511
Bronchiolitis, in children, 509

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Bronchitis
acute, complementary and alternative medicine (CAM) for, 90
in children, 509
Bronchomalacia, 508
treatment, 528
Bronchoscopy. *See also* Panendoscopy
flexible
advantages and disadvantages, 682
complications, 682
indications for, 682
procedure for, 682
rigid, 681
advantages and disadvantages, 681
complications, 682
diagnostic, 681
indications for, 681
procedure for, 681
therapeutic, 681
with vascular ring, 527
Bronchus, anomalies, ICD-9 code, 275
Brow
aesthetic evaluation, 654
ptosis, 653
Brow lifting, 653–656
endoscopic, 655–656
surgical approaches and techniques, 654–656
Brucellosis, transfusion-related, 54
BSER (brainstem evoked responses). *See* Auditory brainstem response (ABR)
Buccal mucosa, 374
cancer, 373–374
prognosis for, 374
survival, by stage, 374
treatment, 374
premalignant conditions, 374
Bullous myringitis, differential diagnosis, 142
Burkitt lymphoma (BL), 431*t*
Burn(s), caustic. *See* Caustic injury
ICD-9 codes, 280
Butterbur, 90
BVFP. *See* Bilateral vocal fold paralysis (BVFP)

C

Calcimimetics, 498
Calcitonin, 461
and calcium metabolism, 502
for hypercalcemia, 504
serum, in patient with thyroid nodule, 469
Calcitriol, for hypoparathyroidism, 501
Calcium
balance
calcitonin and, 461
disorders, 501–504
distribution in body, 501
metabolism, regulation, 502
plasma/serum
corrected, 50
free ionized, 50
imbalance, 50
normal concentration, 50
serum, elevated, in hyperparathyroidism, 497–498
supplementation
in hypocalcemia, 503
in hypoparathyroidism, 501
Calcium alginate swab, 235
Calcium carbonate, for hypocalcemia, 65
Calcium-channel blockers, for migraine, 191, 192*t*
Calcium gluconate, for hypocalcemia, 65
Calcium hydroxylapatite, 648*t*
Calgiswab, 235, 239
Calvarial bone graft(s), for facial reconstruction, 640–641
CAM. *See* Complementary and alternative medicine (CAM)
c-ANCA. *See* Antineutrophil cytoplasmic antibody (c-ANCA)
Cancer of unknown primary (CUP), 349–350
Candida infection. *See* Candidiasis
Candidiasis
laryngitis caused by, 282
otitis externa caused by, 143
Canthoplasty, 625
Canthus, other malignant neoplasm, ICD-9 code, 410, 414
Capsulopalpebral fascia, 661
Captique. *See* Hyaluronic acid
Carbon dioxide (CO_2) laser, 82, 83, 85, 513, 658
Carcinoma ex pleomorphic adenoma, salivary gland, 452
Cardiac catheterization, with angiography, with vascular ring, 527
Cardiac system, preoperative assessment, 6*t*
Cardiovascular system
benzodiazepines and, 39*t*
etomidate and, 42*t*
isoflurane and, 44*t*
ketamine and, 42*t*
opioids and, 37*t*
propofol and, 41*t*

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Carhart notch, 161
Carotid artery
  aneurysm, deep neck infection and, 335
  branches, 326–327, 327*f*
  branchial cleft anomalies and, 574, 575*f*
  stenosis, radiation-related, 358
Carotid artery blowout, 63–64
  after neck dissection, 406
Carotid body, malignant neoplasms, ICD-9 code, 439
Carotid body tumor, 437
Carotid precautions, 63
Carotid sheath, 326, 327*f*
Cartilage graft(s)
  for facial reconstruction, 639–642
    complications, 641–642
    evaluation for, 640
    indications for, 640
    outcomes with, 642
    sources, 641
  harvesting, 641
Catagen, 678
Catel-Manzke syndrome, 538
Cat-scratch disease
  lymphadenitis caused by, in children, 566
  treatment, 567
Caustic injury
  clinical presentation, 278
  epidemiology, 278
  evaluation, 278–279
  follow-up, 279
  ICD-9 codes, 280
  outcomes with, 279
  physical examination of patient with, 279
  treatment, 279
  work-up for, 278–279
Cavernous sinus thrombophlebitis
  and anticoagulation, 224
  signs and symptoms, 222
  sinusitis and, 222
Cavernous sinus thrombosis, 219, 219*t*, 220
  treatment, 221
Cefepime, for intracranial complications of sinusitis, 223
Cefotaxime, for meningitis prophylaxis, with repair of congenital nasal mass, 583
Cefoxitin, for deep neck infection, 335
Ceftazidime, for malignant otitis externa, 147, 148*t*
Ceftin, for chronic rhinosinusitis, 241
Ceftriaxone
  for acute rhinosinusitis, adult dosage and administration, 236, 236*t*
  for intracranial complications of sinusitis, 223
  for meningitis prophylaxis, with repair of congenital nasal mass, 583
  for upper respiratory infection, in children, 510
Cefuroxime axetil
  for acute rhinosinusitis
    adult dosage and administration, 235, 236*t*
    pediatric dosage and administration, 236
  for adenotonsillitis, 547
  for deep neck infection, 335
Cellulitis
  auricular, 117, 118
  in children, 567
  orbital, 219*t*, 220
    ICD-9 code, 221
  periauricular, 141–142
  peritonsillar, ICD-9 code, 336
  postseptal, treatment, 221
  preseptal, 219*t*
    treatment, 221
  preseptal (periorbital), 220
Central compartment (of neck), 325, 326*f*
Central nervous system (CNS)
  benzodiazepines and, 39*t*
  etomidate and, 42*t*
  injury, aspiration in, 313
  isoflurane and, 44*t*
  ketamine and, 42*t*
  opioids and, 37*t*
  propofol and, 41*t*
Cephalexin, for bacterial lymphadenitis, in children, 567
Cephalosporins
  for acute otitis media, 119
  for orbital complications of sinusitis, 221
Cerebellopontine angle (CPA), anatomy, 196
Cerebellopontine angle tumor, 195–201. *See also* Meningioma; Vestibular schwannoma
  clinical presentation, 196
  differential diagnosis, 196
  evaluation, 196–197
  and facial nerve function, 98
  follow-up, 199–201

STUDENTS-HUB.com Uploaded By: tuka abuzneid

hearing loss caused by, 165
ICD-9 codes, 201
imaging, 196
outcomes with, 199
physical examination of patient with, 196
resection
approaches for, 198–199, 198*f*
complications, 199, 200*t*
signs and symptoms, 196
and tinnitus, 196
vertigo and disequilibrium caused by, 196
Cerebritis, 220
Cerebrospinal fluid (CSF)
findings
in encephalitis, 223, 224*t*
in meningitis, 223, 224*t*
normal, 223, 224*t*
intraoperative management, in ear surgery, 126
leak
with anterior skull base disruption, 225–228
naso-orbital-ethmoid fractures and, 601
postoperative, 199, 200*t*
with repair of congenital nasal mass, 583
leaks, 199
otorrhea, 199
treatment, 108
rhinorrhea, 199, 225–228
causes, 225
detection, 612
differential diagnosis, 225
epidemiology, 225
evaluation, 225–226
follow-up, 228
history taking in, 225–226
ICD-9 code, 228
imaging, 226
laboratory investigation, 226
outcomes with, 228
physical examination of patient with, 226
signs and symptoms, 225
treatment, 108, 227–228
work-up for, 226–227, 227*t*
Cerebrovascular accident
neurolaryngeal involvement in, treatment, 288
pathophysiology, 288
Cerebrovascular disease, hearing loss caused by, 166
Ceruminosis, in elderly, 80
Cervical fascial planes, 325, 325*f*
Cervical lymphadenopathy, with nasopharyngeal carcinoma, 365
Cervical plexus, 327, 328*f*
anatomy, 33
deep, block, 34
innervation of external auditory canal, 98
superficial, block, 33–34
Cervical spine
mobility, assessment, 9
radiography, 339
Cervical vestibular evoked myogenic potential (cVEMP), testing, indications for, 160
Cetacaine, for awake intubation, 14
Cetuximab
for head and neck cancer, 349
mechanism of action, 354
as radiation sensitizer, 358
Chagas disease, transfusion-related, 54
Chandler classification, 219, 219*t*
Charcot-Marie-Tooth disease
and hearing loss, 561
laryngeal manifestations, 322
CHARGE association, 538
and choanal atresia, 584
Cheek flap, 632–633
Chemical peels, 656–658
complications, 658–659
outcomes with, 659
postprocedure care for, 659
Chemoradiotherapy
concomitant, 352, 353
regimens, 353
for nasopharyngeal carcinoma, 367
for oropharyngeal cancer, 380
Chemotherapy
and acute invasive fungal rhinosinusitis, 215
adjuvant, 353
adverse effects and side effects, 354
for anaplastic thyroid cancer, 489
complications, 354
concurrent with radiation, 352, 353
for head and neck cancer, 349, 352–355
agents for, 353–354
for Hodgkin lymphoma, 432
for idiopathic midline destructive disease, 436
induction, 352

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Chemotherapy *(continued)*
for malignant salivary gland tumors, 454
neoadjuvant
advantages and disadvantages, 352
definition, 352
regimens, 352
for non-Hodgkin lymphoma, 433
platinum-based, ototoxicity, 164, 164*t*
postoperative, 352
postradiotherapy, 352
preoperative, 352
preradiotherapy, 352
Chest radiograph(s)
indications for, 384
of vascular ring, 526
Child(ren)
audiometric assessment, 563, 564*t*
hearing loss in, 553–565
meningitis in, antibiotics for, 132*t*
neck mass in, infectious, 565–569
Chinese herbal medicine, 91
for asthma, 90
CHL. *See* Conductive hearing loss (CHL)
Chloramphenicol, *p*-aminobenzenesulfonamide, and amphotericin, with or without hydrocortisone (CSF powder), for chronic otitis media, 123, 124*t*
Chloride, daily requirements for, 49
Choanal atresia, 508, 584–586
associated anomalies, 584
bilateral, 584
differential diagnosis, 585
differential diagnosis, 585
epidemiology, 584
evaluation, 585
follow-up, 586
imaging, 585
outcomes with, 586
pathology, 585
physical examination of patient with, 585
signs and symptoms, 585
surgery for, 586
treatment, 584, 586
unilateral, 584
differential diagnosis, 585
Cholesteatoma
acquired, 135
epidemiology, 136
pathology, 138
primary, 138
secondary, 138
signs and symptoms, 136
and chronic otitis media, 121, 136
clinical features, 136
complications, 136, 139
congenital, 135
epidemiology, 136
pathology, 137–138
signs and symptoms, 136
differential diagnosis, 136
epidemiology, 136
evaluation, 136–137
and facial paralysis, 122, 130
follow-up, 126, 140
and hearing loss, 562
ICD-9 code, 126, 140
imaging, 137
and labyrinthine fistula, 128
outcomes with, 140
pathology, 123, 137–138
pathophysiology, 135, 137–138
physical findings with, 136
recurrence, 126
monitoring for, 140
signs and symptoms, 121
treatment, 138–139
surgical, 125, 128–129
Chondromyxoid fibroma, 251
Chondrosarcoma, 251, 360
of ear and temporal bone, 426
Chordoma, of ear and temporal bone, 426
Christmas disease. *See* Hemophilia B
Chromophore(s), 82
Chronic otitis media (COM), 121–126
and cholesteatoma, 136
clinical features, 121–122
complications, 125, 127–135, 139
differential diagnosis, 122
with effusion, 121
epidemiology, 121
evaluation, 122–123
and facial paralysis, 130
follow-up, 126
ICD-9 codes, 126, 140
imaging in, 122
laboratory investigation, 122
microbiology, 123
outcomes with, 126
pathology, 123
physical examination of patient with, 122
signs and symptoms, 121–122
treatment, 121, 123–125
medical, 123, 124*t*
surgical, 124–125
Chronic rhinosinusitis (CRS), 237–243
clinical features, 238
CT findings in, 239*t*

STUDENTS-HUB.com Uploaded By: tuka abuzneid

definition, 237
diagnosis, 238
differential diagnosis, 234, 238
with eosinophilic mucin, 240
epidemiology, 238
evaluation, 238–240
follow-up, 242
in HIV-infected (AIDS) patients, 240
ICD-9 codes, 242
imaging, 239
laboratory investigation, 240
microbiology, 240
minor and major diagnostic factors, 238
nasal endoscopic findings in, 239*t*
with or without polyps, 240
outcomes with, 242
pathogenesis, 240
physical examination of patient with, 239
risk factors for, 240
signs and symptoms, 238
subtypes, 240
treatment, 262
medical, 240–241, 241*t*
surgical, 241–242
Chronic serous otitis, 121
Chronic suppurative otitis media (CSOM)
with or without cholesteatoma. *See* Chronic otitis media (COM)
and temporal bone cancer, 424
Churg-Strauss syndrome
diagnosis, 260*t*
nasal manifestations, 238
sinonasal manifestations, 259
treatment, 260*t*
Chvostek sign, 65, 502
Chylothorax, perioperative, 528
Chylous fistula, after neck dissection, 62–63, 406
Cidofovir, for papillomatosis, 293
Ciliary beat frequency, 214*t*
Ciliary electyron microscopy, 214*t*
Cinacalcet hydrochloride, 498
Cinefluoroscopy, in dysphagia, 309–310
Ciprodex
for chronic otitis media, 124*t*
for otitis externa, 143*t*
for tympanostomy tube recipient, 120
Ciprofloxacin
for malignant otitis externa, 147–148, 148*t*
topical, for otitis externa, 144
Ciprofloxacin/dexamthasone, topical, for otitis externa, 144
Cipro HC otic
for chronic otitis media, 124*t*
for otitis externa, 143*t*
Cisatracurium
clinical considerations with, 46*t*
dosage and administration, 46*t*
pharmacology, 46*t*
Cisplatin
for head and neck cancer, 349
mechanism of action, 353
Citrate toxicity, transfusion-related, 54
Clarithromycin, for acute rhinosinusitis
adult dosage and administration, 236*t*
pediatric dosage and administration, 236
Clark levels, for squamous cell carcinoma of skin (head, face, and neck), 416
Cleft lip, 587–594
epidemiology, 588
evaluation, 589
ICD-9 code, 594
imaging, 589
nonsyndromic, 587
repair, 590*f*, 591, 591*f*
complications, 593
follow-up, 593–594
outcomes with, 593–594
timing, rule of tens for, 591
signs and symptoms, 588
syndromic, 587, 588
treatment, 590–592
Cleft palate, 587–594
epidemiology, 588
evaluation, 589
ICD-9 code, 537, 594
imaging, 589
nonsyndromic, 587
in Pierre Robin sequence, 534
repair, 592, 592*f*, 593*f*
complications, 593
follow-up, 593–594
outcomes with, 593–594
signs and symptoms, 588
syndromic, 587, 588
treatment, 590–592
Clindamycin
for acute otitis media, 119
for acute rhinosinusitis, adult dosage and administration, 236, 236*t*
for bacterial lymphadenitis, in children, 567–568
for deep neck infection, 335

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Clindamycin *(continued)*
dosage and administration
adult, 567
pediatric, 567
for orbital complications of sinusitis, 221
Clonazepam
for perimenstrual migraine with vertigo, 192
for vestibular neuritis, 188, 188*t*
Clostridium difficile
antibiotic resistance in, 64
colitis, postoperative, 64
and infection control, 64
CMV. *See* Cytomegalovirus (CMV) infection
CN. *See* Cranial nerve(s) (CN)
Coagulation study(ies), 55–56
Coagulopathy, transfusion-related, 54
Cocaine
intoxication, treatment, 40
nasal, for awake intubation, 14
Coccidioidomycosis, laryngitis caused by, 282
Cochlea, 100
embryology, 96*f*
malformation, 165
Cochlear aqueduct, 100, 101
embryology, 96*f*
Cochlear duct, 96*f*, 100, 101*f*
Cochlear implant (CI), 157, 171–174
candidates for
adult, 172
history taking from, 172
imaging, 172
pediatric, 171–172
physical examination, 172
complications, 173
components, 171
evaluation for, 172–173
follow-up, 173–174
ICD-9 codes, 174
indications for, 167, 170, 562, 564
outcomes with, 173–174
programming, 174
surgical approach for, 173
Cochlear nerve, 691
Coenzyme Q_{10}, 90
Colloid, thyroid, 461, 463
COM. *See* Chronic otitis media (COM)
Communication method(s), postlaryngectomy, 399
Complementary and alternative medicine (CAM), 89–92
for acute sinusitis, 90
for allergic rhinitis, 90
for asthma, 90
dangerous practices in, 91
for tinnitus, 90, 194–195
for upper respiratory infections, 90
use
patients' reasons for, 89
prevalence, 89
for vertigo, 90
Computed tomographic angiography, of neck trauma, 339
Computed tomography (CT), 22–24, 251. *See also* Positron emission tomography (PET) with computed tomography (PET-CT)
in acute invasive fungal rhinosinusitis, 216
advantages and disadvantages, 22–23
attenuation of body components in, 24, 24*t*
of cancer of unknown primary, 350
of cholesteatoma, 137
and chronic otitis media, 122
of chronic rhinosinusitis, 239
of cochlear implant candidate, 172
for computer-assisted surgical navigation, 24, 24*t*
contrast-enhanced, 24
for lateral sinus thrombosis, 133
coronal, preoperative review, 242, 242*t*
with CSF rhinorrhea, 226, 227*t*
of deep neck infection, 334
in facial paralysis/paresis, 111
of head and neck carcinoma, 348
helical, 23
of hypopharyngeal cancer, 384
indications for, 22–23, 118, 255–256
in-office flat plate cone beam, 23
of intranasal mass, 231
of Ludwig angina, 332
multidetector, 23
of nasopharyngeal carcinoma, 366
of neck mass, 342
of neck trauma, 339
of necrotizing soft tissue infection, 329
in oral cancer, 371
of orbital infection, 220
of orbits, in hyperthyroidism, 472
parathyroid, 497
principles, 23, 23*f*
of rhinosinusitis, 235
temporal bone, indications for, 146, 146*t*
of temporal bone, indications for, 160, 172, 202–203

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of thyroid nodule, 468
of vascular ring, 527
Concha bullosa, auricular, 97*f*
Conchal cartilage graft(s), for facial reconstruction, sources, 641
Conductive hearing loss (CHL)
audiogram in, 150, 150*f*
causes, 159–160
with cholesteatoma, 136, 137
congenital, 560
atresia and, 560
syndromic, 560
evaluation, 152–154, 160
external auditory canal–related, 159
ICD-9 codes, 162, 171
imaging, 160
middle ear–related, 159–160
pediatric
acquired postnatal, 562
epidemiology, 554
trauma-related, 561
temporal bone fracture and, 108
treatment, 160–161. *See also* Hearing aid(s)
complications, 162
follow-up, 162
outcomes with, 162
tympanic membrane–related, 159–160
Confusion, postoperative, 59–61
differential diagnosis, 59
work-up for, 59–60
Congenital anomaly(ies), 537–544
and difficult airway, 16*t*
epidemiology, 537
evaluation, 544
history taking for, 544
imaging, 544
laboratory investigation, 544
physical examination of patient with, 544
treatment, 544
Connexin 26 anomalies, 165, 556
and hearing loss in children, 556
Continuous positive airway pressure (CPAP), for obstructive sleep apnea, 67
Contrast media, for esophageal fluoroscopy, 27
Conversion disorder, ICD-9 code, 304
Cormack and Lehane grades, 10, 10*f*
Cornea, care for
with Bell palsy, 113
before facial reanimation, 113
Cortical deafness, 157
Corticosteroids
chronic therapy with, and acute invasive fungal rhinosinusitis, 215
for thyroiditis, 482
Cortisporin otic suspension
for chronic otitis media, 124*t*
for otitis externa, 143*t*, 144
Corti tunnel, 101*f*
Cosmetic surgery. *See* Blepharoplasty; Brow lifting; Facial plastic and reconstructive surgery; Forehead lift; Liposuction; Otoplasty; Rhinoplasty; Rhytidectomy
Costal cartilage graft(s), for facial reconstruction, sources, 641
Costello syndrome, 538–539
Cotton-Mayer grading system, for subglottic stenosis, 530, 531*t*
Cough, radiation-related, 358
Coumadin, epistaxis caused by, 230
Coxsackie virus
sialadenitis caused by, 443–444
subacute thyroiditis caused by, 479
CPA. *See* Cerebellopontine angle (CPA)
CPAP. *See* Continuous positive airway pressure (CPAP)
Cranial nerve(s) (CN), 327, 689–694
I (olfactory). *See* Olfactory nerve (cranial nerve I)
II (optic). *See* Optic nerve (cranial nerve II)
III (oculomotor). *See* Oculomotor nerve (cranial nerve III)
IV (trochlear). *See* Trochlear nerve (cranial nerve IV)
V (trigeminal). *See* Trigeminal nerve (cranial nerve V)
VI (abducens). *See* Abducens nerve (cranial nerve VI)
VII (facial). *See* Facial nerve (cranial nerve VII)
VIII (vestibulocochlear). *See* Vestibulocochlear nerve (cranial nerve VIII)
IX (glossopharyngeal). *See* Glossopharyngeal nerve (cranial nerve IX)
X (vagus). *See* Vagus nerve (cranial nerve X)
XI (spinal accessory). *See* Spinal accessory nerve (cranial nerve XI)
XII (hypoglossal). *See* Hypoglossal nerve (cranial nerve XII)
airway innervation by, 7–8
anatomy, 327
benign neoplasm, ICD-9 code, 201

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Cranial nerve(s) (CN) *(continued)*
branchial cleft anomalies and, 574, 575*f*
injury to
ICD-9 code, 256
in neck dissection, 406
palsy, 122, 145, 196
and taste, 257
Cranial neuropathy(ies)
with malignant otitis externa, 145, 148
with nasopharyngeal carcinoma, 365
paragangliomas and, 437, 439
with petrous apicitis, 129
with sinonasal cancer, 360
tumors causing, 196
Craniopharyngioma, hypothyroidism caused by, 475
Craniotomy, complications, 200*t*
Cricoid cartilage, 268, 269*f*
Cricoidectomy
partial, 314, 316*t*
subperichondrial, 314, 315*t*
Cricoid pressure, for endotracheal intubation, 12
Cricoid split, for subglottic stenosis, 532
Cricothyroid membrane, 268–269
Cricothyroid muscle, innervation, 8
Cricothyroidotomy, 17–19, 19*f*
complications, 688
contraindications to, 17
indications for, 17
needle, 19
procedure for, 688
in trauma patient, 340
Cricotracheal ligament, 268–269
Cricovocal membrane, 269
Cri-du-chat syndrome, 539
Cromolyn, nasal, for chronic allergy, 249
Croup
ICD-9 code, 284
signs and symptoms, 281
viral, 508
Crouzon syndrome, 559
CRS. *See* Chronic rhinosinusitis (CRS)
Crus commune, 100, 101
Cryoprecipitate
components, 52
for hemophilia A, 57
indications for, 52
von Willebrand factor in, 58
Cryosurgery, for basal cell carcinoma, 409
Cryptococcosis, laryngitis caused by, 282
Cryptophthalmos, 540
Crystalloid, for anaphylaxis, 249
CSF. *See* Cerebrospinal fluid (CSF)
CSF powder, for chronic otitis media, 123, 124*t*
CSOM. *See* Chronic suppurative otitis media (CSOM)
CT. *See* Computed tomography (CT)
CUP. *See* Cancer of unknown primary (CUP)
Cutaneous flap(s)
advancement, 631, 631*t*
bipedicled (H-plasty), 631–632, 631*t*
pedicles, 631, 631*t*
unipedicled (U-plasty), 631–632, 631*t*, 632*f*
V-Y island, 631*t*, 632, 632*f*
interpolated, 631, 631*t*, 634
interposition, 631, 631*t*
island, 631, 631*t*
local
blood supply in, 630
for facial reconstruction, 629–635
complications, 635
nomenclature for, 630–631, 631*t*
pivotal, 631, 631*t*, 632–633
rotation, 631, 631*t*, 632–633
transposition, 631, 631*t*, 633–634, 633*f*, 634*f*
bilobed, 633, 634*f*
melolabial, 633–634
rhombic, 633, 633*f*
CWD. *See* Tympanomastoidectomy, with canal wall down (CWD)
CWU. *See* Tympanomastoidectomy, with canal wall up (CWU)
Cyclobenzaprine, for temporomandibular joint disorders, 76
Cyclooxygenase (COX) II inhibitors, preoperative, 10*t*
Cylindroma. *See* Adenoid cystic carcinoma
Cymba concha, 97*f*
Cyst(s)
dental, 69
periapical, clinical features, 69–70
radicular, clinical features, 69–70
thyroid, 467, 469–470, 576
vocal fold, 295–297
Cytomegalovirus (CMV) infection
and acquired prenatal hearing loss, 560
lymphadenitis caused by, in children, 566
transfusion-related, 54
Cytomel. *See* Liothyronine

D

da Vinci Surgical System (dVSS), 86–87
DCP. *See* Deep cervical plexus (DCP)
DDAVP. *See* Desmopressin acetate (DDAVP)

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Decadron, for vestibular neuritis, 188
Decongestant(s)
oral
for acute rhinosinusitis, 236
for chronic allergy, 249
topical
for acute rhinosinusitis, 236
for chronic allergy, 249
for chronic rhinosinusitis, 241
Dedo laryngoscope, 8, 16
Deep cervical fascia, 325*f*
Deep cervical plexus (DCP), block, 34
Deep neck infection(s), 333–336. *See also* Ludwig angina
clinical features, 333–334
complications, 335
diagnosis, 333
differential diagnosis, 334
evaluation, 334
follow-up, 335
ICD-9 codes, 336
imaging, 334
laboratory investigation, 334
microbiology, 334
outcomes with, 335
pediatric, signs and symptoms, 334
physical examination of patient with, 334
risk factors for, 333, 335
signs and symptoms, 333–334
treatment, 333, 334–335
medical, 335
surgical, 335
Degenerative disorders
aspiration in, 312
voice changes caused by, 286–290
Delirium, postoperative, 61
Delta sign, with lateral sinus thrombosis, 133
Demerol. *See* Meperidine
Dental infection, 329
in children, 567
differential diagnosis, 616
Dental radiograph(s), in oral cancer, 371
Dermabrasion, 645, 650, 652, 656
Dermatitis, radiation-related, 358
Dermatochalasis, 660
Dermis, 627
Dermoid cyst
congenital midline, 577–580
signs and symptoms, 581–582
congenital nasal, 581–584
pathology, 583
treatment, 583
pathology, 579
signs and symptoms, 578
treatment, 580
Desflurane
pharmacology, 43–44
physiologic effects, 44
Desmopressin
for hemophilia A, 57
indications for, 232
for von Willebrand disease, 58
Desmopressin acetate (DDAVP), indications for, 232
Dexamethasone
for anaphylaxis, 249
for angioedema, 249
intratympanic perfusion, for Ménière disease, 185
Dexmedetomidine
for awake intubation, 14
dosage and administration, 40
pharmacology, 40
Diabetes mellitus
and acute invasive fungal rhinosinusitis, 215, 218
blood glucose level in, and necrotizing otitis externa, 142
malignant otitis externa in, 147
Diabetic ketoacidosis, and acute invasive fungal rhinosinusitis, 215, 218
Diamox, for Ménière disease, 167
Diarrhea, postoperative, 64
Diazepam
dosage and administration, 39
pharmacology, 39
preoperative, 10*t*
for temporomandibular joint disorders, 76
for vestibular neuritis, 188, 188*t*
DIC. *See* Disseminated intravascular coagulation (DIC)
Difficult airway. *See* Airway(s), difficult
DiGeorge syndrome
hypoparathyroidism in, 500
otologic manifestations, 208
Diiodotyrosine (DIT), 463
Diode laser, 84
Diphenhydramine
for anaphylaxis, 249
for angioedema, 249
2,3-Diphosphoglycerate, decreased, 54
Discoid lupus erythematosus, chronic
otologic manifestations, 206
treatment, 206
Disequilibrium, 176–177

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Disseminated intravascular coagulation (DIC), 55
deep neck infection and, 335
DIT. *See* Diiodotyrosine (DIT)
Diuretics
for hypercalcemia, 504
for Ménière disease, 167, 185
ototoxicity, 164, 164*t*
Dix-Hallpike maneuver, 177, 179, 180, 181
Dizziness, epidemiology, 176
Docetaxel, mechanism of action, 354
Domeboro, for otitis externa, 144
Down syndrome, 539
Doxorubicin
for anaplastic thyroid cancer, 489
mechanism of action, 353
Doxycycline, for acute rhinosinusitis, adult dosage and administration, 235, 236*t*
DPG. *See* 2,3-Diphosphoglycerate
Drug(s)
epistaxis caused by, 230, 230*t*
hypothyroidism caused by, 475
Drug–drug interactions
benzodiazepines and, 39*t*
opioids and, 37*t*
dVSS. *See* da Vinci Surgical System (dVSS)
Dyazide. *See* Diuretics
Dye laser, 84–85
Dynamic posturography, 177
Dysgeusia, 109, 258
Dysphagia, 308–311
after tracheoesophageal fistula/esophageal atresia repair, 524
associated symptoms, 308
clinical features, 308–309
definition, 308
differential diagnosis, 309
in elderly, 81
epidemiology, 308
evaluation, 309–310
ICD-9 codes, 311
imaging, 309–310
outcomes with, 311
physical examination of patient with, 309
radiation-related, 358
signs and symptoms, 308
trauma-related, 338
treatment, 310, 314
work-up for, 309–310
Zenker diverticulum and, 305–307
Dysphonia, postlaryngectomy, 398–401
evaluation, 399
signs and symptoms, 399

E

EA. *See* Esophageal atresia (EA)
EAC. *See* External auditory canal (EAC)
Ear(s)
embryology, 95–97, 402
external, carcinoma, lymph node metastases, 343*f*
foreign body in, 114–116
acute, 114
chronic, 114
clinical features, 114
differential diagnosis, 114
epidemiology, 114
evaluation, 114–115
follow-up, 116
history taking for, 114–115
ICD-9 code, 116
imaging, 115
outcomes with, 116
physical examination of patient with, 115
removal, 115
signs and symptoms, 114
treatment, 114, 115
innervation, 402
malignant neoplasms, 424–427
biopsy, 425
clinical features, 424–425
differential diagnosis, 424–425
evaluation, 425
follow-up, 427
ICD-9 codes, 427
imaging, 425
outcomes with, 427
pathology, 425
physical examination of patient with, 425
signs and symptoms, 424
staging, 426
treatment, 426–427
melanoma, ICD-9 code, 420
pain. *See also* Otalgia
in children, 117
differential diagnosis, 117
with malignant otitis externa, 145
in otitis externa, 142
skin
malignant neoplasm, ICD-9 code, 427
melanoma, ICD-9 code, 427

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other malignant neoplasm, ICD-9 code, 410, 414, 427
trauma
clinical features, 105
differential diagnosis, 105
epidemiology, 104
evaluation of patient with, 105–107
follow-up, 108
outcomes with, 108
penetrating, treatment, 107
physical examination of patient with, 105–107
treatment, 107–108
Ear candling, 91
Earlobe, 97*f*
deformity, after rhytidectomy, 652
Ear plug(s), indications for, 120
EBV. *See* Epstein-Barr virus (EBV); Estimated blood volume (EBV)
ECF. *See* Extracellular fluid (ECF)
Echinacea, for cold prevention and treatment, 90
Echocardiography, with vascular ring, 527
Echovirus
sialadenitis caused by, 443–444
subacute thyroiditis caused by, 479
ECochG. *See* Electrocochleography (ECochG)
Ectrodactyly-ectodermal dysplasia-clefting (EEC) syndrome, 539
Ectropion, postoperative, 613
Ehlers-Danlos syndrome, and hearing loss, 561
Eikenella corrodens, deep neck infection, 334
Elavil, for tinnitus, 195
Elderly patient(s). *See also* Geriatric otolaryngology
demographics, 78
end-of-life care wishes, 79
general approach to, 78, 78*t*
history taking from, 78–79, 78*t*
medication history for, 79
medication reconciliation for, 79
otologic disorders common in, 80
physical examination, 79
preoperative risk stratification for, 80, 80*t*
primary care records for, 78
review of systems for, 80, 80*t*
rhinologic disorders in, 81
social history for, 79
Electrocochleography (ECochG), 158
assessment procedure, 158
clinical applications, 159
factors affecting, 158
in Ménière disease, 183, 185
sensitivity, 158
Electrolarynx, 394
postlaryngectomy use, 399, 400
Electrolyte(s), 48–49, 63, 64
daily requirements for, 49
imbalance, postoperative, 65
in perioperative fluids, 49, 49*t*
postoperative management, 65
Electromyography (EMG). *See also* Laryngeal electromyography (LEMG)
in facial paralysis, 112
Electroneuronography (ENoG)
clinical applications, 159
in facial paralysis, 112
factors affecting, 158
timing, 158
Electronystagmography (ENG), 177
with cerebellopontine angle tumor, 197
factors affecting, 178
Emergency(ies), 695–696
allergic, 248
anesthetic, 47–48
esophageal, 272
laryngeal, 272
neck, 328
otologic. *See also* Bell palsy; Trauma, ear; Trauma, temporal bone
sudden sensorineural hearing loss as, 102–104
ololaryngologic, requiring immediate intervention, 696–697
rhinologic, 215–233
Emesis. *See* Postoperative nausea and vomiting (PONV)
EMG. *See* Electromyography (EMG)
Empyema
deep neck infection and, 335
subdural
epidemiology, 134
imaging, 134
pathophysiology, 134
signs and symptoms, 134
treatment, 134
Encephalocele
congenital nasal, 581–584
pathology, 583
signs and symptoms, 582
treatment, 583
ICD-9 code, 584
sincipital, 581

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Encephalomeningocele, 581
Encephalomeningocystocele, 581
Endocrine disorders, in elderly, 81
Endocrine system
  etomidate and, 42*t*
  opioids and, 37*t*
Endolaryngeal stenting, 314, 316*t*
Endolymph, 100
Endolymphatic duct, 96*f,* 100, 101
Endolymphatic hydrops. *See* Ménière disease
Endolymphatic sac, 101
  embryology, 96*f*
  surgery, for Ménière disease, 185–186
  tumors, 425
    hearing loss caused by, 165
Endolymphatic sac tumor, vertigo caused by, 183
Endoscopic examination, 4. *See also* Panendoscopy
  in head and neck cancer, 348–349
  in hypopharyngeal cancer, 385
  nasal, 226, 235, 239
Endoscopic sinus surgery
  for chronic rhinosinusitis, 241–242
  instrumentation, 242
Endoscopy
  for forehead and brow lifting, 655–656
  nasal, indications for, 551
  operative
    for inverted papilloma, 252–253
    for laryngeal clefts, 519–520
  in trauma patient, 340
Endotracheal intubation. *See* Intubation, endotracheal
Endotracheal tube(s)
  armored, 11
  laser-resistant, 11
  length of insertion, 11
  nerve-monitoring, 11
  for pediatric patient, 11
  Ring-Adair-Elwyn (RAE), 11
  size, recommendations, 11
  types, 11
ENG. *See* Electronystagmography (ENG)
Enlarged vestibular aqueduct syndrome, 165
ENoG. *See* Electroneuronography (ENoG)
Enterovirus, lymphadenitis caused by, in children, 566
Eosinophilic granuloma, 238
  ear canal, 425
Ephedra, 91
Epidermal growth factor receptor(s) (EGFR), inhibitors, mechanism of action, 354
Epidermis, 627
Epidermoid, 196
Epidermolysis bullosa, laryngeal manifestations, 321
Epidural abscess. *See* Abscess, epidural
Epiglottic flap laryngeal closure, 314, 315*t*
Epiglottis, 268, 269*f,* 389
  anatomy, 7–8
Epiglottitis
  bacterial, 281–282
  in children, 508
  ICD-9 code, 284
Epinephrine, for anaphylaxis, 249
Epistaxis, 229–233
  anterior, 229
    treatment, 232
  bleeding source in, localization, 231, 232
  causes, 229, 230*t*
  definition, 229
  differential diagnosis, 229
  drug-related, 230, 230*t*
  in elderly, 81
  epidemiology, 229
  follow-up, 233
  history taking for, 230–231
  ICD-9 code, 233
  imaging, 231
  infection and, 230*t*
  laboratory investigation, 231
  with nasopharyngeal carcinoma, 365
  neoplasms causing, 229, 230*t*
  outcomes with, 233
  physical examination of patient with, 231
  posterior, 229
    treatment, 232
  refractory, management, 232
  signs and symptoms, 229
  in systemic disease, 230*t*
  trauma-related, 229, 230*t*
  treatment, 231–233
Epitympanum, 98
Epley repositioning maneuver, 179, 181–182, 181*f*
Epstein-Barr virus (EBV)
  and idiopathic midline destructive disease, 436
  infection, transfusion-related, 54
  latent membrane proteins, 364
  lymphadenitis caused by, in children, 566

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and nasopharyngeal carcinoma, 346, 364, 366, 367
Erbitux. *See* Cetuximab
Erbium,chromium:yttrium-scandium-gallium-garnet (Er,Cr:YSGG) laser, 84
Erbium:yttrium-aluminum-garnet (Er:YAG) laser, 84
Erythrocyte sedimentation rate, 214*t*
  indications for, 103
Erythroplasia, 370
Escherichia coli, sialadenitis caused by, 445
Esophageal atresia (EA), 521–525
  associated anomalies, 521
  classification, 522, 523*f*
  epidemiology, 521
  evaluation, 522
  ICD-9 code, 524
  imaging, 522
  outcomes with, 524
  physical examination of patient with, 522
  signs and symptoms, 521
  treatment, 522
    complications, 522–524
Esophageal emergency(ies), 272
Esophageal manometry, 310
Esophageal pH testing, 310
Esophageal reflux, 317–319
Esophageal speech, postlaryngectomy use, 399, 400–401
Esophageal stenosis, ICD-9 code, 524
Esophageal strictures, after tracheoesophageal fistula/esophageal atresia repair, 524
Esophageal voice, 394–395
Esophagitis, reflux, 317–319
Esophagoscopy. *See also* Panendoscopy
  complications, 683
  equipment for, 682
  flexible, 682
    advantages and disadvantages, 683
    diagnostic, 683
  procedure for, 682–683
  rigid, 682–683
    advantages and disadvantages, 683
    diagnostic, 683
    indications for, 683
    therapeutic, 683
Esophagram
  contrast media for, 27
  indications for, 27
Esophagus
  anatomy, 270–271
  blood supply to, 271
  caustic injury, 278–280
    grading, 280*t*
  innervation, 271
  lymphatic drainage, 271
  physiology, 271
ESR. *See* Erythrocyte sedimentation rate
Essential tremor
  ICD-9 code, 290
  treatment, 290
Esthesioneuroblastoma, 251, 360
  differential diagnosis, 238
Estimated blood volume (EBV), 51
Ethmoidal artery(ies), 212, 213*f*
Ethmoidal sinus, malignant neoplasm, ICD-9 code, 361
Ethmoid sinus, 211–212, 212*f*
  cancer, 359. *See also* Sinonasal cancer
    staging, 362
Etomidate
  cardiovascular effects, 42*t*
  CNS effects, 42*t*
  dosage and administration, 41
  endocirne effects, 42*t*
  pharmacology, 41
  physiologic effects, 41, 42*t*
  respiratory effects, 42*t*
Eustachian tube
  anatomy, 100, 266*f*
  embryology, 95
  function, testing, 154
  functions, 100
  length, 100
  obstruction, by mass lesion, 117, 118
  penetration of nasopharyngeal secretions into, in infants, 119
Explosion(s), laser-related, 85
External auditory canal (EAC), 97*f*
  anatomy, 97–98
  embryology, 95
  foreign body in, 114–116
    differential diagnosis, 117
  hearing loss related to, 159
  infection. *See* Otitis externa (OE)
  innervation, 98
  melanoma, ICD-9 code, 420
  skin
    melanoma, ICD-9 code, 427
    other malignant neoplasm, ICD-9 code, 410, 414, 427

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External auditory canal (EAC) *(continued)*
squamous cell carcinoma, 424
trauma, differential diagnosis, 117
External carotid artery
anatomy, 326
branches, 326, 327*f*
nasal blood supply, 212, 213*f*
Extracellular fluid (ECF), 49
Extubation, 31, 31*t*
criteria for, 22
Eye(s), care for
with Bell palsy, 113
with facial weakness, 199
Eyebrow(s). *See* Brow
Eye care, 624–625
Eye injury(ies), laser-related, 85
Eyelid(s). *See also* Blepharoplasty
age-related changes in, 660
anatomy, 660, 661*f*
lower, wedge resection, 625
prosthetics for, insertion, 625
skin, other malignant neoplasm, ICD-9 code, 410, 414

F

Face
aging. *See* Aging face
bone and articular cartilage, malignant neoplasm, ICD-9 code, 427
injury, ICD-9 code, 341
melanoma, ICD-9 code, 420
nerve block, 32–33
relaxed skin tension lines, 630, 643, 643*f*
skin. *See* Facial skin
Face bone(s), fractures, ICD-9 code, 602
Facelift. *See* Facial plastic and reconstructive surgery; Rhytidectomy
Face mask(s), 8
Facial artery, 212, 213*f*
Facial defects
causes, 629
evaluation, 629–630, 630*t*
repair, local cutaneous flaps for, 629–635
Facial enhancement, indications for, 113
Facial nerve (cranial nerve VII), 98, 213, 257, 266, 309, 327, 540, 689*f*, 690
cross grafting, 624
decompression, middle fossa, 113
dysfunction, with petrous apicitis, 129
embryology, 574*t*
function, House-Brackmann grading scale, 111, 111*t*
functions, 689*f*, 690
injury. *See also* Facial reanimation
extratemporal, 621
iatrogenic, 125, 613
intracranial, 620
intratemporal, 621
in neck dissection, 406
innervation of external auditory canal, 98
interpositional graft, 622–623, 623*f*
monitoring, intraoperative, 125
neuromas, management, 199
neurorrhaphy, 621–622, 622*f*
Ramsay Hunt branch, ear innervation by, 402
recurrent, evaluation of patient with, 113
segments, 689*f*
and taste, 257
weakness, postoperative, 125
Facial numbness, tumors causing, 196
Facial paralysis/paresis. *See also* Facial reanimation
acute, 109–113. *See also* Bell palsy
differential diagnosis, 109–110, 110*f*
electrical testing with, 112
evaluation of patient with, 110–112
history taking in, 110
imaging in, 111
laboratory testing with, 111
pathology, 112
physical examination of patient with, 111
treatment, 112–113
cholesteatoma and, 122, 130
with cochlear implant, 173
etiology, 130
eye care in, 624–625
follow-up, 113
iatrogenic injury and, 449, 450
imaging, 130
outcomes with, 113
pathology, 130
surgery for, 113
trauma-related
electrophysiologic testing in, 107
treatment, 108
treatment, 130
Facial plastic and reconstructive surgery. *See also* Facial reconstruction
fillers for, 646–649
implants for, 646–649
incision planning for, 643–646
scar revision, 643–646
Facial reanimation, 620–624
ancillary techniques, 624
cross facial nerve grafting for, 624

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indications for, 113
interpositional graft for, 622–623, 623*f*
muscle transfer techniques for, 624
nerve crossover for, 623
options for, 621–624
Facial reconstruction
bone grafts for, 639–642
cartilage grafts for, 639–642
free tissue transfer for, 636–639
local cutaneous flaps for, 629–635
skin grafts for, 626–629
Facial skin, other malignant neoplasm, ICD-9 code, 410, 415
Facial vein(s), 212
Facial weakness
postoperative, with cerebellopontine angle tumor resection, 199
progression, 109
tumors causing, 196
Factor III, infusion, for von Willebrand disease, 58
Factor V, depletion, in stored blood, 55
Factor VIII
concentrate
for hemophilia A, 57
von Willebrand factor in, 58
deficiency, 57. *See also* Hemophilia A
depletion, in stored blood, 55
Factor IX, deficiency, 57. *See also* Hemophilia B
Falciform (transverse) crest, 101
Familial hyperparathyroidism-jaw tumor syndrome, 496
Famotidine, preoperative, 10*t*
Fasciocutaneous flap(s), for facial reconstruction, 637–638
Fat grafting, 646, 647
Fatigue, radiation-related, 358
FDG. *See* [^{18}F]fluorodeoxyglucose
FEES. *See* Fiberoptic endoscopic evaluation of swallowing (FEES)
Fentanyl
adverse effects and side effects, 36
for awake intubation, 14
dosage and administration, 36
pharmacology, 36
preoperative, 10*t*
Fetal alcohol syndrome, and Pierre Robin sequence, 534
Fever, postoperative, 58–59
work-up for, 59
[^{18}F]fluorodeoxyglucose, 27, 28
FFP. *See* Fresh-frozen plasma (FFP)
Fiberoptic endoscopic evaluation of swallowing (FEES), 310, 313, 314
Fibrous dysplasia
otologic manifestations, 208
pathophysiology, 208
temporal bone, hearing loss caused by, 165
Fillers, injectable, 646–647, 648*t*
Finasteride, 679
Fine-needle aspiration biopsy (FNAB)
of benign salivary gland tumor, 449
of head and neck cancer, 348
of lymphoma, 432
of malignant salivary gland tumor, 454
of neck mass, 345, 390
of thyroid, 466
of thyroid nodule, 468–469
ultrasound-guided, 26
Fire(s)
airway, 47, 552
laser-related, 85
Fistula(s)
labyrinthine
epidemiology, 128
etiology, 128
imaging, 128
physical examination of patient with, 128
signs and symptoms, 128
treatment, 128–129
salivary gland, ICD-9 code, 447
semicircular canal, ICD-9 code, 204
Flagyl, for *Clostridium difficile* colitis, 64
Flexeril. *See* Cyclobenzaprine
Floor of mouth
cancer
clinical presentation, 374
survival, by stage, 374
treatment, 374
malignant neoplasm, ICD-9 code, 376
Floxin Otic
for chronic otitis media, 124*t*
for otitis externa, 143*t*
for tympanostomy tube recipient, 120
Fluid(s), 48–50
body, functional compartments, 49
intraoperative, management, 49–50
perioperative
electrolytes in, 49, 49*t*
maintenance requirement, 49
management, 48–50
postoperative management, 59, 64
third space (redistribution loss), 50
Fluid deficit, 50

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Flumazenil
dosage and administration, 40
pharmacology, 40
Fluorescein, intrathecal, for CSF leak detection, 227, 227*t*
Fluorescent treponemal antibody absorption test (FTA-ABS), 214*t*
indications for, 103
Fluoroquinolones, for malignant otitis externa, 147–148, 148*t*
5-Fluorouracil
for basal cell carcinoma, 409
for head and neck cancer, 349
FM system(s), classroom, 157
FNAB. *See* Fine-needle aspiration biopsy (FNAB)
Forehead
aesthetic evaluation, 654
anatomy of, 653–654
Forehead flap, 634
Forehead lift, 653–656
endoscopic, 655–656
surgical approaches and techniques, 654–656
Foreign body
airway
in children, 509, 510
and difficult airway, 16*t*
aspiration, 507, 508
imaging, 509
in ear, 114–116
otitis externa caused by, 143, 144
Foscan, in diagnosis of oral cancer, 371
Fragile X syndrome, 539
Fraser syndrome, 540
Free tissue transfer, for facial reconstruction, 636–639
complications, 638
evaluation for, 636–637
follow-up, 638–639
imaging for, 637
indications for, 636
laboratory assays for, 637
medical therapeutics for, 637
outcomes with, 638–639
surgical techniques for, 637–638
work-up for, 636–637
Freidrich ataxia, and hearing loss, 561
Fresh frozen plasma (FFP), 52
Frey syndrome, 450
Frontal bone, anatomy, 605–606
Frontalis muscle(s), 653
Frontal sinus, 211–212, 212*f*
anatomy, 605–606
cancer, 359. *See also* Sinonasal cancer
fractures, 605–609
complications, 608
differential diagnosis, 606
epidemiology, 606
evaluation, 606–607
follow-up, 608
ICD-9 code, 608
imaging, 607
laboratory investigation, 607
outcomes with, 608
physical examination of patient with, 606
signs and symptoms, 606
treatment, 607–608
work-up for, 606–607
malignant neoplasm, ICD-9 code, 361
FT3. *See* Triiodothyronine (T_3), free
FT4. *See* Thyroxine (T_4), free
FTA-ABS. *See* Fluorescent treponemal antibody absorption test (FTA-ABS)
FTSG. *See* Full-thickness skin graft (FTSG)
Full-thickness skin graft (FTSG), 626, 627
for facial reconstruction, operative technique for, 628
Fungal infection. *See also* Acute invasive fungal rhinosinusitis
laryngitis caused by, 282
otitis externa caused by, 141, 143
thyroiditis caused by, 481
Furlow palatoplasty, for cleft palate, 592, 593*f*
Furosemide, for hypercalcemia, 504
Furstenberg test, 582, 582*t*
Furuncle, localized, 142
Fusobacterium, deep neck infection, 334

G

Gabapentin
benzodiazepines and, 38
preoperative, 10*t*
for tinnitus, 195
Gallium-67 citrate scan, indications for, 146*t*, 147
Gallium nitrate, for hypercalcemia, 504
Gamma-aminobutyric acid (GABA), barbiturates and, 43
Gamma knife stereotactic radiosurgery, 356
for vestibular schwannoma, 197–198
Gangrene, ICD-9 code, 330
Gastric reflux, in children, 508

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Gastroesophageal reflux disease (GERD), 317–319
  complications, 319
  epidemiology, 317
  evaluation, 318
  outcomes with, 319
  signs and symptoms, 317
  treatment, 318–319
Gastrografin, 27
Gastrointestinal system, opioids and, 37*t*
Gatifloxacin, for acute rhinosinusitis, adult dosage and administration, 236
Gell and Coombs classification, of allergic reactions, 247, 247*t*
General anesthesia, 29
Gene therapy
  for head and neck cancer, 349
  for thyroid cancer, 488
Genetics, 537
Gentamicin, intratympanic, for Ménière disease, 185
Gentian violet, aqueous
  for chronic otitis media, 123, 124*t*
  for otitis externa, 143*t*
GERD. *See* Gastroesophageal reflux disease (GERD)
Geriatric Care Otolaryngology, 81
Geriatric otolaryngology, 77–81. *See also* Elderly patient(s)
  evaluation in, 78, 78*t*
Giant cell granuloma, 251
Gingiva, malignant neoplasm, ICD-9 code, 375
Ginkgo biloba, 90
Ginseng, 90
Glabella, 211, 211*f*
GlideScope, 8, 15–16
Glioma, congenital nasal, 581–584
  pathology, 583
  signs and symptoms, 582
  treatment, 583
Glomus jugulare tumor, 437. *See also* Paraganglioma(s)
Glomus tumor(s). *See* Paraganglioma(s)
Glomus tympanicum tumor, 437. *See also* Paraganglioma(s)
Glomus vagale tumor, 437. *See also* Paraganglioma(s)
Glossectomy, midline, for obstructive sleep apnea, 68
Glossopharyngeal nerve (cranial nerve IX), 266, 309, 327, 575*f*, 692*f*
  airway innervation by, 7–8
  block, 35
  branches, 8
  embryology, 574*t*
  functions, 691
  and taste, 257
Glossoptosis, in Pierre Robin sequence, 534
Glottic closure, surgical, 314, 315*t*
Glottis, 388–389
  malignant neoplasm, ICD-9 code, 395
Glucocorticoid(s), for hypercalcemia, 504
Glycopyrrolate, for awake intubation, 14
Goiter, 458, 482
  lingual, 460*f*
  toxic multinodular, 472, 473
    ICD-9 code, 474
Goldenhar syndrome, 557
  hearing loss in, 165*t*
Gold weight implant, for eyelid, 625
Gorlin syndrome
  and basal cell carcinoma, 407
  clinical features, 407
Gout
  otologic manifestations, 207
  pathophysiology, 207
Gradenigo syndrome, 129
Graft-versus-host disease (GVHD), transfusion-related, 53
Granuloma(s), periapical, 69
  clinical features, 69–70
  differential diagnosis, 70
  epidemiology, 69
  evaluation, 70
  signs and symptoms, 69–70
  treatment, 70
Granulomatous disease, otologic manifestations, 204–206
Graves disease, 471–472
  differential diagnosis, 471–472, 482
  epidemiology, 471
  etiology, 471
  HLA association in, 471
  ICD-9 code, 474
  physical examination of patient with, 471
  signs and symptoms, 471
  treatment, 473
Graves ophthalmopathy, 471, 472
  treatment, 473
Gray (Gy), 355

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Great auricular nerve, 327, 328*f*
ear innervation by, 402
Greater occipital nerve, block, 33
Guaifenesin
for chronic rhinosinusitis, 241
nasal, for acute rhinosinusitis, 236
Guedel tube, 12*f*
Guillain-Barré syndrome, laryngeal manifestations, 322
Gum, malignant neoplasm, ICD-9 code, 375
Gunshot wound(s), temporal bone, 107, 108
Gustation. *See* Taste
GVHD. *See* Graft-versus-host disease (GVHD)

H

Haemophilus influenzae
antibiotic resistance in, 119
deep neck infection, 334
laryngitis caused by, 282
nontypable, acute otitis media caused by, 119
rhinosinusitis caused by, 235
sialadenitis caused by, 445
sinusitis, orbital complications, 220
upper respiratory infection, in children, 510
Hair, growth, 677–678
Hair follicle(s), anatomy of, 677
Hairline, deformity, after rhytidectomy, 652
Hair loss, 678. *See also* Alopecia
radiation-related, 358
Hair restoration, 677–679
Hair transplantation, 679
Haldol, for postoperative agitation, 61
Haller cell, 212
Hamartoma, 251
Hard palate, cancer, 375
Hashimoto thyroiditis, 474, 479, 480*t*, 481
HBV. *See* Hepatitis B virus (HBV)
HCV. *See* Hepatitis C virus (HCV)
Headache
with malignant otitis externa, 145
sinus, 238
Head and neck
blood vessel, injury, ICD-9 code, 341
cancer, 346–351. *See also* Head and neck squamous cell carcinoma (HNSCC)
cervical lymph node metastases, 347, 347*t*, 404
chemotherapy for, 352–355
differential diagnosis, 347
in elderly, 81
epidemiology, 399
evaluation, 348
follow-up, 350
history taking for, 348
ICD-9 code, 350
imaging, 348
laboratory investigations, 348
metastatic work-up, 348
outcomes with, 350
pathology, 349
physical examination of patient with, 348
radiation therapy for, 355–358
signs and symptoms, 347
staging, 351
treatment, 349
of unknown primary, 349–350
PET-CT
of cancer of unknown primary, 350
indications for, 384
in melanoma, 418
in oral cancer, 371
superficial nerves, injury, ICD-9 code, 341
Head and neck squamous cell carcinoma (HNSCC), 346–351
biopsy, 348
endoscopic assessment, 348–349
epidemiology, 346
risk factors for, 346
with second primary, 348
treatment, 349
Head injury(ies), hypothyroidism caused by, 475
Head thrust test, in vestibular neuritis, 187
Hearing aid(s), 168–171. *See also* Bone-anchored hearing aid (BAHA); Implantable hearing devices
BiCROS, 170
for children, 157, 563–564
CROS, 167, 170
evaluation for, 169, 169*t*
features, 170
FM systems used with, 170
follow-up, 168, 170
indications for, 167
maintenance, 170
new technologies, 167
outcomes with, 168, 170
pediatric, 170
underuse, 168

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for unilateral complete hearing loss, 199
use, prevalence, 168
Hearing impairment. *See* Hearing loss
Hearing loss
acquired, 154–155
assistive devices for, 170
associated symptoms, 166
asymmetric, 163
differential diagnosis, 165–166
bilateral
rapidly progressive, 165
slowly progressive, 163
causes, 154–155
in children
imaging in, 166
laboratory testing in, 167
with cholesteatoma, 136, 137
cholesteatoma and, 121, 136
clinical features, 149–152
conductive. *See* Conductive hearing loss
congenital, 154, 164–165, 164*f*, 554
causes, 164–165
epidemiology, 156
hereditary, 554
nonhereditary, 554
non-hereditary, 164–165
diagnostic testing in, 167
in elderly, prevalence, 149
epidemiology, 149
evaluation, 152–154, 166, 169, 169*t*
fluctuating, 166, 167
in Ménière disease, 183
follow-up, 168
hereditary, 164–165
nonsyndromic, 154
syndromic, 154, 164–165, 165*f*
history taking in, 166
ICD-9 code, 565
imaging in, 166
laboratory testing in, 167
medical referral in, 155
mixed, audiogram in, 150*f*
noise-induced, 163
audiogram in, 150
treatment, 167
otitis externa and, 141–142
outcomes with, 168
pathology, 154–155
pediatric, 553–565
acquired, 560–562
acquired postnatal, 561
acquired prenatal, 560–561
clinical features, 156
epidemiology, 156, 554
evaluation, 156, 562–563
follow-up, 157, 564–565
imaging in, 562
laboratory investigation, 562–563
outcomes with, 157, 564
physical examination of patient with, 562
signs and symptoms, 156, 555
treatment, 156–157, 563–564
complications, 564
types, 554, 554*f*
physical examination of patient with, 166
present at birth, 164–165
causes, 164–165
classification, 164, 164*f*
progressive, 166, 167
sensorineural. *See* Sensorineural hearing loss (SNHL)
severity, classification, 167
signs and symptoms, 149
sudden, 102–104
tumors causing, 196
symmetric, 163
syndromic, 543
treatment, 155
medical, 167
surgical, 167
unilateral
treatment, 199
tumors causing, 165, 196
untreated
adverse effects, 168–169
prevalence, 168
in vestibular disorders, 177
X-linked, 559
Hearing test(s), 152–154
in children, 156
Heck disease, 71–73
Heerfordt syndrome, 205, 446
Helix, auricular, 97*f*
Hemangioma(s)
in children, 569–573
differential diagnosis, 571
epidemiology, 570
evaluation, 571
outcomes with, 572
signs and symptoms, 570
treatment, 572
ear canal, 425
ICD-9 code, 573
subglottic, 508, 510
Hematologic disorders, 56–58
Hematology, 50–58

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Hematoma
after neck dissection, 406
after rhytidectomy, 651
postoperative, 61–62
with thyroid surgery, 62
Hematopoietic tissue, malignant neoplasm, ICD-9 code, 434
Hemidiaphragm paresis, after neck dissection, 406
Hemilaryngectomy, vertical, for laryngeal cancer, 393
Hemochromatosis, hypoparathyroidism in, 500
Hemoglobin S (HbS), 56
Hemophilia A, 57
cryoprecipitate for, 52
Hemophilia B, 57
Hemorrhage
in neck dissection, 406
postoperative, with adenotonsillectomy, 551–552, 552*t*
posttonsillectomy, 551–552, 552*t*
tonsillar, 547, 551–552, 552*t*
Hepatitis, viral, transfusion-related, 54
Hepatitis B virus (HBV), infection, transfusion-related, 54
Hepatitis C virus (HCV), infection, transfusion-related, 54
Herbal medicine, 91
for asthma, 90
for papillomatosis, 293
Hereditary angioedema, ICD-9 code, 322
Herpes simplex virus (HSV)
and facial paralysis/paresis, 112
lymphadenitis caused by, in children, 566
and vestibular neuritis, 188
Herpes zoster oticus, 110
differential diagnosis, 117, 142
and facial paralysis, 109
outcomes with, 113
Hillocks of His, in auricular embryology, 95, 95*t*
Histamine (H_1) antagonists, for chronic allergy, 249
Histamine (H_2) antagonists
for anaphylaxis, 249
for angioedema, 249
for reflux, 318
Histiocytic tissue, malignant neoplasm, ICD-9 code, 434
Histiocytosis X, sinonasal manifestations, 259
Histoplasmosis, laryngitis caused by, 282
History taking
from elderly patient, 78–79, 78*t*
from of head and neck surgery patient, 3, 3*t*
from new patient, 3, 3*t*
from otolaryngology patient, 3, 3*t*
Hitselberger sign, 98
HIV. *See* Human immunodeficiency virus (HIV)
HL. *See* Hodgkin lymphoma (HL)
Hoarseness
in adults, 292
in children, 292
ICD-9 code, 290, 322
radiation-related, 358
systemic disorders causing, 319–322
trauma-related, 338
Hodgkin lymphoma (HL), 428–434. *See also* Lymphoma(s)
biopsy, 433
clinical features, 428–429
epidemiology, 428
ICD-9 code, 434
prognosis for, 433–434
staging, 429*t*
treatment, 432
complications, 434
WHO/REAL classification, 432, 433*t*
Holinger laryngoscope, 8, 16
Holmium:yttrium-aluminum-garnet (Ho:YAG) laser, 84
Homeopathy, 90, 91
Horner syndrome, after neck dissection, 406
House-Brackmann classification, 111, 111*t*
Ho:YAG laser. *See* Holmium:yttrium-aluminum-garnet (Ho:YAG) laser
Human immunodeficiency virus (HIV)
infection
and acute invasive fungal rhinosinusitis, 215
otologic manifestations, 208
transfusion-related, 54
sialadenitis caused by, 443–444
Human immunodeficiency virus (HIV) infection, sinonasal manifestations, 259
Human leukocyte antigen(s) (HLA)
in Graves disease, 471
in nasopharyngeal carcinoma, 366
and subacute thyroiditis, 479

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Human papillomavirus (HPV)
focal epithelial hyperplasia caused by, 71
and head and neck cancer, 346
and oral cancer, 370
and oropharyngeal cancer, 378
vaccine, 293
and verrucous carcinoma, 372
warts caused by, 71
Humate-P, 58
Hungry bone syndrome, 500, 501
Hyaluronic acid, 648*t*, 649
Hyaluronidase, injectable, 649
Hydration, intravenous, for vestibular neuritis, 188, 188*t*
Hydrocephalus
otitic
imaging, 134
pathology, 134
pathophysiology, 134
physical findings in, 134
signs and symptoms, 134
treatment, 135
tumors causing, 196
Hyoid suspension, for obstructive sleep apnea, 68
Hyperacusis, assessment, 203
Hyperbaric oxygen therapy, for necrotizing soft tissue infection, 330
Hyperbilirubinemia, and acquired prenatal hearing loss, 561
Hypercalcemia, 501–504
acute severe, treatment, 504
chronic severe, treatment, 504
etiology, 503
evaluation, 504
familial hypocalciuric, 498
hyperparathyroidism in, 499
ICD-9 code, 504
laboratory investigation, 504
malignancy-associated, 503–504
signs and symptoms, 504*t*
treatment, 65, 504
Hyperkalemia, postoperative, management, 64
Hyperlipidemia, in hypothyroidism, 476
Hyperparathyroidism, 495–499
bones, stones, and groans in, 496–497
definition, 495
familial, non-MEN, 498–499
familial isolated, 496
ICD-9 code, 499
nonrenal, ICD-9 code, 499
with osteopenia, in elderly, 81
primary, 495–499
clinical presentation, 496–497
epidemiology, 496
etiology, 496
evaluation, 497
ICD-9 code, 499
imaging, 497
laboratory investigation, 497–498
treatment
medical, 497–498
surgical, 498–499
secondary, 495, 499
ICD-9 code, 499
tertiary, 495, 499
types, 495
Hyperphosphatemia
hyperparathyroidism in, 499
hypoparathyroidism with, 500–501
Hypersomnia, ICD-9 code, 68
Hyperthyroidism, 470–474. *See also* Thyroid storm
clinical features, 470–471
definition, 470
etiology, 471–472
evaluation, 472
follow-up, 473
ICD-9 codes, 473–474
imaging, 472
iodine-induced, 472
laboratory investigation, 472
neonatal, ICD-9 code, 477
outcomes with, 473
secondary to increased thyroid hormone synthesis, 471–472
secondary to thyroiditis, 472
signs and symptoms, 470–471
treatment, 473
Hypocalcemia, 501–504
acute, treatment, 503
chronic, treatment, 503
clinical presentation, 502
etiology, 502
evaluation, 503
hyperparathyroidism in, 499
hypoparathyroidism with, 500–501
ICD-9 code, 504
postoperative, 65
treatment, 503
Hypofibrinogenemia, cryoprecipitate for, 52
Hypogeusia, definition, 257

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Hypoglossal nerve (cranial nerve XII), 309, 327, 328*f*, 575*f*, 694*f*
  functions, 691
  injury to, in neck dissection, 406
Hypoglycemia, postoperative, 60–61
Hyponatremia, in hypothyroidism, 476
Hypoparathyroidism, 500–501
  autoimmune, 500
  differential diagnosis, 500–501
  in DiGeorge syndrome, 500
  etiology, 500
  in hungry bone syndrome, 500
  iatrogenic (surgical), 500
  ICD-9 code, 501
  idiopathic, 500
  in infiltrative disease, 500
  radiation-related, 500
  signs and symptoms, 500
  in storage disorders, 500
  treatment, 501
Hypopharyngeal cancer, 383–388
  biopsy, 385
  clinical features, 384
  differential diagnosis, 384
  distant metastases, staging, 387
  epidemiology, 383
  evaluation, 384–385
  follow-up, 385–386
  history taking for, 384
  ICD-9 codes, 386
  imaging, 384
  laboratory studies in, 385
  outcomes with, 385
  pathology, 385
  physical examination of patient with, 384
  prognosis for, 385
  recurrence, monitoring for, 385–386
  regional lymph node metastases
    signs and symptoms, 384
    staging, 386–387
  signs and symptoms, 384
  stage groupings, 387
  staging, 386–387
  treatment, 385
Hypopharynx
  anatomy, 7–8, 266*f*, 383
  malignant neoplasm, ICD-9 code, 386
  posterior wall, malignant neoplasm, ICD-9 code, 386
Hypophysitis, hypothyroidism caused by, 475
Hypopnea, definition, 66
Hyposmia, definition, 254
Hypotension, trauma-related, 338
Hypothermia, transfusion-related, 54
Hypothyroidism
  after RAI therapy, 473
  central, 474, 475
    epidemiology, 475
  in children, 475
  clinical features, 475–476
  complications, 477
  congenital, 475
    ICD-9 code, 477
  definition, 474
  differential diagnosis, 476
  drug-related, 475
  epidemiology, 475
  follow-up, 477
  iatrogenic, 473, 474
  ICD-9 codes, 477
  in infants, 475
  laboratory investigation, 476
  laryngeal manifestations, 322
  outcomes with, 477
  postablative, 474
    ICD-9 code, 477
  postsurgical, 474
    ICD-9 code, 477
  primary, 474
    epidemiology, 475
    etiology, 474–475
  radiation-related, 358
  secondary, 474, 475
  signs and symptoms, 476
  subclinical, 474
  tertiary, 474, 475
  transient, 475
  treatment, 476–477
    in elderly, 477
    in heart disease, 477
    in pregnancy, 477
    in surgical patient, 477
Hypoxia
  and acquired prenatal hearing loss, 560
  postoperative
    causes, 59–60
    and mental status change, 59–60

I

Ibuprofen
  for migraine, 191, 192*t*
  for temporomandibular joint disorders, 75

STUDENTS-HUB.com Uploaded By: tuka abuzneid

ICF. *See* Intracellular fluid (ICF)
Idiopathic midline destructive disease (IMDD), 434–436. *See also* Lethal midline granuloma
  clinical features, 435
  differential diagnosis, 435
  epidemiology, 435
  Epstein-Barr virus and, 436
  evaluation, 435–436
  follow-up, 436
  ICD-9 code, 436
  imaging, 435
  outcomes with, 436
  pathology, 436
  physical examination of patient with, 435
  signs and symptoms, 435
  survival rates, 436
  treatment, 436
Ifosfamide, for head and neck cancer, 349
IGRT. *See* Image-guided radiotherapy (IGRT)
IHS. *See* International Headache Society (IHS)
Iliac crest bone graft(s), for facial reconstruction, 641
Image-guided radiotherapy(IGRT), 356
Imaging. *See also specific disorder; specific entity; specific modality*
  in acute otitis media, 118
  of cancer of unknown primary, 350
  and chronic otitis media, 122
  of cochlear implant candidate, 172
  diagnostic, of head and neck, 22–29
  in facial paralysis/paresis, 111
  of nasopharyngeal carcinoma, 366
  of new patient, reviewing, 5
  of temporal bone trauma, 106
Imbalance, 177
  tumors causing, 196
Imbibition, in skin graft healing, 627
IMDD. *See* Idiopathic midline destructive disease (IMDD)
Imipenem, for deep neck infection, 335
Imitrex. *See* Sumatriptan
Immittance testing, 149
  results, 151–153
Immune system, disorders, sinonasal manifestations, 259
Immune therapy, for head and neck cancer, 349
Immunocompromised patient(s), oral cancer in, 370
Immunodeficiency
  otologic manifestations, 208
  primary/congenital, otologic manifestations, 208
Immunosuppression
  and acute invasive fungal rhinosinusitis, 215, 218
  and oral cancer, 370
  transfusion-related, 53–54
Immunotherapy, for chronic allergy, 249
Implantable hearing devices, 174–176. *See also* Auditory brainstem implant (ABI); Bone-anchored hearing aid (BAHA); Middle ear implant (MEI)
Implants, alloplastic, 646–649
Impression, of new patient, 5
IMRT. *See* Intensity-modulated radiation therapy (IMRT)
Imu-Max, 90
Incudomalleolar joint, 99*f,* 100
Incudostapedial joint, 99*f,* 100
Incus
  anatomy, 98, 99*f*
  embryology, 95–97, 96*t*
Indole-3-carbinol
  adverse effects and side effects, 293
  dosage and administration, pediatric, 293
  for papillomatosis, 293
Infant(s)
  estimated blood volume, 51
  full-term, estimated blood volume, 51
  meningitis in, antibiotics for, 132*t*
  premature, estimated blood volume, 51
Infection(s)
  deep neck space. *See* Deep neck infection(s)
  and difficult airway, 16*t*
  hearing loss caused by, 166
  laryngeal, 280–285
  laser-related transmission, 85
  odontogenic, 331–333
  otologic manifestations, 204–206
  rhinogenic
    intracranial hematogenous spread, 212
    orbital spread, 211–212, 219–221
  sinonasal manifestations, 259
  transfusion-related, 54
  treatment, 262
Infection control, for *Clostridium difficile,* 64
Infectious organism(s), ICD-9 codes, 330

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Inferior jugular chain region, 325, 326*f*
Inflammation
  and facial paralysis/paresis, 112
  sinonasal manifestations, 259
Influenza, subacute thyroiditis caused by, 479
Influenza virus, sialadenitis caused by, 443–444
Infraorbital nerve, block, 33
Inner ear
  anatomy, 100–101, 101*f*
  autoimmune disease
    differential diagnosis, 180
    hearing loss caused by, 165
    and vertigo, 183
  embryology, 96*f*, 97
  injury, by foreign body, 114, 115
  vertigo and disequilibrium related to, 183
Inner hair cell(s), 101*f*
Innominate artery, anomalous, 525
  treatment, 528
Inosculation, in skin graft healing, 627
INR. *See* International normalized ratio (INR)
Insect(s), in ear, 114, 115
Intensity-modulated radiation therapy (IMRT), 356, 358
Interdental papillae, malignant neoplasm, ICD-9 code, 375
Interferon
  adverse effects and side effects, 293, 419
  hypothyroidism caused by, 475
  for melanoma, 419
  for papillomatosis, 293
  thyroiditis caused by, 482
Interleukin(s) (IL), IL-2
  hypothyroidism caused by, 475
  for melanoma, 419
  thyroiditis caused by, 482
Internal auditory canal
  anatomy, 101
  tumors, hearing loss caused by, 165
Internal carotid artery
  anatomy, 326
  branches, nasal blood supply, 212, 213*f*
International Headache Society (IHS), diagnostic criteria for migraine, 191*t*
International normalized ratio (INR), 56
Intertragic incisure, 97*f*
Intracellular fluid (ICF), 49
Intracranial pathology, vertigo and disequilibrium caused by, 183
Intracranial pressure, increased, postoperative, 200*t*
Intubation
  for airway compromise, in children, 507–508
  for angioedema, 249
  conscious (awake), 14
    indications for, 14
    preparation for, 14
    procedure for, 14
  difficulty, 274–275
    predicting, 9
  endotracheal. *See also* Endotracheal tube(s)
    complications, 12
    confirmation, 11
    cricoid pressure for, 12
    equipment for, 11
    indications for, 10–11
    pediatric, 11
    procedure for, 11–12
    tube size for, recommendations, 11
  nasotracheal, 13
  orotracheal, 12–13, 12*f*
    procedure for, 13
  rapid-sequence, 13
    indications for, 13
    procedure for, 13
  tracheal, fiberoptic-assisted, 14
    nasal technique for, 14
    oral technique for, 14
Inverted papilloma, 250–254
  biopsy, 252
  and cancer, 250
  clinical features, 250
  differential diagnosis, 251
  endoscopic removal, 252–253
  epidemiology, 250
  evaluation, 251
  history taking for, 251
  ICD-9 code, 253
  imaging, 251
  malignant transformation, 251, 360
  physical examination of patient with, 251
  recurrence, 253
  signs and symptoms, 250
  treatment, 251–253
    complications, 253
    follow-up, 253
    outcomes with, 253
    preoperative planning for, 252

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Iodine
deficiency, 475
and follicular thyroid cancer, 485
excess, 475
Iodine-131 (^{131}I). *See also* Radioactive iodine (RAI)
for brachytherapy, 356
for thyroid scintigraphy, 28
Iodine-induced hyperthyroidism, 472
Ioflupane (^{123}I), for thyroid scintigraphy, 28
Ipratropium bromide, for nonallergic rhinitis, 246
Isoflurane
adverse effects and side effects, 43
cardiovascular effects, 44*t*
neurologic effects, 44*t*
neuromuscular effects, 44*t*
pharmacology, 43
physiologic effects, 43, 44*t*
renal effects, 44*t*
respiratory effects, 44*t*

J

Jacobson's nerve, ear innervation by, 402
Jaw(s), upper, resection, 360, 361*f*
Jerger classification system, for tympanometry, 151–152, 151*f*
Jervell-Lange-Nielson syndrome, 557
evaluation, 563
hearing loss in, 165*t*
Jugular digastric region, 404
Juvederm. *See* Hyaluronic acid

K

Kallmann syndrome, anosmia in, 255
Kaposi sarcoma, otologic involvement, 208
Kartagener syndrome, diagnosis, 214*t*
Kayexalate enema, 64
Keloid, ICD-9 code, 646
Keratoacanthoma, 413
Keratoma. *See* Cholesteatoma
Ketamine
adverse effects and side effects, 42
cardiovascular effects, 42*t*
CNS effects, 42*t*
dosage and administration, 42
mechanism of action, 41
pharmacology, 41–42
physiologic effects, 41, 42*t*
respiratory effects, 42*t*
Khat, and head and neck cancer, 346
Kidney(s), isoflurane and, 44*t*
Kiesselbach plexus, 212, 213*f*, 229
Klebsiella pneumoniae
deep neck infection, 334
laryngitis caused by, 282
Klonopin. *See* Clonazepam
KTP laser. *See* Potassium titanyl phosphate (KTP) laser

L

Laboratory testing, of new patient, reviewing, 5
Labyrinthectomy, for Ménière disease, 185–186
Labyrinthine fistula, 180
Labyrinthitis, acute suppurative, 130–131
complications, 130
etiology, 130
pathology, 130
signs and symptoms, 130
treatment, 131
Lacrimal sac neoplasms, 251
Langerhans cell histiocytosis
otologic manifestations, 206
treatment, 206
Lansoprazole, 510
Larsen syndrome, 540
Laryngeal airway surgery
for aspiration, 314, 315*t*–316*t*
for bilateral vocal fold paralysis, 516
complications, 516
follow-up, 516
irreversible, 314, 315*t*
outcomes with, 516
reversible, 314, 315*t*–316*t*
Laryngeal anomalies, ICD-9 code, 511
Laryngeal cancer, 388–398
classification, by location, 389
clinical features, 389–390
differential diagnosis, 389–390
distant metastases, staging, 397
epidemiology, 389, 399
evaluation, 390–391
follow-up, 394–395, 401
glottic
signs and symptoms, 389
staging, 395–396
treatment, by stage, 391–393
history taking for, 390
ICD-9 codes, 395
imaging, 390
lymph node metastases, 343*f*
otalgia caused by, 117
outcomes with, 394–395, 401
pathology, 390–391

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Laryngeal cancer *(continued)*
physical examination of patient with, 390
regional lymph node metastases, staging, 396–397
signs and symptoms, 389
spread, patterns, 388
stage groupings, 397–398
staging, 395–397
subglottic
signs and symptoms, 389
staging, 396
treatment, by stage, 391–393
supraglottic
signs and symptoms, 389
staging, 395
treatment, by stage, 391–392
treatment
complications, 394
by stage, 391–393, 399
Laryngeal cartilages, malignant neoplasm, ICD-9 code, 395
Laryngeal clefts, 508, 517–520
associated abnormalities, 517, 518*f*
classification, 519
differential diagnosis, 518
epidemiology, 517
evaluation, 518–519, 518*f*
ICD-9 code, 520
imaging, 519
pathology, 519
signs and symptoms, 517
syndromic, 517
treatment, 519–520
complications, 520
follow-up, 520
outcomes with, 520
Laryngeal disorders, in elderly, 81
Laryngeal edema, radiation-related, 358
Laryngeal electromyography (LEMG), 288
Laryngeal emergency(ies), 272
Laryngeal fracture, 275–278
and airway obstruction, 276
clinical features, 276
differential diagnosis, 276
epidemiology, 276
evaluation, 276–277
follow-up, 277
ICD-9 codes, 277
laboratory investigation, 276
outcomes with, 277
physical examination of patient with, 276
severity, 276
signs and symptoms, 276
treatment, 277
work-up for, 276–277
Laryngeal infection(s), 280–285
causes, 282–283
differential diagnosis, 283–284
evaluation, 284
follow-up, 284
history taking in, 284
ICD-9 codes, 284
imaging in, 284
laboratory investigation, 284
outcomes with, 284
physical examination of patient with, 284
treatment, 284
Laryngeal mask airway (LMA), 15
complications, 15
contraindications to, 15
indications for, 15
insertion, 15
Laryngeal motility, assessment, 348
Laryngeal reinnervation, for bilateral vocal fold paralysis, 516
Laryngeal spasm, ICD-9 code, 290
Laryngeal tremor
pathophysiology, 289
treatment, 288–290
Laryngeal web, 508
Laryngectomy
aphonia after, 398–401
dysphonia after, 398–401
for laryngeal cancer, 391–394
narrow-field, 314, 315*t*
partial
for laryngeal cancer, 393–394
voice rehabilitation after, 400
supracricoid, for laryngeal cancer, 394
supraglottic, for laryngeal cancer, 394
total
for laryngeal cancer, 394
voice rehabilitation after, 400–401
Laryngitis
acute, ICD-9 code, 284
bacterial, signs and symptoms, 281–282
in children
clinical presentation, 281
diagnosis, 282
clinical presentation, 281
definition, 280
fungal, 282
infectious, 280–285
causes, 280
in leprosy, 283

STUDENTS-HUB.com Uploaded By: tuka abuzneid

syphilitic, 283
tuberculous, 282
viral
epidemiology, 281
signs and symptoms, 281
Laryngomalacia, 508, 510, 511–513
differential diagnosis, 512
epidemiology, 511
evaluation, 512
ICD-9 code, 513
imaging, 512
pathology, 512–513
physical examination of patient with, 512
and reflux, 512
signs and symptoms, 511
treatment
medical, 513
surgical, 513
Laryngopharyngeal reflux (LPR), 317–319
complications, 319
epidemiology, 317
evaluation, 318
outcomes with, 319
signs and symptoms, 317
treatment, 318–319
Laryngoplasty, vertical, 314, 316*t*
Laryngoscope(s)
intubating, 8, 12–13, 12*f*
Macintosh (curved blade), 8, 12*f*, 13
Miller (straight) blade, 8, 13
operative (surgical), 8, 16
rigid, fiberoptic video, 8, 15–16
types, 684
Laryngoscopy. *See also* Panendoscopy
direct
glottic view on, Cormack and Lehane grades, 10, 10*f*
in hypopharyngeal cancer, 385
in laryngeal cancer, 390
flexible fiberoptic, 285
indications for, 348
mirror, indications for, 348
rigid direct microscopic
complications, 684–685
contraindications to, 684
indications for, 684
with or without biopsy, 684
procedure for, 684
Laryngotracheal diversion, 314, 315*t*
Laryngotracheal separation, 314, 315*t*
Laryngotracheitis, signs and symptoms, 281
Laryngotracheoplasty, for subglottic stenosis, 532
Larynx
anatomy, 7–8, 267–270, 268*f*, 269*f*, 388–389
anomalies, ICD-9 code, 275
benign neoplasm, ICD-9 code, 294
blood supply to, 270
cartilages, 268, 269*f*
innervation, 8, 270
motor, 8
sensory, 8
lymphatic drainage, 270
malignant neoplasm, ICD-9 code, 395
musculature, 270
neurologic disorders involving, 285–291
paralysis. *See also* Vocal fold(s), motion impairment
ICD-9 code, 300
physiology, 270
skeleton, 268, 269*f*
soft tissue, 268–269
specified sites, malignant neoplasm, ICD-9 code, 395
subglottic, 388–389
supraglottic, 388–389
systemic disorders affecting, 319–322
unspecified, malignant neoplasm, ICD-9 code, 395
Laser(s), 82–86. *See also* Transoral laser microsurgery
adjacent thermal effects, 82–83
argon, 84
biophysics, 82–83
carbon dioxide, 83
and chromophores, 82
clinical applications, 83
depth effect, 82
diode, 84
dye, 84–85
endotracheal tube resistant to, 11
erbium,chromium:yttrium-scandium-gallium-garnet (Er,Cr:YSGG), 84
erbium:yttrium-aluminum-garnet (Er:YAG), 84
in facial plastic surgery, 83
fluence, 83
in general otolaryngology, 83
holmium:yttrium-aluminum-garnet (Ho:YAG), 84
in laryngology, 83
Neodymium:yttrium-aluminum-garnet (Nd:YAG), 84
in otology, 83

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Laser(s) *(continued)*
potassium titanyl phosphate (KTP), 84
precautions with, 47
residual char with, 83
safety considerations with, 85
therapy with
for epistaxis, 233
for head and neck cancer, 349
for papillomatosis, 294
for subglottic stenosis, 531
types, 83–85
Laser-assisted uvulopalatoplasty (LAUP), for obstructive sleep apnea, 68
Laser skin resurfacing, 656, 658–659
Lateral sinus thrombosis
imaging, 133
and otitic hydrocephalus, 134–135
otitis media–related, 133
etiology, 133
pathogenesis, 133
signs and symptoms, 133
treatment, 133
LAUP. *See* Laser-assisted uvulopalatoplasty (LAUP)
Lee Silverman Voice Treatment, 304
LeFort fractures, 610, 610*f*
LEMG. *See* Laryngeal electromyography (LEMG)
Lemierre syndrome, deep neck infection and, 335
Lenticular process, 98, 99*f*
Lentigo maligna, 418
Leprosy
laryngitis caused by, 283
nasal manifestations, 238
sinonasal manifestations, 259
Lesion(s), midline destructive, 434–436
Lesser occipital nerve, 328*f*
block, 33
innervation of auricle, 98
Lethal midline granuloma, 431*t*. *See also* Idiopathic midline destructive disease (IMDD)
diagnosis, 260*t*
ICD-9 code, 436
treatment, 260*t*
Leukemia, otologic manifestations, 207
Leukoplakia, 370. *See also* Proliferative verrucous leukoplakia (PVL)
and floor of mouth cancer, 374
Leukotriene inhibitors, for chronic allergy, 249
Levaquin, for chronic rhinosinusitis, 241
Levator palpebral superioris muscle, anatomy, 661
Levofloxacin, for acute rhinosinusitis, adult dosage and administration, 236
Levothroid, 476–477
Levothyroxine
replacement therapy, 476–477
for thyroiditis, 482
TSH suppression therapy, for thyroid cancer, 487
Levoxyl, 476–477
Licorice, 91
Lid(s). *See* Eyelid(s)
Lidocaine, topical, for awake intubation, 14
Lightheadedness, 177
Lingual nerve, injury to, in neck dissection, 406
Liothyronine, postoperative use, in thyroid cancer, 487
Lip(s). *See also* Cleft lip
cancer
pathology, 373
treatment, 373
embryology, 587–588
lower, carcinoma, lymph node metastases, 343*f*
malignant neoplasm, ICD-9 code, 375
melanoma, ICD-9 code, 420
other and ill-defined sites within, malignant neoplasm, ICD-9 code, 376
skin, other malignant neoplasm, ICD-9 code, 410, 414
Lipoma
congenital midline, 577–580
epidemiology, 578
pathology, 579
signs and symptoms, 578
treatment, 580
Liposuction, of face and neck, 675–677
complications, 676
contraindications to, 675
follow-up, 676
indications for, 675
outcomes with, 676
procedure for, 675–676
Lithium, thyroiditis caused by, 482
Lithium carbonate, hypothyroidism caused by, 475
Little area, 212, 213*f*, 229

STUDENTS-HUB.com Uploaded By: tuka abuzneid

LMA. *See* Laryngeal mask airway (LMA)
Loop diuretics, ototoxicity, 164, 164*t*
Lorazepam
  dosage and administration, 40
  pharmacology, 39–40
  preoperative, 10*t*
Lotrimin cream, for otitis externa, 143*t*
Lower jugular region, 325, 326*f*, 405
LPR. *See* Laryngopharyngeal reflux (LPR)
Ludwig angina, 331–333
  clinical features, 331
  complications, 332
  differential diagnosis, 331
  epidemiology, 331
  evaluation, 332
  ICD-9 code, 333
  imaging, 332
  laboratory investigation, 332
  mortality rate for, 331
  odontogenic, 331–333
  pathophysiology, 331
  physical examination of patient with, 332
  risk factors for, 331
  signs and symptoms, 331
  treatment
    medical, 332
    surgical, 332
Ludwig scale, for female pattern alopecia, 678
Lumbar drainage, for otitic hydrocephalus, 135
Lumbar puncture
  contraindications to, 134
  indications for, 223
  interpretation, 223, 224*t*
  in meningitis, 131
Lyme disease
  and facial paralysis, 110
  otologic manifestations, 205
  serology, indications for, 187, 194, 203
  treatment, 205
Lyme serology, indications for, 103
Lymphadenitis. *See also* Adenitis
  acute, ICD-9 code, 569
  cervical
    acute unilateral, 566
    in children, 565
      etiology, 566
Lymphangiomas. *See also* Lymphatic malformation(s)
  ICD-9 code, 573
Lymphatic malformation(s). *See also* Lymphangiomas
  in children, 569–573
    differential diagnosis, 571
    epidemiology, 570
    evaluation, 571
    outcomes with, 572
    signs and symptoms, 571
    treatment, 572
Lymphatic tissue, malignant neoplasm, ICD-9 code, 434
Lymph node(s)
  cervical, cancer metastases to, 347, 347*t*
  of head and neck, 343*f*
Lymphoepithelial-like carcinoma, oropharyngeal, 380
Lymphoid tissue, malignant neoplasm, ICD-9 code, 434
Lymphoma(s), 428–434. *See also* Hodgkin lymphoma (HL); Non-Hodgkin lymphoma (NHL)
  angiocentric, 431*t*
  bone marrow biopsy in, 432
  diffuse large cell, diagnostic criteria for, 430*t*
  evaluation, 429–432
  fine-needle aspiration biopsy, 432
  follicular, diagnostic criteria for, 430*t*
  follow-up, 433–434
  history taking for, 429
  ICD-9 codes, 434
  and idiopathic lethal midline destructive disease, 434
  imaging, 432
  laboratory investigation, 432
  mantle cell, 431*t*
  marginal zone (monocytoid), 431*t*
  nasal T-cell, 431*t*
  natural killer cell or T-cell (NK/T-cell)
    extranodal, nasal type, 435
    sinonasal, 434
  neck mass caused by, in children, 565
  oropharyngeal, 380
  outcomes with, 433–434
  pathology, 432
  physical examination of patient with, 432
  primary cutaneous anaplastic large cell, 431*t*
  primary system C, 431*t*
  salivary gland involvement in, 453
  small lymphocytic, diagnostic criteria for, 430*t*

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Lymphoma(s) *(continued)*
of temporal bone, 425
thyroid, 484*t*
cellular origin, 484*t*
clinical presentation, 490
diagnosis, 490–491
epidemiology, 484*t*, 490
staging, 491
treatment, 484*t*, 491
treatment, 432–433
Lymphosarcoma, ICD-9 code, 434

M

MAC. *See* Minimum alveolar concentration (MAC)
Macroglossia, 508
Macrolides
for acute otitis media, 119
for acute rhinosinusitis
adult dosage and administration, 235, 236*t*
pediatric dosage and administration, 236
Magnetic resonance angiography (MRA), 24–25
Magnetic resonance imaging (MRI), 24–25, 251
in acute invasive fungal rhinosinusitis, 216
advantages and disadvantages, 24
of cancer of unknown primary, 350
of chronic rhinosinusitis, 239
of cochlear implant candidate, 172
contraindications to, 25
contrast-enhanced, 24
with CSF rhinorrhea, 226, 227*t*
of deep neck infection, 334
in facial paralysis/paresis, 111
gadolinium-enhanced, 24
of head and neck carcinoma, 348
of hypopharyngeal cancer, 384
indications for, 24, 103, 118, 146, 146*t*, 256
of intranasal mass, 231
of nasopharyngeal carcinoma, 366
of neck mass, 342
of oral cancer, 371
of orbits, in hyperthyroidism, 472
principles, 25
of thyroid nodule, 468
time to demagnetize (spin relaxation time; T2), 25*t*
time to echo (TE), 25*t*
time to inversion (TI), 25*t*
time to magnetize (spin lattice relaxation time; T1), 25*t*
time to repetition (TR), 25*t*
of vascular ring, 527
Magnetic resonance venography (MRV), for lateral sinus thrombosis, 133
Ma huang, 91
Major salivary gland(s)
benign neoplasm, ICD-9 code, 450
ICD-9 code, 450
malignant neoplasm, ICD-9 code, 456
Malar fractures, ICD-9 codes, 605
Malaria, transfusion-related, 54
Malignancy
hypercalcemia caused by, 503–504
second, radiation-related, 358
sinonasal, 359–364
Malignant hyperthermia (MH), 47–48
complications, 47–48
pathophysiology, 47
prevention, 47
signs, 47, 47*t*
treatment, 48, 48*t*
Malignant melanoma. *See* Melanoma
Malignant otitis externa (MOE)
clinical features, 145
complications, 145, 148
diagnostic tests for, 146, 146*t*
differential diagnosis, 145
epidemiology, 145
evaluation, 146, 147
follow-up, 148
fungal, 145, 147
history taking for, 146
ICD-9 code, 148
imaging in, 146–147, 146*t*
laboratory findings in, 147
outcomes with, 148
pathology, 147
physical findings in, 146
risk factors for, 145, 146
signs and symptoms, 145
treatment, 145, 147–148
medical, 147–148, 148*t*
Mallampti classification, 9, 9*f*
Malleus
anatomy, 98, 99*f*
anterior process, 98, 99*f*
embryology, 95–97, 96*t*
head, 98, 99*f*
lateral process, 98, 99*f*

STUDENTS-HUB.com Uploaded By: tuka abuzneid

manubrium, 98, 99*f*
neck, 98, 99*f*
Malocclusion
dentoalveolar fractures and, 611
fractures causing, 603
mandibular fractures and, 615, 617
midface fractures and, 611, 613
Mandible, anatomy, 616*f*
Mandibular dislocation, 77
Mandibular distraction osteogenesis, for airway obstruction in Pierre Robin sequence, 536
Mandibular fracture(s), 614–620
causes, 614–615
classification, 614, 615*f*
complications, 618
differential diagnosis, 616
epidemiology, 614–615
evaluation, 616–617
favorable (for healing), 614, 615*f*
follow-up, 618–619
ICD-9 codes, 619
imaging, 616
laboratory investigation, 616–617
open reduction and internal fixation, 617–618
outcomes with, 618–619
pathology, 617
physical examination of patient with, 616
signs and symptoms, 615
sites, 615, 616*f*
surgery for, 617–618
treatment, 617–618
unfavorable (for healing), 614, 615*f*
Mandibular nerve (V_3), 690
ear innervation by, 402
Mandibulomaxillary advancement, for obstructive sleep apnea, 68
Manual circumlaryngeal reposturing, 304
Marshall syndrome, 540
Massive transfusion, and dilutional thrombocytopenia, 55
Massive transfusion(s), 52
and coagulopathy, 54
Mastoidectomy
canal wall down, indications for, 128–129
cortical, indications for, 127, 128, 129
indications for, 131, 132
for lateral sinus thrombosis, 133
for subdural empyema, 134
Mastoiditis
acute coalescent, acute otitis media and, 117, 118
coalescent
imaging, 129
otitis media and, 129
physical findings in, 129
progression, time course, 129
signs and symptoms, 129
treatment, 129
otitis media and, 127, 128
Maxalt. *See* Rizatriptan
Maxillary artery, 212, 213*f*
Maxillary fracture, ICD-9 codes, 605
Maxillary nerve (V_2), 690
Maxillary puncture, for acute rhinosinusitis, 236
Maxillary sinus, 211, 212*f*
cancer, 359. *See also* Sinonasal cancer
staging, 362
treatment, 360, 361*f*
malignant neoplasm, ICD-9 code, 361
Maxillectomy, open medial, for inverted papilloma, 252
Maxillomandibular fixation (MMF)
for mandibular fractures, 617–618
nonrigid, 617–618
rigid, 617–618
for midface fractures, 612
Measles, otologic manifestations, 205
Meckel cartilage, 95, 96*t*
Median nasal prominences (MNPs), 587–588
Mediastinitis, deep neck infection and, 335
Medication(s)
nephrotoxic, 164
ototoxic, 164, 164*t*
hearing loss caused by, 164
otoprotective strategies for, 167
and tinnitus, 194, 195
preoperative, 10, 10*t*
Medication history, for elderly patient, 79
Medication reconciliation, for elderly patient, 79
Medrol, for chronic rhinosinusitis, 240–241, 241*t*
Medrol dose pack
for Ménière disease, 185
for vestibular neuritis, 188, 188*t*
MEI. *See* Middle ear implant (MEI)
Melanoma, 360
ABCDE mnemonic for, 417
acral lentiginous, 418

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Melanoma *(continued)*
American Joint Committee on Cancer (AJCC) classification, 419, 421–422
auricular, 424
Breslow classification, 419, 420
Clark classification, 419, 420
clinical features, 417–418
cutaneous
prognosis for, 419–420
stage groupings, 422
staging, 420–422
differential diagnosis, 417–418
of ear, 425
epidemiology, 417
evaluation, 418
of head and neck, 416–423
epidemiology, 416–417
pathology, 416–417
ICD-9 codes, 420
imaging, 418
lentigo maligna, 418
metastases
laboratory diagnosis, 418
treatment, 419
mortality rate for, 417
mucosal, 251, 417
American Joint Committee on Cancer (AJCC) staging, 423
clinical course, 417
diagnosis, 417
differential diagnosis, 238
metastases, 417, 420
prognosis for, 417
recurrence, 420
staging, 422–423
survival rate for, 417
nodular, 418
outcomes with, 419
pathology, 418
physical examination of patient with, 418
recurrence, 419
risk factors for, 417
sentinel lymph node biopsy in, 419
signs and symptoms, 417
staging, 418–419
superficial spreading, 418
of temporal bone, 425
treatment, 417, 418–419, 427
medical, 419
surgical, 419
work-up for, 418
Melatonin, for tinnitus, 195
Melkersson-Rosenthal syndrome, and facial paralysis, 109
Membranous labyrinth
anatomy, 100
embryology, 97
Ménière disease
audiogram in, 151
audiograms in, 184–185
clinical features, 183
cochleovestibular, ICD-9 code, 186
diagnosis, 159, 183, 184*t*
differential diagnosis, 180, 183
epidemiology, 176, 183
follow-up, 186
hearing loss in, 165, 183
ICD-9 codes, 186
imaging in, 184
outcomes with, 186
pathology, 185
physical findings in, 184
serology in, 184
signs and symptoms, 183
tinnitus caused by, 183
treatment, 167, 183
medical, 185
surgical, 185–186
vertigo in, 183
vestibular, ICD-9 code, 186
Meningioma
ear canal, 425
hearing loss caused by, 165
imaging, 196
Meningitis
acute rhinosinusitis and, 237
acute suppurative labyrinthitis and, 130, 131
antibiotic prophylaxis, with repair of congenital nasal mass, 583
bacterial, signs and symptoms, 222
and cochlear implant, 172
with cochlear implant, 173
deep neck infection and, 335
due to unspecified bacterium, ICD-9 code, 224
hearing loss caused by, 166, 561
imaging, 131
of nonrhinologic origin, 222
otitis media–related, 131
pathology, 131
postoperative, 61, 200*t*
of rhinologic origin, 222
signs and symptoms, 131

STUDENTS-HUB.com Uploaded By: tuka abuzneid

sinusitis and, 222
treatment, 61, 131, 132*t*
work-up for, 61
Meningocele
congenital nasal, 581–584
ICD-9 code, 584
Mental status change, postoperative, 59–61
Meperidine
dosage and administration, 38
pharmacology, 37–38
preoperative, 10*t*
Meropenem, for deep neck infection, 335
Metabolic disorders
otologic manifestations, 207
transfusion-related, 54
Metabolic status, preoperative assessment, 6*t*
Metastatic disease
of ear and temporal bone, 426
hearing loss caused by, 165
otologic manifestations, 207
salivary gland involvement in, 453
of temporal bone, 425
Methicillin-resistant *Staphylococcus aureus* (MRSA), 241, 241*t*
chronic otitis media caused by, 123
Methimazole
for hyperthyroidism, 473
hypothyroidism caused by, 475
Methohexital
dosage and administration, 43
pharmacology, 43
Methotrexate
for head and neck cancer, 349
mechanism of action, 353
for thyroiditis, 482
Methoxyisobutylisonitrile (MIBI). *See* Technetium-99m sestamibi
Methylprednisolone, for vestibular neuritis, 188, 188*t*
Metoclopramide, 510
preoperative, 10*t*
for reflux, 318
Metronidazole, for deep neck infection, 335
MH. *See* Malignant hyperthermia (MH)
MIBI. *See* Technetium-99m sestamibi
Michel aplasia, 555
Micrognathia, in Pierre Robin sequence, 534
Microsmia, definition, 254
Microtia, 627, 665
Microvascular free tissue transfer, for facial reconstruction, 636–639
Midazolam
adverse effects and side effects, 39
for awake intubation, 14
dosage and administration, 39
pharmacology, 39
preoperative, 10*t*
Middle cervical fascia, 325*f*
Middle ear
anatomy, 98
basal cell carcinoma, 424–427
embryology, 95–97
injury, by foreign body, 114, 115
pathology, acute otitis media and, 119
persistent serous effusion, 121
squamous cell carcinoma, 424–427
surgery, for conductive hearing loss, 161
Middle ear implant (MEI)
principles, 175
use rate, 175
Middle ear space, boundaries, 98
Middle jugular region, 325, 326*f,* 404
Midface
buttresses, 609, 609*f*
masticatory forces transmitted through, 609, 609*f*
Midface fractures, 609–614
adjacent injuries, evaluation for, 611
complications, 613
epidemiology, 610
evaluation, 611–612
follow-up, 613
ICD-9 codes, 613
imaging, 611
LeFort classification, 610, 610*f*
outcomes with, 613
physical examination of patient with, 611
signs and symptoms, 610–611
treatment, 612
work-up for, 611–612
Midline destructive lesions. *See* Idiopathic midline destructive disease
Midline lethal granuloma. *See* T-cell lymphoma
Migraine
abortive medical management, 191–192, 192*t*
aura, 190
clinical features, 190
diagnostic criteria for, 191*t*
epidemiology, 189–190
hearing loss caused by, 165
history taking for, 190–191

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Migraine *(continued)*
perimenstrual, 190, 191
treatment, 192, 192*t*
prevention (prophylaxis), 191–192, 192*t*
recurrent, differential diagnosis, 234, 238
signs and symptoms, 190
variants, ICD-9 code, 192
vertigo and disequilibrium caused by, 183, 189–193
Migrainous vestibulopathy, 180
Millard rotation advancement flap repair, for cleft lip, 590*f*, 591
Miller syndrome, 540
Minimum alveolar concentration (MAC), 43
Minor salivary gland(s)
benign neoplasm, ICD-9 code, 450
biopsy, in Sjögren syndrome, 445
carcinoma, oropharyngeal, 380
malignancy, 360
neoplasms/tumors, 251
Minoxidil, 677, 678–679
MIT. *See* Monoiodotyrosine (MIT)
Mitomycin
mechanism of action, 353
as radiation sensitizer, 358
MMF. *See* Maxillomandibularfixation (MMF)
MNPs. *See* Median nasal prominences (MNPs)
Modified barium swallow (MBS), 27
Modiolus, 100
MOE. *See* Malignant otitis externa (MOE)
Moebius syndrome, 540
Mohs micrographic excision, 413, 414*t*
for basal cell carcinoma, 409, 410*f*
Mondini dysplasia, 555
Mondini malformation, 165
and cochlear implant, 172
in DiGeorge syndrome, 208
Monoiodotyrosine (MIT), 463
Moraxella catarrhalis
acute otitis media caused by, 119
rhinosinusitis caused by, 235
Morphine
adverse effects and side effects, 36
dosage and administration, 36
pharmacology, 36
preoperative, 10*t*
Mouth. *See also* Floor of mouth; Oral cavity
anatomy, 7–8
other and unspecified parts, malignant neoplasm, ICD-9 code, 376
Mouth opening, assessment, 9
Moxifloxacin, for acute rhinosinusitis, adult dosage and administration, 236, 236*t*
MPS. *See* Mucopolysaccharidosis (MPS)
MRI. *See* Magnetic resonance imaging (MRI)
MRSA. *See* Methicillin-resistant *Staphylococcus aureus* (MRSA)
MRV. *See* Magnetic resonance venography (MRV)
Mucocele
ethmoid, 220
salivary gland, ICD-9 code, 447
Mucociliary clearance, 213
Mucoepidermoid carcinoma, salivary gland, 451, 453
Mucopolysaccharidosis (MPS), 540–541
otologic manifestations, 207
pathophysiology, 207
Mucor, identification, 218
Mucormycosis. *See also* Acute invasive fungal rhinosinusitis
ICD-9 code, 218
rhinocerebral. *See* Acute invasive fungal rhinosinusitis
Mucosa-associated lymphoid tissue (MALT), lymphoma, diagnostic criteria for, 430*t*
Mucositis, radiation-related, 358
Multiple endocrine neoplasia
and hyperparathyroidism, 496, 496*t*
medullary thyroid cancer in, 489
type 1, 496, 496*t*, 499
type 2A, 496, 496*t*, 499
type 2B, 496, 496*t*
Multiple myeloma
otologic manifestations, 206
pathology, 206
Multiple sclerosis (MS)
hearing loss in, 166
laryngeal manifestations, 322
Mumps
otologic manifestations, 205
sialadenitis caused by, 443–444
subacute thyroiditis caused by, 479
treatment, 205
Mupirocin, nasal irrigation, 241, 241*t*
Muscle enzyme(s), serum, high, thyroid function testing with, 476
Muscle relaxant(s). *See also* Neuromuscular blocking drugs
adverse effects and side effects, 76
for temporomandibular joint disorders, 76

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Muscle relaxation, 30
  intraoperative, agents for, 44–46
Muscle transfer(s), for facial reanimation, 624
Myasthenia gravis (MG), laryngeal manifestations, 322
Mycetoma, clinical course, 215
Mycobacteria
  infection
    laboratory investigation, 261
    sinonasal manifestations, 259
  nontuberculous, lymphadenitis caused by, in children, 566
  thyroiditis caused by, 481
Mycobacterium tuberculosis. *See* Tuberculosis
Myocardial infarct/infarction, parathyroid, 497
Myocutaneous flap(s), for facial reconstruction, 637–638
Myringitis, bullous, differential diagnosis, 142
Myringotomy
  indications for, 127, 128, 129, 130
  for meningitis, 131
Myxedema, 477
  pretibial, 471
Myxedema coma, 477

N

Nager syndrome, 541
  and Pierre Robin sequence, 534
Naloxone
  dosage and administration, 38
  pharmacology, 38
Naproxen, for temporomandibular joint disorders, 75
NAR. *See* Nonallergic rhinitis (NAR)
Naratriptan, for migraine, 192*t*
Narcotics, and aspiration, preoperative, 10*t*
NARES. *See* Nonallergic rhinitis with eosinophilia syndrome
Nasal biopsy, 216
Nasal bone, 211, 211*f*
  closed fracture. *See also* Nasal fracture; Naso-orbito-ethmoid (NOE) fractures
    ICD-9 code, 599, 602
  open fracture. *See also* Nasal fracture; Naso-orbito-ethmoid (NOE) fractures
    ICD-9 code, 599, 602
Nasal cartilage(s), 211, 211*f*
Nasal cavity
  benign neoplasm, ICD-9 code, 253
  cancer. *See also* Sinonasal cancer
    staging, 362
  vasculature, 212, 213*f*
Nasal fracture, 597–599. *See also* Naso-orbito-ethmoid (NOE) fractures
  clinical presentation, 597
  closed manipulation, 598
  epidemiology, 597
  evaluation, 597–598
  follow-up, 599
  formal septorhinoplasty for, 599
  ICD-9 codes, 599
  mechanism of injury, 597
  modified open reduction with osteotomies, 598
  open nasal/septal repair, 598
  outcomes with, 599
  treatment, 598–599
Nasal lavage study(ies), 245
Nasal mass
  congenital midline, 581–584
    clinical features, 581–582
    differential diagnosis, 582
    epidemiology, 581
    evaluation, 582–583, 582*t*
    pathology, 583
    physical examination of patient with, 582
    signs and symptoms, 581–582
  unilateral, 250
Nasal mucosa
  in allergic rhinitis, 245
  in granulomatous rhinitis, 245
  innervation, 7
  in irritative rhinitis, 245
  in nonallergic rhinitis, 245
  in rhinitis medicamentosa, 245
Nasal obstruction
  causes, 259, 260*t*
  unilateral, 250
Nasal packing, for epistaxis, 232
Nasal physiology, 213, 214*t*
Nasal septum, 211, 211*f*
  anatomy, 673
  deviated, 672–674
    ICD-9 code, 599
    treatment, 672–674
Nasal skeleton, 211, 211*f*
Nasofrontal angle, 669, 669*f*, 670
Nasogastric (NG) tube, indications for, 48, 62

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Nasolabial angle, 669, 669*f*, 670
Nasolacrimal duct, 211
Nasolaryngoscopy, flexible fiberoptic, 4
Naso-orbito-ethmoid (NOE) fractures, 599–602
  classification, 601
  CSF leak with, 601
  differential diagnosis, 600
  epidemiology, 600
  evaluation, 600–601
  follow-up, 601
  history taking for, 600
  ICD-9 codes, 602
  imaging, 601
  laboratory investigation, 601
  mechanism of injury, 600
  outcomes with, 601
  physical examination of patient with, 600–601
  signs and symptoms, 600
  treatment, 601
Nasopharyngeal carcinoma
  Epstein-Barr virus and, 346
  keratinizing, 366
  nonkeratinizing, 366
  pathology, 366
  poorly differentiated, 366
  treatment, 353
  types, 366
  undifferentiated, 366
  World Health Organization (WHO) classification, 366
Nasopharyngeal carcinoma (NPC), 364–369
  clinical features, 365
  differential diagnosis, 365
  distant metastases, staging, 368
  endemic, 364
  epidemiology, 364–365
  Epstein-Barr virus and, 364, 366, 367
  evaluation, 365–366
  follow-up, 367
  history taking for, 365
  HLA association, 366
  ICD-9 code, 367
  imaging, 366
  metastases, 365
  outcomes with, 367
  pathogenesis, 364
  physical examination of patient with, 365
  prognosis for, 367
  radiation therapy for, 366–367
  regional lymph node metastases, staging, 368
  signs and symptoms, 365
  stage groupings, 368–369
  staging, 367–368
  treatment, 366–367
Nasopharynx
  anatomy, 266*f*, 364
  malignant neoplasm, ICD-9 code, 367
Nasotracheal intubation. *See* Intubation, nasotracheal
Nausea and vomiting. *See also* Postoperative nausea and vomiting (PONV)
  radiation-related, 358
  in vestibular disorders, 177
Nd:YAG laser. *See* Neodymium:yttrium-aluminum-garnet (Nd:YAG) laser
Neck. *See also* Head and neck
  anatomy, 325–328
  blood supply, 326–327, 327*f*
  inadequate extension, and difficult airway, 16*t*
  injury, ICD-9 code, 341
  innervation, 327, 328*f*
  levels, 325, 326*f*, 404–405
  lymphatic drainage, 326–327
  melanoma, ICD-9 code, 420
  nerve blocks, 33–35
  nodal levels, 325, 326*f*
  skin, other malignant neoplasm, ICD-9 code, 410, 415
  trauma, 337–341
    blunt, 337, 338
    clinical features, 337–338
    differential diagnosis, 338
    epidemiology, 276, 337
    evaluation, 338–339
    follow-up, 341
    history taking in, 338
    ICD-9 codes, 341
    imaging, 339
    laboratory investigation, 339
    outcomes with, 341
    penetrating, 337, 338, 339*t*, 340, 340*t*
    physical findings in, 338
    signs and symptoms, 337–338
    symptoms, 338
    treatment, 339–340
  venous drainage, 326
  zones, 338, 339*t*. *See also* Neck, levels
    trauma in, management, 338, 339*t*, 340
Neck dissection, 403–407. *See also* Radical neck dissection (RND); Selective neck dissection (SND)
  anterior compartment, 406
  central compartment, 406

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chylous fistula after, 62–63
classification, 405–406
complications, 406
intraoperative, 406
postoperative, 406
historical perspective on, 404
indications for, 404
lateral compartment, 406
modified, 405
posterior lateral, 406
supraomohyoid, 405
Neck exploration, injuries sought and treated during, 340, 340*t*
Neck mass(es), 341–345, 440
in adults, 341
age and, 341
biopsy, 345
in children, 341
clinical features, 342
congenital
differential diagnosis, 344*t*
location, 342
congenital midline, 577–580
clinical presentation, 578
differential diagnosis, 578
evaluation, 578–579
imaging, 579
pathology, 579
physical examination of patient with, 578
differential diagnosis, 341, 344*t*
evaluation, 342–345
fine-needle aspiration biopsy, 390
history taking for, 342
ICD-9 code, 345, 569
imaging, 342
infectious
in children, 565–569
complications, 568–569
differential diagnosis, 566
evaluation, 566–567
follow-up, 569
ICD-9 codes, 569
imaging, 567
laboratory investigation, 567
microbiology, 566
outcomes with, 569
physical examination of patient with, 566
signs and symptoms, 566
surgery for, 568–569
treatment, 567–568
work-up for, 567
differential diagnosis, 344*t*
inflammatory, differential diagnosis, 344*t*
laboratory investigation, 344
lymph node metastases, 343*f*
malignant
differential diagnosis, 344*t*
location, 342
neoplastic, differential diagnosis, 344*t*
painful, 342
painless, 342
physical examination of patient with, 342
signs and symptoms, 342
in systemic disease, differential diagnosis, 344*t*
treatment
medical, 345
surgical, 345
Necrotizing fasciitis, 328–330
ICD-9 code, 330
Neisseria, deep neck infection, 334
Neodymium:yttrium-aluminum-garnet (Nd:YAG) laser, 84
Neonate(s), meningitis in, antibiotics for, 132*t*
Neoplasm(s)
benign, of nasal cavity, ICD-9 code, 253
epistaxis caused by, 230*t*
facial asymmetry (paralysis) caused by, 110
intranasal, differential diagnosis, 238
otologic manifestations, 206–207
Neostigmine, 46
Neo-Synephrine
for awake intubation, 14
for decongestion of nose, for nasolaryngoscopy, 4
for nasal examination, 231
Neovascularization, in skin graft healing, 627
Nephrotoxicity, 164
Nerve block(s). *See also* Regional anesthesia
of neck, 33–35
of scalp and face, 32–33
Nerve of Galen, 461
Nervous system, neoplasm, ICD-9 code, 442
Nervus intermedius, 690
Neurilemoma, 440
Neurofibroma, 251. *See also* Peripheral nerve sheath tumor (PNSTs)
epidemiology, 440

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Neurofibromatosis (NF)
clinical features, 558
and hearing loss, 558
ICD-9 code, 442
type 1, 440, 558
ICD-9 code, 442
type 2, 196, 558
ICD-9 code, 201
vestibular schwannoma in, treatment, 197, 199
Neurolaryngeal disorders, 285–291
clinical features, 285–286
differential diagnosis, 286
in elderly, 81
history taking in, 286
signs and symptoms, 285–286
treatment, 288–290
medical, 288–289
work-up for, 286–288
Neurolaryngeal examination, 285
Neurologic disorders. *See also* Cranial neuropathy(ies)
hearing loss caused by, 166
laryngeal involvement in, 285–291
trauma-related, 338
Neuromuscular blocking drugs, 44–46. *See also specific drug*
clinical applications, 44–45
depolarizing, 45, 45*t*. *See also* Succinylcholine
nondepolarizing, 45–46, 46*t*. *See also specific agent*
clinical considerations with, 46*t*
dosage and administration, 46*t*
mechanism of action, 45–46
pharmacology, 45–46
reversal, 46
Neuromuscular disorders
aspiration in, 312
laryngeal manifestations, 322
Neuromuscular system, isoflurane and, 44*t*
Neurotransmitter(s)
barbiturates and, 43
benzodiazepines and, 38
Neutropenia, and acute invasive fungal rhinosinusitis, 215, 218
NF1. *See* Neurofibromatosis (NF), type 1
NF2. *See* Neurofibromatosis (NF), type 2
NG tube. *See* Nasogastric (NG) tube
NHL. *See* Non-Hodgkin lymphoma (NHL)
Nitrous oxide
pharmacology, 44
physiologic effects, 44
Nodule(s)
thyroid. *See* Thyroid nodule(s)
vocal fold, 295–297
NOE. *See* Naso-orbito-ethmoid (NOE) fractures
Nonallergic rhinitis (NAR), 243–246
classification, 244–245
clinical features, 243–244
differential diagnosis, 244
epidemiology, 243
evaluation, 245
follow-up, 246
history taking in, 245
ICD-9 code, 246
laboratory investigation, 245
outcomes with, 246
physical findings in, 245
signs and symptoms, 243–244
subtypes, 244–245
treatment
medical, 245–246
surgical, 246
work-up for, 245
Nonallergic rhinitis with eosinophilia syndrome, 245
treatment, 246
Non-Hodgkin lymphoma (NHL), 428–434. *See also* Lymphoma(s)
biopsy, 433
diagnostic criteria for, 430*t*–431*t*
epidemiology, 428
of midface, 434
salivary gland involvement in, 453
staging, 429*t*
treatment, 433
Nonsteroidal anti-inflammatory drugs (NSAIDs)
and epistaxis, 230
preoperative, 10*t*
rhinitis caused by, 244
Noonan syndrome, 541
Norketamine, 42
Norrie syndrome, 559
Nortriptyline, for migraine, 192*t*
Norwood scale, for male pattern alopecia, 678
Nose
acquired deformity, ICD-9 code, 672
anatomy, 7–8, 211–212, 211*f*, 669
blood supply to, 212, 213*f*, 229
congenital deformity. *See also* Nasal mass, congenital midline
ICD-9 code, 584
innervation, 212–213

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Nosebleed. *See* Epistaxis
Nothing by mouth (NPO). *See* NPO status
NPC. *See* Nasopharyngeal carcinoma (NPC)
NPO status
  American Society of Anesthesiologists guidelines for, 6*t*
  for aspiration, 314
  and fluid management, 48–50
  indications for, postoperative, 62, 63
NPO status, preoperative assessment, 6*t*
Nuclear medicine, 27–29
Nutritional supplements, 91
Nystagmus, 202
  fatiguing, 179, 180
  geotropic rotatory, 179
  latency, 179
  in vestibular neuritis, 187

O

OAEs. *See* Otoacoustic emissions (OAEs)
Obesity, and difficult airway, 16*t*
Obstructive sleep apnea (OSA), 65–69
  in children, 510, 549–553
  clinical features, 66
  diagnosis, 67
  differential diagnosis, 66
  epidemiology, 66
  evaluation, 66–67
  follow-up, 68
  history taking for, 66
  ICD-9 codes, 68
  imaging in, 67
  outcomes with, 68
  physical findings in, 66
  prognosis for, 68
  sequelae, 68
  signs and symptoms, 66
  treatment, 67–68
    medical, 67–68
    surgical, 68
  work-up for, 67
Obstructive uropathy, postoperative, 64
Occlusion class(es), 617
Ochronosis
  otologic manifestations, 207
  pathophysiology, 207
Oculomotor nerve (cranial nerve III), 327
  function, 690
Odontogenic cyst(s), 69–71
Odontogenic disorders, 69–71
  clinical features, 69–70
  complications, 70
  differential diagnosis, 70
  epidemiology, 69
  follow-up, 70
  outcomes with, 70
  signs and symptoms, 69–70
  treatment, 70
OE. *See* Otitis externa (OE)
Ofloxacin, topical, for otitis externa, 144
Öhngren's plane, 361, 361*f*
Olfactory disorders, 254–257
  causes, 254, 255
  differential diagnosis, 255
  epidemiology, 254
  evaluation, 255–256
  iatrogenic, 255
  ICD-9 codes, 256
  imaging in, 255–256
  objective testing for, 255
  signs and symptoms, 254–255
Olfactory loss. *See* Anosmia
Olfactory nerve (cranial nerve I), 212, 254, 327
  function, 690
  nasal innervation by, 212
Olfactory testing
  indications for, 239
  objective, 255
OME. *See* Otitis media with effusion (OME)
Oncocytoma, salivary gland, 448*t*, 449
Open reduction and internal fixation (ORIF), of mandibular fractures, 617–618
Ophthalmic artery, 212, 213*f*
Ophthalmic nerve (V_1), 690
Ophthalmic vein(s), 212
Ophthalmoplegia, 220
Opioid(s). *See also specific drug*
  cardiovascular effects, 37*t*
  CNS effects, 37*t*
  drug interactions, 37*t*
  endocrine effects, 37*t*
  gastrointestinal effects, 37*t*
  mechanism of action, 36
  pharmacodynamics, 36
  physiologic effects, 37*t*
  respiratory effects, 37*t*
  reversal, 38
Opioid receptor(s), 36, 38*t*
Opitz G syndrome, 541
Optic nerve (cranial nerve II), 220, 232, 253, 327, 604, 606
  function, 690
Optiz-Frias syndrome, laryngeal clefts in, 517

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Oral appliances, for obstructive sleep apnea, 68
Oral cavity
  anatomy, 265, 369
  blood supply to, 265
  cancer, 369–377
    advanced, 375
    biopsy, 371
    differential diagnosis, 370
    distant metastases, staging, 376
    epidemiology, 369–370
    evaluation, 370–372
    follow-up, 375
    grading, 372, 372*t*
    history taking for, 370
    ICD-9 codes, 375
    imaging, 371
    outcomes with, 375
    pathology, 372
    physical examination of patient with, 371
    regional lymph node metastases, staging, 376
    risk factors for, 370
    screening for, 371
    signs and symptoms, 370
    stage groupings, 377
    staging, 376
    survival rates, 369–370
    treatment, 373–375
  caustic injury, 278–280
  focal epithelial hyperplasia, 71–73
    clinical features, 71–72
    differential diagnosis, 72
    epidemiology, 71
    evaluation, 72
    follow-up, 73
    ICD-9 code, 73
    outcomes with, 73
    signs and symptoms, 71–72
    treatment, 72
  innervation, 265
  lymphatic drainage, 265
  malignant neoplasm, ICD-9 code, 375
  musculature, 265
  opening, Mallampti classification, 9, 9*f*
  other and ill-defined sites within, malignant neoplasm, ICD-9 code, 376
  physiology, 266
Oral contraceptives, rhinitis caused by, 244
Oral pathology, benign, 71–73
Orbicularis muscles, 660–661
Orbital apex syndrome, 220
Orbital decompression, for Graves ophthalmopathy, 473
Orbital disease, Chandler classification, 219, 219*t*
Orbital edema, sinonasal causes, 220
Orbital fat compartments, 662
Orbital floor blowout, 602–605
  closed, ICD-9 code, 602
  open, ICD-9 code, 602
  treatment, 604
Orbital fractures, 602–605
  complications, 604
  differential diagnosis, 603
  evaluation, 603–604
  follow-up, 604
  imaging, 604
  outcomes with, 604
  physical examination of patient with, 603
  signs and symptoms, 603
  treatment, 604
Orbital septum, anatomy, 661
Organ of Corti, 100
ORIF. *See* Open reduction and internal fixation (ORIF)
Oro-facial-digital syndrome, 541–542
  type I, 541–542
  type II, 541–542
Oropharyngeal cancer, 378–383
  biopsy, 379
  clinical features, 378–379
  clinical presentation, 378
  differential diagnosis, 379
  distant metastases, staging, 381
  epidemiology, 378
  evaluation, 379–380
  follow-up, 380
  history taking for, 379
  ICD-9 codes, 380–381
  imaging, 379
  outcomes with, 380
  pathology, 380
  physical examination of patient with, 379
  regional lymph node metastases, staging, 381
  risk factors for, 378
  signs and symptoms, 378
  stage groupings, 382
  staging, 381
  treatment, 380

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Oropharyngeal disorders, in elderly, 81
Oropharynx
anatomy, 7–8, 266*f*, 378
innervation, 8
lateral wall, malignant neoplasia, ICD-9 code, 381
malignant neoplasia, ICD-9 code, 380
posterior wall, malignant neoplasia, ICD-9 code, 381
Orotracheal intubation. *See* Intubation, orotracheal
OSA. *See* Obstructive sleep apnea (OSA)
Oscillopsia, 177, 202
Osler-Weber-Rendu disease, treatment, 233
Ossicles
anatomy, 98–100, 99*f*
embryology, 95–97, 96*t*
hearing loss related to, 160
Ossicular chain, 99*f*
disease
hearing loss caused by, 161–162
prostheses for, 162
disruption, hearing loss caused by, 160
reconstruction, 161–162
Osteitis deformans. *See* Paget disease
Osteogenesis imperfecta, 559
otologic manifestations, 207
pathophysiology, 207
Osteoma
differential diagnosis, 238
ear canal, 425
Osteomyelitis
mandibular, deep neck infection and, 335
skull base, imaging, 146–147, 146*t*
vertebral, deep neck infection and, 335
Osteopetrosis
otologic manifestations, 208
pathophysiology, 208
Osteoradionecrosis, 329, 358
Osteosarcoma, 360
Otalgia. *See also* Ear(s), pain
in acute otitis media, 117
associated symptoms, 403
evaluation, 402–403
history taking for, 402–403
ICD-9 codes, 403
physical examination of patient with, 403
referred, 117, 401–403
definition, 401
etiology, 402, 402*t*
ICD-9 code, 403
treatment, 403
unspecified, ICD-9 code, 403
Otic capsule, 97
Otic placode, 97
Otic vesicle, 402
Otitic hydrocephalus. *See* Hydrocephalus, otitic
Otitis, serous, in elderly, 80
Otitis externa (OE)
acute, 141
epidemiology, 141
chronic, treatment, 144
clinical features, 141–142
differential diagnosis, 117, 118, 142
evaluation, 142–143
follow-up, 144
ICD-9 code, 144
imaging, 142
malignant. *See also* Malignant otitis externa (MOE)
facial asymmetry (paralysis) caused by, 110
microbiology, 143
necrotizing
in diabetes, 142
facial asymmetry (paralysis) caused by, 110
outcomes with, 144
pathology, 143
physical findings in, 142
signs and symptoms, 141–142
treatment, medical, 143–144, 143*t*
Otitis media. *See also* Chronic suppurative otitis media (CSOM)
acute. *See* Acute otitis media (AOM)
bacterial, 116
chronic
with cholesteatoma, facial asymmetry (paralysis) caused by, 110
and facial paralysis, 110
complications, 117, 118
with effusion, Pierre Robin sequence and, 534
tuberculous, 122
unilateral, serous, with nasopharyngeal carcinoma, 365
viral, 116
Otitis media with effusion (OME), 116
differential diagnosis, 117

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Otoacoustic emissions (OAE)
assessment procedure, 158
indications for, 167
clinical applications, 158
clinical significance, 158
factors affecting, 158
norms, 158
Otoconia, 100
Otocyst, 96*f*, 97
Otogenic pain, referred, ICD-9 code, 403
Otolaryngology referral, red flags for, 155, 169, 169*t*
Otolith(s), 179, 180
Otomycosis, signs and symptoms, 141–142
Oto-palatal-digital syndrome, 542, 543, 559, 560
Otoplasty, 665–668
complications, 667–668
evaluation for, 666
follow-up, 668
outcomes with, 668
surgical techniques, 666–667
Otorrhea
cerebrospinal fluid, treatment, 108
with cholesteatoma, 136
cholesteatoma and, 136
in chronic otitis media, 121
painless, with tympanic membrane perforation, 121, 122
with tympanostomy tubes, 120
Otosclerosis
cochlear, 165
definition, 161
epidemiology, 161
hearing loss caused by, 161
treatment, 161
Ototoxicity, 164, 164*t*
hearing loss caused by, 164
protection against, 167
and tinnitus, 194, 195
Oto-Wick, indications for, 115
Outer ear, surgery, for conductive hearing loss, 161
Outer hair cell(s), 101*f*
Oxacillin, for orbital complications of sinusitis, 221
Oxymetazoline
for acute rhinosinusitis, 236
for chronic rhinosinusitis, 241
for decongestion of nose, 231
for nasolaryngoscopy, 4
for orbital complications of sinusitis, 221

P

Paan
and head and neck cancer, 346
and oral cancer, 370
and oropharyngeal cancer, 378
Packed red blood cells (PRBCs)
group O Rh-negative, 52
transfusion guidelines, 50, 51
Paclitaxel, mechanism of action, 354
Paget disease
otologic manifestations, 207
pathophysiology, 207
temporal bone, hearing loss caused by, 165
Palatal flap, 593, 594*f*
Palate. *See also* Cleft palate
embryology, 587–588
hard. *See* Hard palate
Palate lengthening procedure, 592, 593, 593*f*
Pallister-Hall syndrome, laryngeal clefts in, 517
Palpebral spring implant, 625
Pamelor. *See* Nortriptyline
Panax quinquefolius. *See* Ginseng
Pancuronium
clinical considerations with, 46*t*
dosage and administration, 46*t*
pharmacology, 46*t*
Panendoscopy
in head and neck cancer, 348–349
in laryngeal cancer, 390
in oral cancer, 371
in oropharyngeal cancer, 380
of upper aerodigestive tract, indications for, 345
Papillary cystadenoma lymphomatosum. *See* Warthin tumor
Papilledema, 220
Papilloma(s)
anatomic distribution, 291
cylindrical, 251
fungiform, 251
intranasal, differential diagnosis, 238
inverted. *See* Inverted papilloma
laryngeal, 291–294
adult onset, 291
epidemiology, 291
juvenile, 291
pathology, 293
squamous, oral, 71–73
differential diagnosis, 72
evaluation, 72
follow-up, 73

STUDENTS-HUB.com Uploaded By: tuka abuzneid

ICD-9 code, 73
outcomes with, 73
signs and symptoms, 72
treatment, 72
surgery for, 294
treatment, 293–294
Papillomatosis, 291–294
Paraganglia, 437
malignant neoplasms, ICD-9 code, 439
neoplasm of uncertain behavior, ICD-9 code, 439
Paraganglioma(s), 437–439
clinical features, 437
differential diagnosis, 437
ear canal, 425
epidemiology, 437
evaluation, 438
follow-up, 439
functional (catecholamine-secreting), 438
ICD-9 codes, 439
imaging, 438
laboratory investigation, 438
outcomes with, 439
pathogenesis, 437
pathology, 438
physical examination of patient with, 438
recurrence, 439
signs and symptoms, 437
sites, in head and neck, 437
treatment, 438–439
Paraglottic space, 389
Parainfluenza virus, laryngitis caused by, 281, 282
Paranasal sinus(es)
anatomy, 211–212, 212*f*
cancer. *See also* Sinonasal cancer
risk factors for, 359
physiology, 213
Parasitic disease, transfusion-related, 54
Parathyrin. *See* Parathyroid hormone (PTH)
Parathyroid adenoma
ectopic, 498
resection, 498
Parathyroid carcinoma, 497
Parathyroidectomy, 498–499
Parathyroid gland(s)
anatomy, 494
blood supply, 495
embryology, 494
functions, 495
histology, 494
physiology, 495
Parathyroid hormone (PTH), 495
and calcium metabolism, 502
recombinant, 501
serum
in hyperparathyroidism, 497–498
in hypoparathyroidism, 500–501
Parathyroid scintigraphy, 28–29
Parkinson disease, 285
ICD-9 code, 290, 322
laryngeal manifestations, 322
neurolaryngeal involvement in, treatment, 288–290
pathophysiology, 289
Parosmia, 256
definition, 254
Parotidectomy
for benign tumor, 449
complications, 450, 455
for malignant tumor, 454
Parotid gland
carcinoma, lymph node metastases, 344*f*
malignant neoplasm, ICD-9 code, 456
neoplasm, in elderly, 81
Pars sacculocochlearis, 96*f*
Pars utriculovestibularis, 96*f*
Partial thromboplastin time (PTT), 55
PCP. *See* Phencyclidine (PCP)
PDT. *See* Photodynamic therapy (PDT)
PDT (percutaneous dilation tracheotomy). *See* Tracheotomy, percutaneous dilation
PE. *See* Pulmonary embolism
PEG tube. *See* Percutaneous endoscopic gastrostomy (PEG) tube
Pelargonium sidoides, 90
Pemphigoid, laryngeal manifestations, 321
Pendred syndrome, 557
evaluation, 563
hearing loss in, 165*t*
laboratory investigation, 562
Penetrating trauma, to neck, 276
Penicillins, for deep neck infection, 335
Peptostreptococcus, deep neck infection, 334
Percutaneous dilation tracheotomy (PDT). *See* Tracheotomy, percutaneous dilation
Percutaneous endoscopic gastrostomy (PEG) tube, 48
indications for, 62
Perilymph, 100
Perilymphatic fistula
causes, 166
hearing loss caused by, 166

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Perilymphatic fistula *(continued)*
treatment of, 178
vertigo caused by, 183
Perilymph fistula, 180
hearing loss caused by, 165
Periodontal disease, differential diagnosis, 234, 238
Periodontitis, apical, epidemiology, 69
Periotic duct, 100
Peripheral nerve sheath tumor (PNSTs), 439–442. *See also* Neurofibroma; Schwannoma
benign, 440
clinical features, 440
differential diagnosis, 440
epidemiology, 440
evaluation, 441
history taking for, 441
ICD-9 codes, 442
imaging, 441
malignant, 440
epithelioid, 441
glandular, 441
mesenchymal, 441
treatment, 442
pathology, 441
physical examination of patient with, 441
signs and symptoms, 440
treatment, 441–442
Peripheral vascular system, anomalies, ICD-9 code, 573
Petadolex, 90
Petasin, 90
Petasites spp. *See* Butterbur
PET-CT. *See* Positron emission tomography (PET) with computed tomography (PET-CT)
Petrous apex, pneumatization, 129
Petrous apicitis
etiology, 129
imaging, 129
signs and symptoms, 129
treatment, 130
Petrous bone, fractures, 104
pH, blood, decrease, transfusion-related, 54
Phantosmia, definition, 254
Pharyngeal pouch, ICD-9 code, 307
Pharyngocutaneous fistula, postoperative, 62
Pharyngoesophageal injury, traumatic, signs, 338
Pharynx
anatomy, 266–267, 266*f*
blood supply to, 267
caustic injury, 278–280
diverticulum. *See also* Zenker diverticulum (pl., diverticula)
ICD-9 code, 307
innervation, 267
lymphatic drainage, 267
malignant neoplasm, ICD-9 code, 375
musculature, 267
other and ill-defined sites within, malignant neoplasm, ICD-9 code, 376
posterior, innervation, 8
Phencyclidine (PCP), 42
Phenergan. *See* Promethazine
Phenol. *See* Chemical peels
Pheochromocytoma, 438
Phosphodiesterase inhibitors, rhinitis caused by, 244
Photodynamic agents, in diagnosis of oral cancer, 371
Photodynamic therapy (PDT)
dye laser for, 85
for head and neck cancer, 349
for papillomatosis, 293
Phrenic nerve, 327, 328*f*
injury to, in neck dissection, 406
Phycomycosis, ICD-9 code, 218
Physical examination
of elderly patient, 79
of head and neck surgery patient, 3, 3*t*
of new patient, 3, 3*t*
of otolaryngology patient, 3–4, 3*t*
Pierre Robin sequence, 533–537, 542, 588
and airway obstruction, treatment, 535–536, 535*t*
associated anomalies, 533, 534
differential diagnosis, 534
epidemiology, 534
evaluation, 534–535
follow-up, 536
ICD-9 codes, 537
imaging, 535
laboratory investigation, 535
outcomes with, 536
pathology, 535
physical examination of patient with, 534–535
signs and symptoms, 533, 534

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Pituitary adenoma(s)
hypothyroidism caused by, 475
TSH-producing, 472
Pituitary apoplexy, hypothyroidism caused by, 475
Pivotal flap(s). *See* Cutaneous flap(s), pivotal
Plain film radiographs
in dysphagia, 309
of necrotizing soft tissue infection, 329
in rhinosinusitis, 235
Plan of care, for new patient, 5
Plasmacytoma, of temporal bone, 206
Platelet count, normal, 51
Platelet function, evaluation, 56
Platelet transfusion(s), 51–52
Platinum compounds
ototoxicity, 164, 164*t*
as radiation sensitizers, 358
Plavix, and epistaxis, 230
Pleomorphic adenoma
minor salivary gland, 448, 448*t*
parotid, 448, 448*t*
salivary gland, 448, 448*t*
pathology, 449
treatment, 449
Plummer-Vinson syndrome, and hypopharyngeal cancer, 383
Pneumococcus, antibiotic resistance in, 119
Pneumocystis, thyroiditis caused by, 481
Pneumocystis carinii, otologic involvement, 208
Pneumonia
aspiration. *See also* Aspiration
ICD-9 code, 316
postoperative, treatment, 59
PNSTs. *See* Peripheral nerve sheath tumor (PNSTs)
Polyarteritis nodosa
otologic manifestations, 206
treatment, 206
Poly-L-lactic acid, 648*t*
Polyp(s)
with cholesteatoma, 136, 137
chronic otitis media and, 121, 122
nasal, inflammatory, 251
vocal fold, 295–297
Polysomnography (PSG), 67
in children, 510
indications for, 551
Pontocaine, for nasal examination, 231
Porphyromonas, deep neck infection, 334
Porus acousticus, 101
Positron emission tomography (PET), of nasopharyngeal carcinoma, 366
Positron emission tomography (PET) with computed tomography (PET-CT), 27–28
Postaxial acrofacial dysostosis. *See* Miller syndrome
Postcricoid region, 383
cancer, 383
malignant neoplasm, ICD-9 code, 386
Posterior canal occlusion, for benign paroxysmal positional vertigo, 182
Posterior hypopharyngeal wall, 383
cancer, 383
malignant neoplasm, ICD-9 code, 386
Posterior triangle (of neck), 325, 326*f,* 405
Postoperative nausea and vomiting (PONV), prevention, 30
Postoperative problem(s), 58–65
Postpolio syndrome, laryngeal manifestations, 322
Potassium
daily requirements for, 49
plasma/serum, increase, transfusion-related, 54
Potassium titanyl phosphate (KTP) laser, 84
PRBCs. *See* Packed red blood cells (PRBCs)
Prednisone
for Bell palsy, 113
for sudden sensorineural hearing loss, 103
Preepiglottic fat, laryngeal cancer spread to, 388
Preepiglottic space, 389
Pregnancy, rhinitis in, 244
Prematurity, hearing loss caused by, 554
Preoperative assessment, 5, 6*t,* 7*t*
Preoperative medication(s), 10, 10*t*
Presbycusis, 154, 163
audiogram in, 150–151
in elderly, 80
treatment, 167
Presbylarynx, in elderly, 81
Presbyosmia, 254, 256
in elderly, 81
epidemiology, 254
Prevacid. *See* Lansoprazole

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Prevotella, deep neck infection, 334
Probiotics
  for allergic rhinitis, 90
  for upper respiratory infections, 90
Procaine, topical intranasal, for nasolaryngoscopy, 4
Prokinetics, for reflux, 318
Proliferative verrucous leukoplakia (PVL), 372
Promethazine, for vestibular neuritis, 188, 188*t*
Propecia. *See* Finasteride
Propofol
  advantages, 40–41
  adverse effects and side effects, 41
  cardiovascular effects, 41*t*
  CNS effects, 41*t*
  dosage and administration, 41
  pharmacology, 40–41
  physiologic effects, 41*t*
  respiratory effects, 41*t*
Propranolol, for migraine, 191, 192*t*
Proptosis
  differential diagnosis, 220
  in Graves disease, 471, 472
  sinonasal causes, 220
Propylthiouracil (PTU)
  for hyperthyroidism, 473
  hypothyroidism caused by, 475
Prothrombin time (PT), 56
Proton pump inhibitors, for reflux, 318
Protriptyline, for migraine, 192*t*
Prussak's space, 98
Pseudoephedrine, for acute rhinosinusitis, 236
Pseudohypoparathyroidism, 502
Pseudomonas aeruginosa
  antibiotic resistance in, 147–148
  and chronic otitis media, 123
  malignant otitis externa caused by, 145, 147
  otitis externa caused by, 143
Pseudotumor, orbital, 220
PSG. *See* Polysomnography (PSG)
Psychiatric disorders, postoperative, 61
Psychosis, postoperative, 61
PT. *See* Prothrombin time (PT)
Pterygopalatine ganglion, nasal innervation by, 212–213
PTH. *See* Parathyroid hormone (PTH)
PTT. *See* Partial thromboplastin time (PTT)
PTU. *See* Propylthiouracil (PTU)
Pulmicort, for chronic rhinosinusitis, 241, 241*t*
Pulmonary artery sling, 525
  treatment, 528
Pulmonary embolism, postoperative, 60
Pulmonary function testing
  in children, 509–510
  with vascular ring, 527
Purpura, posttransfusion, 53
PVL. *See* Proliferative verrucous leukoplakia (PVL)
Pyriform sinus(es), 383
  cancer, 383
  malignant neoplasm, ICD-9 code, 386

Q

Quadrangular membrane, 268–269
Queckenstedt's test, for lateral sinus thrombosis, 133
Quinine, ototoxicity, 164*t*
Quinsy, ICD-9 code, 336

R

Raccoon eyes, 105
rad (unit), 355
Radial forearm free flap, for facial reconstruction, 637–638
Radiation dose, definition, 355
Radiation/radiation therapy/radiotherapy
  accelerated, 356, 358
  adjuvant, rationale for, 357
  adverse effects and side effects, 358
  for anaplastic thyroid cancer, 489
  for basal cell carcinoma, 409
  complications, 358
  definitive (curative), rationale for, 357
  delivery, 356–357
  external beam, 356–357
  fractionation, 355–356
  head and neck, and thyroid cancer risk, 484–486
  for head and neck cancer, 349, 355–358
  for Hodgkin lymphoma, 432
  hyperfractionated, 356, 358
  hypofractionated, 356, 358
  for idiopathic midline destructive disease, 436
  for laryngeal cancer, 393
  for malignant salivary gland tumors, 454
  mechanism of action, 355
  for nasopharyngeal carcinoma, 366–367
  for non-Hodgkin lymphoma, 433

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for oropharyngeal cancer, 380
palliative, rationale for, 357
planning, 356–357
principles, 355–356
for squamous cell carcinoma, 413, 414*t*
therapeutic ratio, improving, 358
Radiation sensitizers, 358
Radical neck dissection (RND)
classification, 405–406
extended, 406
historical perspective on, 404
Radicular cyst(s), 69
Radiesse. *See* Calcium hydroxylapatite
Radioactive iodine (RAI)
ablation, of thyroid remnant, 487
thyroiditis caused by, 481
Radioactive iodine (RAI) ablation
for Graves disease, 473
for hyperthyroidism, 473
Radioactive iodine (RAI) uptake, 465, 465*t,* 468, 472
Radioactive pledget scanning, 226–227, 227*t*
Radioallergosorbent test (RAST), 214*t,* 248–249
Radionuclide(s)
for parathyroid scintigraphy, 28
for thyroid scintigraphy, 28
RAE. *See* Ring-Adair-Elwyn (RAE) endotracheal tube
RAI. *See* Radioactive iodine (RAI)
Rainbow Passage, 286
Ramsay-Hunt syndrome, and facial paralysis, 109
Ranula, 447
Rapid plasma reagin (RPR) test, 214*t*
indications for, 103
RAS. *See* Reticular activating system (RAS)
RAST. *See* Radioallergosorbent test (RAST)
RDI. *See* Respiratory disturbance index (RDI)
Recurrent laryngeal nerve, 693*f*
airway innervation by, 7–8
identification, in thyroid surgery, 458, 459, 459*t*
injury, iatrogenic, 520, 528
intraoperative integrity, monitoring, 11
laryngeal cancer spread to, 388
thyroid innervation by, 461
Recurrent respiratory papillomatosis (RRP), 291–294
differential diagnosis, 292
epidemiology, 291–292
evaluation, 292–293
follow-up, 294
imaging, 292
laboratory investigation, 293
outcomes with, 294
pathology, 293
physical examination of patient with, 292
signs and symptoms, 292
surgery for, 294
treatment, 293–294
Red blood cell(s), for transfusion, DPG in, decreased, 54
Red flags, for otolaryngology referral, 155, 169, 169*t*
Reflux
airway irritation by, in children, 510
in elderly, 81
Reflux esophagitis, 317–319
Reflux Symptom Severity Index, 318
Regional anesthesia, 30. *See also* Nerve block(s)
for awake intubation, 14
benefits, 32
complications, 32
contraindications to, 32
Reglan. *See* Metoclopramide
Rehabilitation, auditory, for children, 156–157
Reichert cartilage, 95, 96*t,* 97
Reissner's membrane. *See* Vestibular (Reissner's) membrane
Relapsing polychondritis
diagnosis, 206, 260*t*
laryngeal manifestations, 320
otologic manifestations, 206
sinonasal manifestations, 261
treatment, 206, 260*t*
Relaxed skin tension lines (RSTLs), of face, 630, 643, 643*f*
Remifentanil
dosage and administration, 37
pharmacology, 36–37
Renal failure, postoperative, 64
Respiratory distress, pediatric
differential diagnosis, 508–509
evaluation, 509
follow-up, 510
history taking in, 509
ICD-9 codes, 511
imaging, 509
laryngeal sources, 508
outcomes with, 510

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Respiratory distress *(continued)*
physical examination of patient with, 509
signs and symptoms, 507
subjective assessment, 507–508, 508*t*
supralaryngeal sources, 508
tracheobronchial sources, 508–509
treatment, 510
Respiratory disturbance index (RDI), 67
Respiratory insufficiency, acute, postoperative, 59–60
Respiratory papillomatosis, 291–294
recurrent, 291–294
Respiratory system
benzodiazepines and, 39*t*
congenital anomalies, ICD-9 code, 537
etomidate and, 42*t*
isoflurane and, 44*t*
ketamine and, 42*t*
opioids and, 37*t*
preoperative assessment, 6*t*
propofol and, 41*t*
Restylane. *See* Hyaluronic acid
Reticular activating system (RAS), suppression, postoperative, 200*t*
Reticulosarcoma, ICD-9 code, 434
RET protooncogene testing, 489
Retromolar trigone, cancer, 374–375
Rhabdomyosarcoma, orbital, 220
Rheumatic disease, diagnosis, 261
Rheumatoid arthritis (RA)
ICD-9 code, 322
laryngeal manifestations, 320
otologic manifestations, 206
treatment, 206
Rheumatoid factor, testing, indications for, 103
Rheumatoid factor (RF), hyperparathyroidism in, 499
Rhinitis
allergic. *See* Allergic rhinitis (AR)
chronic, ICD-9 code, 246
definition, 243
drug-induced, 244
in elderly, 81
granulomatous, autoimmune, 246
gustatory, 244
hormonal, 244
ICD-9 code, 249
mixed, 243
nonallergic. *See* Nonallergic rhinitis (NAR)
occupational (irritative-toxic), 244
of pregnancy, 244
vasomotor. *See* Vasomotor rhinitis (VMR)
Rhinitis medicamentosa, 236, 238, 241
pathogenesis, 244
pathophysiology, 244
Rhinocerebral mucormycosis. *See* Acute invasive fungal rhinosinusitis
Rhinologic emergency(ies), 215–233
Rhinomanometry, 214*t*
indications for, 551
Rhinoplasty, 668–672
anatomical considerations in, 669, 669*f*
approaches for, 670–671
complications, 672
contraindications to, 670
follow-up, 672
incisions for, 670
nasal analysis for, 669–670
outcomes with, 672
patient motivations for, 668
surgical techniques for, 671–672
Rhinorrhea
cerebrospinal fluid. *See* Cerebrospinal fluid (CSF), rhinorrhea
clear, differential diagnosis, 225
watery and thin, differential diagnosis, 225
Rhinoscleroma, 238, 259
diagnosis, 260*t*
treatment, 260*t*, 262
Rhinosinusitis
acute, 233–237
clinical features, 234
definition, 237
differential diagnosis, 234
epidemiology, 234
evaluation, 234–235
follow-up, 237
ICD-9 codes, 237
imaging, 235
intracranial complications, 237
major and minor diagnostic factors, 234
microbiology, 235
outcomes with, 236–237
physical examination of patient with, 234–235
recurrent, 236–237
signs and symptoms, 234
treatment, 233, 235–236
acute invasive fungal. *See* Acute invasive fungal rhinosinusitis

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allergic fungal, 215, 239, 240, 251
chronic. *See* Chronic rhinosinusitis (CRS)
chronic invasive (indolent) fungal, 215
diagnosis, 238
epidemiology, 234, 238
fungal, 239
acute invasive. *See* Acute invasive fungal rhinosinusitis
allergic, 215
chronic invasive (indolent), 215
differential diagnosis, 234
subacute, definition, 237
Rhinosporidiosis, 238, 259
diagnosis, 260*t*
treatment, 260*t*, 262
Rhytidectomy, 649–652
approaches for, 651
complications, 651–652
evaluation for, 650–651
follow-up, 651
outcomes with, 651
postoperative care for, 651
secondary surgery after, 651
techniques for, 651
tuck-ups after, 651
Rickettsioses, transfusion-related, 54
Rifampin, for acute rhinosinusitis, adult dosage and administration, 236
Ring-Adair-Elwyn (RAE) endotracheal tube, 11
Rinne test, 152, 154*f*
in conductive hearing loss, 160
Risk stratification, preoperative, 5
Rizatriptan, for migraine, 192*t*
RND. *See* Radical neck dissection (RND)
Robotic-assisted surgery, 86–89
advantages and disadvantages, 88
in neurotology, 88
Rocaltrol, for hypocalcemia, 65
Rocuronium
clinical considerations with, 46*t*
dosage and administration, 46*t*
pharmacology, 46*t*
Rogaine. *See* Minoxidil
Romberg test, in vestibular neuritis, 187
Rosenmüller's fossa, 364
Roseola, lymphadenitis caused by, in children, 566
Rotational chair tests, 177
RPR. *See* Rapid plasma reagin (RPR) test
RRP. *See* Recurrent respiratory papillomatosis (RRP)
RSTLs. *See* Relaxed skin tension lines (RSTLs)
Rubella
and acquired prenatal hearing loss, 560
laboratory diagnosis, 563
Rubeola. *See* Measles
RX-130 robot, 88

S

Saccule, 100
Saddle nose deformity, 260*t*
treatment, 262
Saline, nasal
for acute rhinosinusitis, 236
for chronic rhinosinusitis, 241
Salivary calculi. *See* Sialolithiasis
Salivary disturbance. *See* Xerostomia
Salivary fistula, after neck dissection, 406
Salivary gland(s). *See also* Major salivary gland(s); Minor salivary gland(s)
abscess, ICD-9 code, 447
atrophy, ICD-9 code, 447
benign tumors, 447–450
classification, 448, 448*t*
clinical features, 448
complications, 449
differential diagnosis, 448
evaluation, 448–449
fine-needle aspiration biopsy, 449
follow-up, 450
history taking for, 448
ICD-9 codes, 450
imaging, 449
laboratory investigation, 449
outcomes with, 450
pathology, 449
physical examination of patient with, 449
signs and symptoms, 448
treatment, 449
disease, 442–447
diagnosis, flowchart for, 443*f*
ICD-9 code, 450
ICD-9 codes, 447
infectious, 443–445
inflammatory, 445–446
noninflammatory, 446–447
fistula, ICD-9 code, 447
hypertrophy, ICD-9 code, 447
malignant tumors, 451–457
classification, 452*t*
clinical features, 453

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Salivary gland(s) *(continued)*
differential diagnosis, 453
distant metastases, staging, 457
epidemiology, 451–453
evaluation, 453–454
fine-needle aspiration biopsy, 454
follow-up, 455
histologic grading, 454, 455*t*
history taking for, 453
ICD-9 codes, 456
imaging, 454
outcomes with, 455
pathology, 454
physical examination of patient with, 454
prognosis for, 455
regional lymph node metastases, staging, 456
signs and symptoms, 453
stage groupings, 457
staging, 456–457
treatment
medical, 454
surgical, 454
metastases to, 453
mucocele, ICD-9 code, 447
swelling
acute, differential diagnosis, 444*f*
chronic, differential diagnosis, 444*f*
tumors, differential diagnosis, 238
Salivary secretion, disturbance, ICD-9 code, 447
Salivary stones. *See* Sialolithiasis
Salmonellosis, transfusion-related, 54
Samter triad, 240
Sarcoid, nasal manifestations, 238
differential diagnosis, 234
Sarcoidosis
diagnosis, 260*t*
ICD-9 code, 322
laboratory investigation, 261
laryngeal manifestations, 320–321
otologic manifestations, 205
sinonasal manifestations, 259
treatment, 205, 260*t*, 262
Sarcoma
differential diagnosis, 238
of ear and temporal bone, 426
of temporal bone, 425
Scala media, 100. *See also* Cochlear duct
Scala tympani, 100, 101*f*
Scala vestibuli, 100, 101*f*
Scalp
anatomy of, 653–654
melanoma, ICD-9 code, 420
nerve block, 32–33
other malignant neoplasm, ICD-9 code, 410, 415
Scalp flap(s), for hair rearrangement, 679
Scalp reduction surgery, 679
Scaphoid fossa, 97*f*
Scar/scarring
formation, unfavorable, etiology, 644, 644*t*
hypertrophic
after rhytidectomy, 652
ICD-9 code, 646
ICD-9 codes, 646
revision, 643–646
outcomes with, 645
Z-plasty for, 645, 645*f*
SCC. *See* Squamous cell carcinoma (SCC)
SCDS. *See* Superior semicircular canal dehiscence syndrome (SCDS)
Scheibe aplasia, 556
Schwannoma, 251. *See also* Peripheral nerve sheath tumor (PNSTs)
Antoni A, 197, 441
Antoni B, 197, 441
differential diagnosis, 238
ear canal, 425
epidemiology, 440
ICD-9 code, 442
pathology, 441
Schweckendiek repair, for cleft palate, 592
Scintigraphy
parathyroid, 28–29
thyroid, 28
Sculptra. *See* Poly-L-lactic acid
SDET. *See* Serial dilution endpoint titration (SDET)
Sedation, 29
for awake intubation, 14
Selective neck dissection (SND), 405
Selective venous sampling, parathyroid, 497
Semicircular canal(s)
anatomy, 100
embryology, 96*f*
fistula, ICD-9 code, 204
horizontal (lateral), 100, 101
and labyrinthine fistula, 128
posterior, 100
superior (anterior), 100
dehiscence, 100

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Semicircular duct(s), 101
Sensorineural hearing loss (SNHL)
  acute-onset, with acute suppurative labyrinthitis, 130
  asymmetric, 163
    differential diagnosis, 165–166
  audiogram in, 150–151, 150*f*
  with cholesteatoma, 137
  congenital, 164–165, 554, 555–559
    autosomal dominant disorders causing, 557–559
    autosomal recessive disorders causing, 556–557
    dysmorphologies causing, 555–556
    evaluation, 562–563, 563*t*
    inherited, 556–559
    X-linked, 559
  evaluation, 152–154, 166
  follow-up, 168
  hereditary, 554
    nonsyndromic, 164–165
    syndromic, 164–165, 165*t*
  history taking in, 166
  ICD-9 codes, 168, 171
  infectious causes, 166
  metabolic causes, 165
  neurologic causes, 166
  outcomes with, 168
  pediatric
    acquired postnatal, 561–562
    epidemiology, 554
    trauma-related, 561
  signs and symptoms, 163
  sudden, 102–104
    audiologic assessment, 103
    causes, 102
    clinical features, 102–103
    differential diagnosis, 102–103
    epidemiology, 102
    evaluation of patient with, 103
    follow-up for, 104
    ICD-9 code, 104
    imaging in, 103
    laboratory investigation, 103
    outcomes with, 104
    physical examination of patient with, 103
    prognosis for, 104
    signs and symptoms, 102
    treatment, 102, 103
  symmetric, 163
    differential diagnosis, 163–165
  tinnitus with, 163
  trauma-related, 166
  treatment, 167. *See also* Hearing aid(s)
Sepsis, neonatal, hearing loss caused by, 554
Septodermoplasty, for epistaxis, 233
Septoplasty, 672–674
  for obstructive sleep apnea, 68
Septorhinoplasty, for nasal fracture, 599
Sequence, definition, 537
Serial dilution endpoint titration (SDET), 248–249
Seroma, postoperative, 61–62
Sestamibi. *See* Technetium-99m sestamibi
SET. *See* Skin endpoint titration (SET)
Sevoflurane
  adverse effects and side effects, 44
  for anesthesia induction, 44
  pharmacology, 44
  physiologic effects, 44
Sheehan syndrome, hypothyroidism caused by, 475
Shoulder dysfunction, after neck dissection, 406
Shprintzen syndrome. *See* Velocardiofacial syndrome
Sialadenitis
  acute, 443–444
    bacterial, 444–445
      microbiology, 445
      signs and symptoms, 444
      treatment, 445
    differential diagnosis, 443
    signs and symptoms, 443
    treatment, 444
    viral, 443–444
  ICD-9 code, 447
  radiation-related, 358
Sialography, in Sjögren syndrome, 445
Sialolithiasis, 446–447
  complications, 447
  differential diagnosis, 446
  epidemiology, 446
  ICD-9 code, 447
  imaging, 446
  signs and symptoms, 446
  treatment, 446
Sicca complex, 445
Sickle cell anemia, 56–57
  clinical features, 56
  management, 57
  pathophysiology, 56

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Sickle crises, 57
aplastic, 57
hemolytic, 57
sequestration, 57
vasoocclusive, 57
Silicone, liquid, as injectable filler, 648*t*
Sim's triangle, 459*t*
Single-photon emission computed tomography (SPECT)
parathyroid, 497
for parathyroid disease, 29
Singular neurectomy, for benign paroxysmal positional vertigo, 182
Sinonasal biopsy, 216
Sinonasal cancer, 359–364
biopsy, 360
clinical features, 359
diagnosis, 360
differential diagnosis, 359
distant metastases, staging, 363
epidemiology, 359
evaluation, 360
follow-up, 361
history taking in, 360
ICD-9 codes, 361
imaging, 360
outcomes with, 361
pathology, 360
physical examination of patient with, 360
prognosis for, 361
regional lymph node metastases, staging, 362–363
risk factors for, 359
signs and symptoms, 359
stage groupings, 363
staging, 362–363
treatment, 360, 361*f*
Sinonasal disease, orbital extension, 219–221
Sinonasal physiology, assessment, 213, 214*t*
Sinonasal undifferentiated carcinoma (SNUC), 251, 360
Sinus, paranasal. *See* Paranasal sinus(es)
Sinus drainage, culture, 214*t*
Sinusitis
acute. *See also* Acute invasive fungal rhinosinusitis
complementary and alternative medicine (CAM) for, 90
ICD-9 code, 218, 221, 237
other, ICD-9 code, 237
unspecified, ICD-9 code, 224, 237
acute bacterial, 216
chronic
ICD-9 code, 218, 221, 224, 242
other, ICD-9 code, 242
unspecified, ICD-9 code, 242
in elderly, 81
fungal, 215
health care costs, 234, 238
intracranial complications, 222–224. *See also* Abscess, intracranial; Meningitis
clinical features, 222
differential diagnosis, 222
epidemiology, 222
evaluation, 223
follow-up, 224
ICD-9 codes, 224
imaging, 223
laboratory investigations, 223
outcomes with, 224
pathogenesis, 223
physical findings in, 223
signs and symptoms, 222
treatment
medical, 223–224
surgical, 224
orbital complications, 219–221
Chandler classification, 219, 219*t*
clinical features, 219–220
differential diagnosis, 220
epidemiology, 219
evaluation, 220
follow-up, 221
ICD-9 codes, 221
imaging, 220
laboratory investigations, 220
microbiology, 220
outcomes with, 221
pathology, 220
physical findings in, 220
signs and symptoms, 219
treatment
medical, 221
surgical, 221
Sistrunk procedure, 580
SIT. *See* Smell Identification Test (SIT)
Sjögren syndrome, 445–446
complications, 446
epidemiology, 445
laboratory investigation, 445

STUDENTS-HUB.com Uploaded By: tuka abuzneid

salivary gland involvement in, 445
signs and symptoms, 445
treatment, 445
Skin
anatomy, 627
layers, 627
melanoma, ICD-9 code, 420
other malignant neoplasm, ICD-9 code, 410, 414
Skin cancer. *See also* Basal cell carcinoma (BCC); Melanoma
nonmelanoma, 396–398
Skin endpoint titration (SET), 214*t*
Skin graft(s). *See also* Full thickness skin graft (FTSG); Split thickness skin graft (STSG)
complications, 628
for facial reconstruction, 626–629
indications for, 627
operative technique for, 627–628
healing, 627
meshed, 628
postoperative care for, 629
recipient bed for, 627
Skin resurfacing, 651, 656–659
laser, 656, 658–659
Skin test(s), for allergy, 248–249
Skull
bone and articular cartilage, malignant neoplasm, ICD-9 code, 427
vault, fractures, ICD-9 code, 608
Skull base
anterior, disruption, CSF leaks with, 225–228
fractures, 104–108
injury
and CSF rhinorrhea, 225
iatrogenic, repair, 227–228
lateral
middle fossa approach to, 198, 198*f*
retrosigmoid (suboccipital) approach to, 198, 198*f*
surgical approaches to, 198, 198*f*
translabyrinthine approach to, 198
transmastoid approach to, 198*f*
osteomyelitis, imaging, 146–147, 146*t*
Sleep apnea. *See also* Obstructive sleep apnea (OSA)
causes, 66
definition, 66
with hyposomnia or insomnia, ICD-9 code, 68
ICD-9 code, 68
nasolaryngoscopy in, 4
signs and symptoms, 66
unspecified, ICD-9 code, 553
Sleep-disordered breathing. *See also* Obstructive sleep apnea (OSA)
epidemiology, 66
Smell disturbance(s). *See also* Olfactory disorders; *specific disturbance* (e.g., Anosmia)
ICD-9 code, 256, 258
Smell Identification Test (SIT), 214*t*, 226, 239, 255
Smokeless tobacco
and oral cancer, 370
and oropharyngeal cancer, 378
Smoking
and head and neck SCC, 346
and laryngeal cancer, 389
and olfactory changes, 255
and oral cavity cancer, 370
and oropharyngeal cancer, 378, 380
and rhinitis, 244
and sinonasal cancer, 359
SND. *See* Selective neck dissection (SND)
SNHL. *See* Sensorineural hearing loss (SNHL)
Sniffing position, 12
SNUC. *See* Sinonasal undifferentiated carcinoma (SNUC)
Social history, for elderly patient, 79
Sodium, daily requirements for, 49
Soft palate, malignant neoplasia, ICD-9 code, 380
Soft tissue fibrosis, radiation-related, 358
Soft tissue infection, necrotizing, 328–330
clinical features, 329
differential diagnosis, 329
epidemiology, 329
evaluation, 329–330
imaging, 329
laboratory investigation, 329
microbiology, 330
mortality rate for, 330
pathology, 330
physical examination of patient with, 329
risk factors for, 329
signs and symptoms, 329
treatment, 330
Soft tissue necrosis, radiation-related, 358
Somatosounds, 193

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Sound sensitivity, in vestibular disorders, 177
SP/AP ratio, elevated, 159
Spasmodic dysphonia, 285
  abductor, 289
    evaluation, 287
  adductor, 289
    evaluation, 286–287
  treatment, 288–290
SPECT. *See* Single-photon emission computed tomography (SPECT)
Speech awareness threshold, in children, 156
Speech recognition threshold (SRT), 149, 152–153
Speech testing, indications for, 167
Sphenoidal sinus, malignant neoplasm, ICD-9 code, 361
Sphenoid sinus, 211, 212*f*
  cancer, 359. *See also* Sinonasal cancer
Sphenopalatine artery (SPA), 212, 213*f*
Sphenopalatine ganglion
  block, 34–35, 246
  Botox injection into, 246
  branches from, airway innervation by, 8
Spinal accessory nerve (cranial nerve XI), 327, 328*f*, 694*f*
  embryology, 574*t*
  functions, 691
Spinal cord, myelopathy, radiation-related, 358
Spiral ganglion, 101*f*
Spiral ligament, 101*f*
Spiral limbus, 101*f*
Spirochetal disease. *See* Lyme disease; Syphilis
Split thickness skin graft (STSG), 626, 627
  for facial reconstruction, operative technique for, 627–628
Squamous cell carcinoma (SCC), 346. *See also* Head and neck squamous cell carcinoma (HNSCCA)
  basaloid, oropharyngeal, 380
  clinical course, 411
  epidemiology, 411
  hypopharyngeal, 385
  intranasal, differential diagnosis, 238
  inverted papilloma and, 250
  laryngeal, 390
  of lip, 373
  metastases, 411
  of middle ear, 424–427
  oral, 372
  oropharyngeal, 380
  risk factors for, 411
  salivary gland, 452
  sinonasal, 251, 360
  of skin (head, face, and neck), 411–416
    adenoid (acantholytic), 413
    anatomic (Clark) levels, 416
    biopsy, 412
    clinical features, 412
    differential diagnosis, 412
    epidemiology, 411
    evaluation, 412–413
    follow-up, 413
    grading, 413
    history taking for, 412
    ICD-9 codes, 414–415
    imaging, 412
    indolent, 413
    in situ, 413
    metastases, 411
    outcomes with, 413
    pathology, 413
    physical examination of patient with, 412
    prevention, 411, 413
    recurrence, 411
    signs and symptoms, 412
    spindle cell, 413
    stage groupings, 415
    staging, 415
      high-risk features for, 415
    treatment, 411, 413, 414*t*
    variants, 413
  subtypes, 425
  of temporal bone, 424–427
SRS. *See* Stereotactic radiosurgery (SRS)
SRT. *See* Speech recognition threshold (SRT)
Stapedectomy, for otosclerosis, 161
Stapedotomy, for otosclerosis, 161
Stapes
  anatomy, 98–100, 99*f*
  anterior crus, 98, 99*f*
  capitulum, 98, 99*f*, 100
  embryology, 97
  footplate, 98–100, 99*f*
  posterior crus, 98, 99*f*, 100
Staphylococci (*Staphylococcus* spp.)
  coagulase-negative, chronic rhinosinusitis caused by, 240
  coagulase-positive, sinusitis, orbital complications, 220
  thyroiditis caused by, 481

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Staphylococcus aureus. See also Methicillin-resistant *Staphylococcus aureus* (MRSA)
and chronic otitis media, 123
chronic rhinosinusitis caused by, 240
deep neck infection, 334
laryngitis caused by, 282
lymphadenitis caused by, in children, 566
otitis externa caused by, 143
sialadenitis caused by, 445
Staphylococcus pneumoniae, acute otitis media caused by, 119
Stenting, endolaryngeal, 314, 316*t*
Stereotactic radiosurgery (SRS), 356
Steroid(s)
for Bell palsy, 113
for brain abscess, 132
for chronic rhinosinusitis, 240–241, 241*t*
intratympanic, for sudden sensorineural hearing loss, 103
intratympanic perfusion, for Ménière disease, 185
for Ménière disease, 167, 185
for meningitis, 131
nasal, for chronic allergy, 249
nasal spray, for nonallergic rhinitis, 246
oral, for chronic allergy, 249
for otitic hydrocephalus, 135
for sudden sensorineural hearing loss, 103
topical nasal
for acute rhinosinusitis, 236
for chronic rhinosinusitis, 241, 241*t*
for vestibular neuritis, 188, 188*t*
Stertor, 507
Stickler syndrome, 557–558, 588
and cleft lip/palate, 587
hearing loss in, 165*t,* 557–558
and Pierre Robin sequence, 534, 535
Storage disorders, 540–541
Streptococci (*Streptococcus* spp.)
β-hemolytic, laryngitis caused by, 282
group A β-hemolytic
adenotonsillitis caused by, 545
carriage rate, 545
pharyngitis caused by, 545
group B, lymphadenitis caused by, in children, 566
rhinosinusitis caused by, 235
sinusitis, orbital complications, 220
thyroiditis caused by, 481
Streptococcus pneumoniae
deep neck infection, 334
laryngitis caused by, 282
rhinosinusitis caused by, 235
upper respiratory infection, in children, 510
Streptococcus pyogenes
deep neck infection, 334
sialadenitis caused by, 445
upper respiratory infection, in children, 510
Streptococcus viridans
deep neck infection, 334
sialadenitis caused by, 445
Stria vascularis, 101*f*
Stridor, 272–275, 507
acute, 272
associated symptoms, 272
biphasic, 507
chronic, 272
clinical presentation, 272
differential diagnosis, 272
evaluation, 273–274
expiratory, 507
follow-up, 275
ICD-9 code, 275, 511, 516
imaging in, 273–274
inspiratory, 507
laboratory investigation, 274
with laryngeal clefts, 517
localization, by anatomic site, 272, 273*t*
outcomes with, 275
pediatric
causes, 511–512
differential diagnosis, 512
physical examination of patient with, 273
treatment, 274–275
Stroboscopy, 287–288
Stroke, 285
neurolaryngeal involvement in, treatment, 288, 289
pathophysiology, 288
postoperative, 61
STSG. *See* Split thickness skin graft (STSG)
Subcutaneous layer (of skin), 627
Subdural empyema. *See* Empyema, subdural
Subglottic stenosis, 508, 510, 529–533
congenital, 529–533
clinical features, 529–530
complications, 532
differential diagnosis, 530
epidemiology, 529

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Subglottic stenosis *(continued)*
evaluation, 530–531
follow-up, 533
grading, 530, 531*t*
ICD-9 code, 533
outcomes with, 532–533
pathology, 531
physical examination of patient with, 530
signs and symptoms, 530
treatment, 531–532
grading, 530, 531*t*
imaging, 531
pathology, 531
Subglottis
malignant neoplasm, ICD-9 code, 395
pediatric, 529
Sublingual gland, malignant neoplasm, ICD-9 code, 456
Submandibular area, 325, 326*f*
Submandibular gland
carcinoma, lymph node metastases, 344*f*
malignant neoplasm, ICD-9 code, 456
Submandibular triangle, 404
Submental region, 325, 326*f*
Submental triangle, 404
Substance abuse, preoperative assessment, 6*t*
Succinylcholine
adverse effects and side effects, 45*t*
contraindications to, 45*t*
dosage and administration, 45*t*
indications for, 45*t*
mechanism of action, 45
pharmacology, 45
for rapid-sequence induction, 13
Sumatriptan, for migraine, 191–192, 192*t*
Superficial cervical fascia, 325*f*
Superficial cervical plexus, block, 33–34
Superior laryngeal nerve, 693*f*
airway innervation by, 7–8
thyroid innervation by, 461
Superior orbital fissure syndrome, 220
Superior semicircular canal dehiscence syndrome (SCDS), 178, 180
audiologic assessment in, 203
clinical features, 202
differential diagnosis, 202
epidemiology, 202
evaluation, 202–203
follow-up, 204
history taking in, 202
ICD-9 code, 204
imaging in, 202–203
outcomes with, 204
pathology, 201, 203
physical examination of patient with, 202
signs and symptoms, 160, 201, 202
treatment, 178
medical, 203
surgical, 203–204
vertigo caused by, 183
Supraglottic area, innervation, 8
Supraglottis, malignant neoplasm, ICD-9 code, 395
Supraglottitis, bacterial, 281–282
Supraglottoplasty, for laryngomalacia, 513
Supraorbital nerve, block, 32–33
Supratrochlear nerve, block, 32–33
Surgery. *See also* Facial plastic and reconstructive surgery
for acute invasive fungal rhinosinusitis, 218
for adenotonsillitis, 547
for airway obstruction in Pierre Robin sequence, 536
for basal cell carcinoma, 409, 410*f*
for Bell palsy, 113
for benign paroxysmal positional vertigo, 182
for benign salivary gland tumors, 449
for bilateral vocal fold paralysis, 516
for branchial cleft cyst, 576
for choanal atresia, 586
for cholesteatoma, 138–139
for chronic otitis media, 124–125
complications, 125–126
for chronic rhinosinusitis, 241–242
for cleft lip/palate, 590–592, 590*f*–593*f*
for conductive hearing loss, 161
for deep neck infection, 335, 336*t*
for epidural abscess, 133
for hearing loss, 167
in hypothyroid patient, 477
for idiopathic midline destructive disease, 436
for infectious neck mass, in children, 568–569
for inverted papilloma, 251–253
for laryngeal cancer, 393–394
for laryngeal clefts, 519–520
for laryngomalacia, 513
laser. *See* Laser(s)
for lateral sinus thrombosis, 133
for malignant neoplasm of ear and temporal bone, 427
for mandibular fractures, 617–618

STUDENTS-HUB.com Uploaded By: tuka abuzneid

for melanoma, 419
for Ménière disease, 185–186
for midface fractures, 612
for necrotizing soft tissue infection, 330
for obstructive sleep apnea, 68
for orbital complications of sinusitis, 221
for oropharyngeal cancer, 380
for papillomatosis, 294
for paraganglioma, 438–439
for petrous apicitis, 130
for reflux disorders, 319
robotic-assisted, 86–89
for subdural empyema, 134
for subglottic stenosis, 531–532
for superior semicircular canal dehiscence syndrome, 203–204
for temporomandibular joint disorders, 76
for thyrotoxicosis, 473
for vascular rings, 527–528
for Zenker diverticulum, 307
Surgilube jelly, for nasolaryngoscopy, 4
Swallowing
impaired. *See* Dysphagia
phases, dysphagia and, 308
physiology, 266
Swallowing disorders. *See* Dysphagia
Swallowing therapy, 310, 314
Swallow reflex, evaluation, 27
Sweating
decreased, radiation-related, 358
gustatory, 450
Swimmer's ear, 141
Syndrome, definition, 537
Synthroid, 476–477
Syphilis
and acquired prenatal hearing loss, 560
diagnosis, 260*t*, 261
laboratory diagnosis, 563
laryngitis caused by, 283
nasal manifestations, 238
otologic manifestations, 205
serology, indications for, 194, 203
sinonasal manifestations, 259
transfusion-related, 54
treatment, 205, 260*t*
Systemic disease(s)
epidemiology, 259
epistaxis caused by, 230*t*
laryngeal manifestations, 319–322
nasal manifestations, differential diagnosis, 234
otologic manifestations, 204–208
rhinologic manifestations, 259–262
sinonasal manifestations, 259
differential diagnosis, 259–261
evaluation, 261
imaging, 261
laboratory investigation, 261
pathology, 261–262
physical examination of patient with, 261
treatment, 262
voice changes caused by, 286–290

T

T3. *See* Triiodothyronine (T_3)
T4. *See* Thyroxine (T_4)
Tapazole. *See* Methimazole
Tarsorrhaphy, 625
Tarsus, 661–662
Taste
innervation, 257
loss, 257–259
physiology, 266
qualities, 257
sensation, 257
Taste buds, 257
innervation, 266
Taste disturbance, 257–259
causes, 258
clinical features, 258
epidemiology, 257–258
evaluation of patient with, 258
follow-up, 258
ICD-9 code, 256, 258
outcomes with, 258
postoperative, 258
radiation-related, 358
treatment, 258
Taste phantoms, 258
Taste receptor(s), 257
Taxanes
for head and neck cancer, 349
mechanism of action, 354
TBW. *See* Total body water (TBW)
T-cell lymphoma, 238, 431*t*
sinonasal manifestations, 259
Tears, artificial, 625
Technetium-99m, for thyroid scintigraphy, 28
Technetium-99m scan, indications for, 146–147, 146*t*
Technetium-99m sestamibi, for parathyroid scintigraphy, 28–29, 497

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Tectorial membrane, 101*f*
TEF. *See* Tracheoesophageal fistula (TEF)
Telecanthus, traumatic, 600–601
Teletherapy, 356–357
Telogen, 678
Telogen effluvium, 678
Temporal area, anatomy, 654
Temporal bone
  basal cell carcinoma, 424–427
  fibrous dysplasia, hearing loss caused by, 165
  fracture
    epidemiology, 104
    hearing loss caused by, 166
    longitudinal, 105, 106*f*
    signs and symptoms, 105
    transverse, 105, 106*f*
    treatment, 108
  malignant neoplasms, 424–427
    biopsy, 425
    clinical features, 424–425
    differential diagnosis, 424–425
    evaluation, 425
    follow-up, 427
    ICD-9 codes, 427
    imaging, 425
    outcomes with, 427
    pathology, 425
    physical examination of patient with, 425
    signs and symptoms, 424
    staging, 426
    treatment, 426–427
  Paget disease, hearing loss caused by, 165
  plasmacytoma, 206
  squamous cell carcinoma, 424–427
  trauma
    clinical features, 105
    differential diagnosis, 105
    epidemiology, 104
    evaluation of patient with, 105–107
    follow-up, 108
    imaging, 106
    outcomes with, 108
    physical examination of patient with, 105–107
    treatment, 107–108
Temporomandibular joint (TMJ)
  anatomy, 73–74
  disease, differential diagnosis, 117
  disorders, 73–77
    arthrogenous, 73
    articular, pathology, 75
    causes, 74
    clinical features, 74
    complications, 77
    differential diagnosis, 74, 238
    epidemiology, 74
    evaluation, 74–75
    follow-up, 77
    history taking in, 74
    ICD-9 code, 77
    imaging, 75
    myogenous, 73
      pathology, 75
    outcomes with, 77
    pathology, 75
    physical findings in, 74–75
    physical therapy for, 75
    signs and symptoms, 74
    treatment
      medical, 75–76
      surgical, 76
  mobility, assessment, 9
  pain, with malignant otitis externa, 145
  physiology, 73–74
Tennison-Randall triangular flap repair, for cleft lip, 591, 591*f*
Tensor veli palatini muscle, 100
Teratogen(s), and cleft lip/palate, 587
Teratoma, congenital nasal, 581–584
Tetany, in hypocalcemia, 65
Tg. *See* Thyroglobulin
TH. *See* Thyroid hormone(s) (TH)
Thiopental
  dosage and administration, 43
  pharmacology, 43
Third space (redistribution fluid loss), 50
Threshold(s), in audiology, 149
Thrombocytopenia
  definition, 51
  dilutional, 55
Thumbprint sign, 282
Thyroglobulin, 461
  measurement, 465
  serum, 469
  synthesis, 462*f*
Thyroglossal duct, 460*f*
Thyroglossal duct cyst, 577–580
  epidemiology, 578
  ICD-9 code, 580
  pathology, 579, 579*f*
  signs and symptoms, 578
  treatment, 580
Thyrohyoid membrane, 268–269

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Thyroid. *See* Thyroid gland
Thyroid adenoma(s), toxic, 472
Thyroid artery(ies)
  inferior, 459
  superior, 459
Thyroid cancer, 483–494
  anaplastic, 483, 484*t*
    biologic behavior, 488
    cellular origin, 484*t*
    clinical presentation, 488
    diagnosis, 488
    epidemiology, 484*t*, 488
    prognosis for, 488
    stage groupings, 494
    treatment, 484*t*, 488–489
  clinical presentation, 467
  diagnosis, 468–469
  distant metastases, staging, 492
  in elderly, 81
  epidemiology, 467, 483
  follicular, 483, 484*t*
    cellular origin, 484*t*
    clear cell, 486
    clinical presentation, 486
    diagnosis, 486
    epidemiology, 484*t*, 485
    Hürthle cell, 486
    insular, 486
    oxyphilic, 486
    prognosis for, 486
    risk factors for, 485–486
    stage groupings, 492–493
    trabecular, 486
    treatment, 484*t*, 486
    variants, 486
  ICD-9 code, 491
  long-term follow-up, 487–488
  medullary, 483, 484*t*
    cellular origin, 484*t*, 489
    clinical presentation, 489
    epidemiology, 484*t*, 489
    familial, 489
    pathology, 489
    postoperative management, 490
    screening, 489
    stage groupings, 493
    treatment, 484*t*, 489–490
  papillary, 483, 484–485, 484*t*
    cellular origin, 484*t*
    clear cell variant, 485
    clinical presentation, 485
    columnar variant, 485
    diffuse sclerosing variant, 485
    epidemiology, 484, 484*t*
    follicular variant, 485
    oxyphilic variant, 485
    pathogenesis, 484
    pathology, 485
    poorly differentiated, 485
    prognosis for, 485
    risk factors for, 484–485
    solid variant, 485
    stage groupings, 492–493
    tall cell variant, 485
    trabecular variant, 485
    treatment, 484*t*, 486
    variants, 485
  physical findings in, 467
  regional lymph node metastases, staging, 492
  stage groupings, 491–494
  staging, 491–494
  undifferentiated, 483, 484*t*
    stage groupings, 494
  well-differentiated, 483–488, 484*t*
    postoperative complications, 487
    postoperative management, 487–488
    radiation therapy for, 488
    recurrence, surveillance for, 487–488
    treatment, 486
Thyroid cartilage, 268, 269*f*
Thyroid cyst, 467, 469–470
Thyroid disease, in elderly, 81
Thyroidectomy
  indications for, 473
  subtotal
    indications for, 473
    transient hypothyroidism caused by, 475
  transaxillary, robotic-assisted, 87
Thyroid follicle(s), 461–463, 462*f*
Thyroid function tests, 464–465, 466*t*
Thyroid gland
  agenesis, 475
  anatomy, 458
  arterial supply, 459
  cells, 459
  dysgenesis, 475
  embryology, 458, 460*f*, 579, 579*f*
  evaluation, 464–466
  fascia and, 458
  fine-needle aspiration biopsy, 466
  follicular cells, 459, 461
  functions, 458
  imaging, 465–466

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Thyroid gland *(continued)*
innervation, 461
isthmus, 458
ligaments and, 458
lobes, 458
lymphatics, 459
malformations, 458, 460*f*
malignant neoplasm, ICD-9 code, 491
morphology, 458
parafollicular cells, 459
physiology, 461–463, 462*f*
radionuclide study, 465–466
ultrasound, 468
venous drainage, 459
weight, 458
Thyroid hormone(s) (TH), 461
actions, 463
binding proteins, 461, 464
excessive, ingestion, ICD-9 code, 474
extrathyroidal sources, 472
regulation, 463
replacement therapy, 476–477
secretion, 462*f*
synthesis, 462*f,* 463
increased, 471–472
tissue resistance to, 475
transport proteins, 461, 464
Thyroid hormone binding protein, abnormal, ICD-9 code, 477
Thyroid hormone uptake test, 465
Thyroiditis, 479–483
acute, 479, 480*t*
suppurative, 480–481, 480*t*
autoimmune, 474, 475
bacterial, 480–481, 480*t*
chronic, autoimmune, 474, 475, 481
de Quervain, 479–480, 480*t*
drug-induced, 479, 480*t,* 482
fibrous, 482
giant cell, 479–480
Hashimoto, 474, 479, 480*t,* 481
hyperthyroidism secondary to, 472
ICD-9 code, 483
infectious, 480–481, 480*t*
clinical features, 481
etiology, 481
evaluation, 481
laboratory investigation, 481
treatment, 481
invasive, 482
laboratory investigation, 469
lymphocytic, 481–482
ICD-9 code, 477
not associated with pain and tenderness, 481–483
with pain and tenderness, 479–481, 480*t*
painless, 480*t,* 481–482
postpartum, 479, 480*t,* 481–482
radiation-induced, 479, 480*t,* 481
Riedel, 482
silent, 479, 480*t,* 481–482
subacute, 479–480, 480*t*
480, 479–480
clinical features, 479–480
etiology, 479
granulomatous, 479–480
HLA association in, 479
ICD-9 code, 477
laboratory investigation, 480
lymphocytic, 481–482
nonsuppurative, 479
treatment, 480
suppurative, 480–481, 480*t*
types, 479, 480*t*
Thyroid nodule(s), 467–470
benign (hot), 467
clinical presentation, 467
cold, 468
cystic, 469–470
epidemiology, 467
evaluation, 467–469
fine-needle aspiration biopsy, 466, 468–469
history taking for, 467
hot, 468
ICD-9 code, 470
imaging, 468
malignant, 467, 483
diagnosis, 468–469
signs and symptoms, 485
physical examination of patient with, 468
toxic, ICD-9 code, 474
warm, 468
Thyroid peroxidase (TPO), 463
antibodies to, 472
Thyroid scintigraphy, 28
Thyroid-stimulating hormone (thyrotropin, TSH), 461
actions, 463
assay, 464
recombinant human, 487
serum
in hyperthyroidism, 472
in hypothyroidism, 474, 476
in patient with thyroid nodule, 469

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suppression therapy, for thyroid cancer, 487
Thyroid-stimulating immunoglobulins (TSIs), 471
in hyperthyroidism, 472
Thyroid storm, 478–479
definition, 478
etiology, 478
evaluation, 478
laboratory investigation, 478
signs and symptoms, 478
treatment, 478–479
Thyroid surgery, hematoma after, 62
Thyroid tissue, ectopic, 460*f*
Thyromental distance, 9
Thyrotoxicosis. *See also* Thyroid storm
causes, 465*t*
definition, 470
etiology, 471–472
treatment, 473
Thyrotropin-releasing hormone (TRH), 461
actions, 463
serum, in hypothyroidism, 474
Thyroxine (T_4), 461, 463
decreased, causes, 464*t*
free, 464
in hyperthyroidism, 472
in hypothyroidism, 476
increased, causes, 464*t*
total, 464
Thyroxine-binding globulin (TBG)
decreased, causes, 464*t*
increased, causes, 464*t*
L-Thyroxine replacement therapy, 476–477
Tibial bone graft(s), for facial reconstruction, 641
Tinnitus, 193–195
characteristics, 193
cholesteatoma and, 121–122
clinical features, 193–194
complementary and alternative medicine (CAM) for, 90, 194–195
differential diagnosis, 193–194
drug-induced, 194, 195
in elderly, 80
epidemiology, 193
evaluation, 194
follow-up, 195
with hearing loss, 163
history taking in, 193
ICD-9 code, 195
imaging of patient with, 194
in Ménière disease, 183
objective (pulsatile)
definition, 193
differential diagnosis, 194
outcomes with, 195
pathology, 194
physical examination of patient with, 194
severity, 193
subjective (nonpulsatile)
definition, 193
differential diagnosis, 193–194
treatment
medical, 194–195
quality of life and, 194
surgical, 195
in vestibular disorders, 177
Tinnitus matching, 194
TIVA. *See* Total intravenous anesthesia (TIVA)
TM. *See* Tympanic membrane (TM)
TNM staging
of head and neck cancer, 351
of hypopharyngeal cancer, 386–387
of laryngeal cancer, 395–397
of melanoma
mucosal, 422–423
of skin, 421–422
of nasopharyngeal carcinoma, 367–369
of oral cavity cancer, 376–377
of oropharyngeal cancer, 381–382
of sinonasal cancer, 362–363
of squamous cell carcinoma of skin (head, face, and neck), 415
of thyroid cancer, 491–494
Tobacco. *See also* Smokeless tobacco; Smoking
and hypopharyngeal cancer, 383
Tobey-Ayer test, for lateral sinus thrombosis, 133
Toluidine blue staining, in oral cancer, 371
Tongue
cancer, 346
anatomical distribution, 373
epidemiology, 370
metastases, 373
oral, 373
treatment, 373
carcinoma, lymph node metastases, 343*f*
malignant neoplasm, ICD-9 code, 375

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Tongue base
  cancer, 378. *See also* Oropharyngeal cancer
  malignant neoplasia, ICD-9 code, 380
  surgery, for obstructive sleep apnea, 68
  suspension, for obstructive sleep apnea, 68
Tongue-lip adhesion, for airway obstruction in Pierre Robin sequence, 536
Tonsil(s)
  abscess, ICD-9 code, 336
  cancer, 346, 378
  hypertrophy, ICD-9 code, 553
  innervation, 8
  malignant neoplasia, ICD-9 code, 380
Tonsil fossa, malignant neoplasia, ICD-9 code, 381
Tonsillar carcinoma, lymph node metastases, 343*f*
Tonsillar pillars, malignant neoplasia, ICD-9 code, 381
Tonsillectomy
  for adenotonsillitis, 547
  complications, 686
  contraindications to, 685
  dissection instruments for, 685
  indications for, 685
    absolute, 685
    relative, 685
  procedure for, 685
Tonsillitis
  acute, ICD-9 code, 547
  chronic, ICD-9 code, 547
TORCH complex, laboratory investigation, 562
TORCHES, hearing loss caused by, 166, 554
TORS. *See* Transoral robotic-assisted surgery (TORS)
Total body water (TBW), 49
Total intravenous anesthesia (TIVA), 31
Toxicity. *See* Nephrotoxicity; Ototoxicity
Toxic multinodular goiter, 472, 473
  ICD-9 code, 474
Toxoplasmosis
  lymphadenitis caused by, in children, 566
  transfusion-related, 54
TPO. *See* Thyroid peroxidase (TPO)
Trachea
  anomalies, ICD-9 code, 275
  innervation, 8
Tracheal anomalies, ICD-9 code, 511
Tracheitis, acute, ICD-9 code, 284
Tracheobronchial stenosis, 509
Tracheoesophageal fistula (TEF), 509, 521–525
  associated anomalies, 521
  classification, 522, 523*f*
  differential diagnosis, 521
  epidemiology, 521
  evaluation, 522
  ICD-9 code, 524
  imaging, 522
  outcomes with, 524
  physical examination of patient with, 522
  recurrent, 524
  signs and symptoms, 521
  treatment, 522
    complications, 522–524
Tracheoesophageal puncture prosthesis
  follow-up, 401
  outcomes with, 401
  postlaryngectomy use, 399, 400
Tracheomalacia, 508
  after tracheoesophageal fistula/esophageal atresia repair, 524
  treatment, 528
Tracheotomy
  for airway impairment secondary to bilateral lesions, 297
  for airway obstruction in Pierre Robin sequence, 536
  in aspiration, 314, 315–316*t*
  awake, 19–20
    technique for, 20
  for bilateral vocal fold paralysis, 516
  complications, 20
  double-barrel, 316*t*
  indications for, 20*t*
  in laryngeal fractures, 276, 277
  for obstructive sleep apnea, 68
  open surgical
    complications, 687–688
    indications for, 687
    procedure for, 687
  pediatric, 510
  percutaneous dilation, 21–22
    contraindications to, 21
    equipment for, 22
    indications for, 21
    technique for, 21–22
  for stridor, 274
  in trauma patient, 340
Tragus, 97*f*

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TRALI. *See* Transfusion-related acute lung injury (TRALI)
β_2-Transferrin, assay, in rhinorrhea fluid, 226, 227*t*, 612
Transfusion reaction(s), 52–55
  acute hemolytic, 52–53
  anaphylactic, 53
  delayed hemolytic, 53
  febrile, 53
  hemolytic, 52–53
    treatment, 55
  immune, 52–53
  nonhemolytic, 53
  urticarial, 53
Transfusion-related acute lung injury (TRALI), 53
Transient ischemic attacks, vertigo and disequilibrium caused by, 183
Transoral laser microsurgery, for laryngeal cancer, 393
Transoral robotic-assisted surgery (TORS), 87
  for laryngeal cancer, 393
  for oropharyngeal cancer, 380
Trauma
  craniomaxillofacial, 597–620
  and difficult airway, 16*t*
  ear, 104–108
  epistaxis caused by, 230*t*
  evaluation, screening tests in, 611–612
  hearing loss caused by, 166
  neck, 337–341
  pediatric, hearing loss caused by, 561
  temporal bone, 104–108
Treacher-Collins syndrome, 558, 588
  and choanal atresia, 584
  and cleft lip/palate, 587
  hearing loss in, 165*t*
  and Pierre Robin sequence, 534
Tremor, vocal
  pathophysiology, 289
  treatment, 288–290
Treponema pallidum. See Syphilis
Treximet, for migraine, 192*t*
TRH. *See* Thyrotropin-releasing hormone (TRH)
Triangular fossa, auricular, 97*f*
Tricyclic antidepressants, for migraine, 192*t*
Trigeminal nerve (cranial nerve V), 327
  airway innervation by, 7–8
  auriculotemporal branch, innervation of external auditory canal, 98
  divisions, 690
  embryology, 574*t*
  function, 690
  maxillary division, block, 34–35
  nasal innervation by, 212–213
Triiodothyronine (T_3), 461
  actions, 463
  free, 464
    in hyperthyroidism, 472
  total, 464
Trimethoprim-sulfamethoxazole
  for acute rhinosinusitis
    adult dosage and administration, 235, 236*t*
    pediatric dosage and administration, 236
  for bacterial lymphadenitis, in children, 567–568
  dosage and administration
    adult, 567
    pediatric, 567
Tripod fracture, zygomaticomaxillary complex, 602–605
Triptans, for migraine, 191
Trismus, 9
  radiation-related, 358
Trisomy 18, and choanal atresia, 584
Trochlear nerve (cranial nerve IV), 327
  function, 690
Troisier sign, 565
Trousseau sign, 502
TSH. *See* Thyroid-stimulating hormone (thyrotropin, TSH)
TSIs. *See* Thyroid-stimulating immunoglobulins (TSIs)
Tubercle of Zuckerkandl, 458, 459*t*
Tuberculosis
  diagnosis, 205
  laryngitis caused by, 282
  lymphadenitis caused by, in children, 566
  nasal manifestations, 238
  otologic manifestations, 204–205
  sinonasal manifestations, 259
  treatment, 205
Tullio phenomenon, 202
Tumor(s)
  and difficult airway, 16*t*
  hearing loss caused by, 165
  intranasal, 251
  vascular, pediatric, 569–573
Tumor redifferentiation agents, for thyroid cancer, 488

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Tuning fork examination, 166
Tuning fork tests, 152. *See also* Rinne test; Weber test
  in conductive hearing loss, 160
Turbinate(s), 211
22q11 deletion syndrome. *See* Velocardiofacial syndrome
Tympanic membrane (TM)
  in acute otitis media, 118
  anatomy, 98
  embryology, 95
  examination, 118
  layers, 98
  pars flaccida, 98
  pars tensa, 98
  perforation
    in acute otitis media, 118
    after tympanostomy tube removal, 120
    and chronic otitis media, 121
    and hearing loss, 159–160, 562
    with painless otorrhea, 121, 122
    traumatic, epidemiology, 104
  rupture (perforation), traumatic
    causes, 104
    clinical presentation, 104
    follow-up, 108
    prognosis for, 108
    treatment, 107
  surgery, for conductive hearing loss, 161
Tympanocentesis, indications for, 118, 120
Tympanogram, 149–150, 151*f*
  acute otitis media and, 118–119
Tympanomastoidectomy
  with canal wall down, for chronic otitis media with cholesteatoma,125
  with canal wall down (CWD), for cholesteatoma, 139
    follow-up, 140
  with canal wall up (CWU), for cholesteatoma, 139
    follow-up, 140
  with intact canal wall, 124–125
Tympanometry
  indications for, 160
  maximum compliance peak, 153
  pediatric, 156
  results, Jerger classification system for, 151–152, 151*f*
Tympanoplasty
  alone or with atticotomy, for cholesteatoma, 139
  indications for, 120
  without mastoidectomy, 124
Tympanostomy tube(s), 564
  complications, 120
  follow-up, 120
  indications for, 120
  insertion, in cleft palate repair, 592
  otorrhea with, 120
  removal, 120

U

Ultrasound, 25–26
  with fine-needle aspiration biopsy, 26
  indications for, 25–26
  of neck mass, 344
  parathyroid, 497
  principles, 26, 26*f*
  of sialolithiasis, 446
  thyroid gland, 466, 468
Umami, 257
Unknown primary carcinoma. *See* Cancer of unknown primary (CUP).
Upper aerodigestive tract
  anatomy, 265–271
  carcinoma, cervical lymph node metastases, 347, 347*t*
  disorders, aspiration in, 313
  malignancy, 117
    in elderly, 81
  physiology, 265–271
Upper airway, nerve blocks for, 34–35
Upper jugular chain lymph node region, 325, 326*f*
Upper respiratory infection(s) (URIs)
  and acute otitis media, 119
  complementary and alternative medicine (CAM) for, 90
  pediatric, treatment, 510
  viral
    differential diagnosis, 234
    and vestibular neuritis, 186
UPPP. *See* Uvulopharyngopalatoplasty (UPPP)
Urinary tract infection(s) (UTI), postoperative, treatment, 59
Urine output, postoperative, 64
URIs. *See* Upper respiratory infection(s) (URIs)

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Usher syndrome, 556, 557*t*
hearing loss in, 165*t*
Utricle, 100
Uveoparotid fever, 205
Uvula, innervation, 8
Uvulopharyngopalatoplasty (UPPP), for obstructive sleep apnea, 68

V

VACTERL association, 542
Vagus nerve (cranial nerve X), 266, 309, 327, 693*f*
branches, airway innervation by, 7–8
embryology, 574*t*
functions, 691
injury to, in neck dissection, 406
innervation of external auditory canal, 98
recurrent laryngeal branch, block, 35
superior laryngeal branch, block, 35
and taste, 257
Valacyclovir
for Bell palsy, 113
for vestibular neuritis, 188, 188*t*
Valium. *See* Diazepam
Vallecula, malignant neoplasia, ICD-9 code, 381
Valtrex. *See* Valacyclovir
Vancomycin
for *Clostridium difficile* colitis, 64
for intracranial complications of sinusitis, 223–224
for meningitis prophylaxis, with repair of congenital nasal mass, 583
for orbital complications of sinusitis, 221
Van der Woude syndrome, 543
Vascular injury, in neck, traumatic, signs, 337–338
Vascular malformations, in children, 569–573
differential diagnosis, 571
epidemiology, 570
evaluation, 571
outcomes with, 572
signs and symptoms, 570
treatment, 572
Vascular rings, 509, 525–529
definition, 525
differential diagnosis, 526
embryology, 525
epidemiology, 526
evaluation, 526–527
follow-up, 528
ICD-9 code, 529
imaging, 526
outcomes with, 528
physical examination of patient with, 526
signs and symptoms, 526
treatment, 527–528
complications, 528
outcomes with, 528
types, 525
Vasomotor rhinitis (VMR), 225, 243, 245
signs and symptoms, 243–244
treatment, 246
surgical, 246
VATER association, 542
laryngeal clefts in, 517
VC. *See* Verrucous carcinoma (VC)
VDRL. *See* Venereal Disease Research Laboratory (VDRL) test
Vecuronium
clinical considerations with, 46*t*
dosage and administration, 46*t*
pharmacology, 46*t*
Velocardiofacial syndrome, 543
and Pierre Robin sequence, 534
VEMPs. *See* Vestibular evoked myogenic potentials (VEMPs)
Venereal Disease Research Laboratory (VDRL) test, 261
Venous sinus(es), intracranial, phlebitis and thrombophlebitis, ICD-9 code, 221, 224
Ventilation, transtracheal, 14–15
Ventriculoperitoneal shunt, for otitic hydrocephalus, 135
Verapamil, for migraine, 191, 192*t*
Verrucous carcinoma (VC), 372
Vertebrobasilar occlusion, hearing loss caused by, 165
Vertical crest. *See* Bill's bar
Vertiginous syndromes, ICD-9 code, 192
Vertigo, 176–177
with acute suppurative labyrinthitis, 130, 131
atypical, evaluation, 180
cholesteatoma and, 121–122
complementary and alternative medicine (CAM) for, 90
disorders causing, 183

STUDENTS-HUB.com Uploaded By: tuka abuzneid

Vertigo *(continued)*
epidemiology, 176
inner ear–related, 183
in Ménière disease, 183
migraine-associated, 189–193
clinical features, 190
diagnostic criteria for, 190
differential diagnosis, 190
evaluation, 190–191
follow-up, 192
ICD-9 codes, 192
imaging in, 191
outcomes with, 192
physical findings in, 191
treatment, 191–192, 192*t*
vestibular testing in, 191
noise-induced, 202
postoperative, 126
recurrent episodic, causes, 183
superior semicircular canal dehiscence and, 183
with tympanic membrane rupture, 106, 107
unspecified, ICD-9 code, 186
in vestibular neuritis, 186–189
Vertigoheel, 90
Vestibular aqueduct, 100
enlarged, 556
Vestibular disorders
central causes, 178
clinical features, 176–177
epidemiology, 176
ICD-9 code, 192
pathology, 178
peripheral causes, 178
signs and symptoms, 176–177
Vestibular evoked myogenic potentials (VEMPs), 177
factors affecting, 178
in superior semicircular canal dehiscence syndrome, 203
testing, indications for, 203
Vestibular (Reissner's) membrane, 101*f*
Vestibular nerve, 691
sectioning, for Ménière disease, 185–186
Vestibular neuritis (VN), 178, 186–189
differential diagnosis, 180, 187
epidemiology, 187
evaluation, 187
follow-up, 189
ICD-9 code, 189
imaging in, 187
outcomes with, 189
pathology, 188
physical findings in, 187
recurrence, 189
signs and symptoms, 187
treatment, 188, 188*t*
Vestibular rehabilitation
for chronic disequilibrium, 192
for vestibular neuritis, 188, 188*t*
Vestibular schwannoma
Antoni A, 197
Antoni B, 197
bilateral, 196
epidemiology, 196
growth, monitoring, 197–198, 197*t*
growth rate, 197
hearing loss caused by, 165
imaging, 196
pathology, 197
surgical approaches for, 198, 198*f*
and tinnitus, 196
treatment, 197–199
vertigo and disequilibrium caused by, 183, 196
Vestibular suppressant(s), for vestibular neuritis, 188, 188*t*
Vestibule, of inner ear
anatomy, 100
embryology, 96*f*
Vestibulocochlear nerve (cranial nerve VIII), 110, 327
dysfunction, with petrous apicitis, 129
embryology, 574*t*
functions, 691
VFD. *See* Vocal fold dysfunction (VFD)
VHI. *See* Voice Handicap Index (VHI)
Videonystagmography (VNG), 177
factors affecting, 178
indications for, 187
in Ménière disease, 185
Vidian nerve, nasal innervation by, 212–213
Vidian neurectomy, 246
Vinblastine, mechanism of action, 354
Vincristine, mechanism of action, 354
Vinegar wash, for chronic otitis media, 123, 124*t*
Viral infection
and facial paralysis/paresis, 112
laryngitis caused by, 281
lymphadenitis caused by, in children, 566
rhinosinusitis caused by, 235

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sialadenitis caused by, 443–444
subacute thyroiditis caused by, 479
transfusion-related, 54
Vitamin A deficiency, and oral cancer, 370
Vitamin C, for cold prevention and treatment, 90
Vitamin D
and calcium metabolism, 502
deficiency, and hyperparathyroidism, 498, 499
supplementation
in hypocalcemia, 503
in hypoparathyroidism, 501
Vitamin E, and epistaxis, 230
Vitrase, 649
Vivactil. *See* Protriptyline
VMR. *See* Vasomotor rhinitis (VMR)
VN. *See* Vestibular neuritis (VN)
VNG. *See* Videonystagmography (VNG)
Vocal disorders
differential diagnosis, 301
epidemiology, 301
evaluation, 286–287, 301–302
acoustic, 302
perceptual, 301
physiological, 302
signs and symptoms, 301
Vocal fold(s)
anatomy, 269
cysts, 295–297
disease, ICD-9 code, 297
edema, 295–297
ICD-9 code, 297
false, 269, 389
immobility, 297–300
motion impairment, 285, 297–300. *See also* Vocal fold(s), paresis/paralysis
in adults, 297–300
in children, 297–300
differential diagnosis, 298
evaluation, 298–299
follow-up, 300
history taking for, 298–299
imaging, 299
laboratory investigation, 299
outcomes with, 300
physical examination of patient with, 299
signs and symptoms, 297–298
treatment, 299–300
nodules, 295–297
nonneoplastic changes, 295–297
differential diagnosis, 295
epidemiology, 295
evaluation, 296
follow-up, 296
history taking for, 296
ICD-9 codes, 297
outcomes with, 296
physical examination of patient with, 296
signs and symptoms, 295
treatment, 296
paralysis, after neck dissection, 406
paresis/paralysis, 286, 508. *See also* Vocal fold(s), motion impairment
bilateral. *See* Bilateral vocal fold paralysis (BVFP)
follow-up, 300
ICD-9 code, 290, 300
imaging, 287
outcomes with, 300
unilateral, 297
polyps, 295–297
ICD-9 code, 297
structure, 269
true, 269, 389
Vocal fold dysfunction (VFD), 285
pathophysiology, 289
treatment, 288–290
Vocal function exercises, 303
Vocal hygiene, 303
Voice
changes. *See also* Aphonia; Dysphonia
postlaryngectomy, differential diagnosis, 399
evaluation, 286–287
rehabilitation, postlaryngectomy, 400–401
evaluation for, 399
Voice disturbance. *See also* Vocal disorders
ICD-9 code, 304
Voice exam, 286–287
Voice Handicap Index (VHI), 301, 302*t*
Voice rehabilitation, 300–304
Voice therapy, 300–304
confidential, 303
Lee Silverman, 304
options for, 303–304
resonant, 303
Von Langenbeck repair, for cleft palate, 592, 592*f*
von Willebrand disease (VWD), 57–58
cryoprecipitate for, 52
management, 58

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von Willebrand factor (VWF), 57–58
  infusion, for von Willebrand disease, 58
VoSol, for otitis externa, 144
VWD. *See* von Willebrand disease (VWD)
VWF. *See* von Willebrand factor (VWF)

W

Waardenburg syndrome, 557
  and cleft lip/palate, 587
  hearing loss in, 165*t*
Wart(s), oral, 71–73
Warthin tumor, 448, 448*t*
  pathology, 449
Weber test, 152, 153*f*
Wegener granulomatosis
  clinical presentation, 122
  diagnosis, 206, 260*t*, 261
  ICD-9 code, 322
  laboratory diagnosis, 122
  laboratory investigation, 261
  laryngeal manifestations, 320
  nasal manifestations, 238
    differential diagnosis, 234
  otologic manifestations, 205
  sinonasal manifestations, 259
  treatment, 206, 260*t*, 262
Weight loss
  for obstructive sleep apnea, 67
  radiation-related, 358
Wheezing, 507
Wick(s), for drug delivery in otitis externa, 143, 144
Wildervanck syndrome, 559
Wilson's disease, hypoparathyroidism in, 500
Word recognition ability, 149, 153
Wound(s), surgical
  infection, 62
    after neck dissection, 406
  postoperative problems, 61–63
    assessment, 61–63
    management, 61–63
    risk factors for, 61
Wound infection
  gas-producing, 329
  postoperative, 62

X

Xeroderma pigmentosa, and basal cell carcinoma, 407
Xerophthalmia, in Sjögren syndrome, 445
Xerostomia
  in elderly, 81
  radiation-related, 358
  in Sjögren syndrome, 445
X-linked hearing loss, 559

Y

Yerba maté
  and head and neck cancer, 346
  and oropharyngeal cancer, 378
Yersiniosis, transfusion-related, 54

Z

Zellballen, 438
Zenker diverticulum (pl., diverticula), 305–307
  differential diagnosis, 305
  dysphagia caused by, 305–307
  epidemiology, 305
  evaluation, 305–306
  history taking for, 305–306
  imaging, 306
  pathology, 306, 306*f*
  physical examination of patient with, 306
  signs and symptoms, 305
  treatment, 307
Zinc, intranasal, and olfactory changes, 255
ZMC. *See* Zygomaticomaxillary complex (ZMC) fractures
Z-plasty, 645, 645*f*
Zygomatic arch fracture
  epidemiology, 602
  ICD-9 code, 605
Zygomaticomaxillary complex (ZMC) fractures, 602–605
  complications, 604
  differential diagnosis, 603
  epidemiology, 602
  evaluation, 603–604
  follow-up, 604
  ICD-9 codes, 605
  imaging, 604
  outcomes with, 604
  physical examination of patient with, 603
  signs and symptoms, 603
  treatment, 604
Zygomycosis. *See also* Acute invasive fungal rhinosinusitis
  ICD-9 code, 218

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