Adam I. Levine Satish Govindaraj Samuel DeMaria, Jr. *Editors*

Anesthesiology and Otolaryngology



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Dedication

To my best friend and wife, Robin, whose commitment, support and sacrifice made this book possible. To my daughter, Sam, whose enthusiasm was always welcome. To my dad, Howie, in loving memory.

Adam I. Levine

To my parents, who taught me the importance of hard work and perseverance; to my wife and best friend, Jocelyn, for her support, patience, and love over the years; and to my children, Maya, Neelam, and Devan, who make me realize each day what is truly important in life.

Satish Govindaraj

To my wife, Tara, for her unwavering support and enthusiasm.

Samuel DeMaria, Jr.

A View on Sharing the Airway

It still is debated as to who deserves credit for administering the first general anesthetic. Many credit Dr. William Morton, a dentist, for first demonstrating ether anesthesia on October 16, 1846, in the "Ether Dome" of the Massachusetts General Hospital, since the case was published in the Boston Medical and Surgical Journal in November 1846. The patient had a vascular tumor of the neck removed in the sitting position by the surgeon, John Collins Warren, without crying out in pain. However, it is well known that Dr. Crawford W. Long, a country physician practicing in rural Georgia, used ether anesthesia in 1842 to remove another tumor of the neck. The patient felt no pain and had a good outcome. This case is documented in the Crawford W. Long Museum in Jefferson, Georgia, which is open today and supported by the Georgia Society of Anesthesiologists. These are the first documented cases utilizing the combined skills of both a clinician administering a general anesthetic and a surgeon performing head and neck surgery.

Spontaneous ventilation during anesthesia was managed by mask without specialized airway devices for almost 50 years after the discovery of the inhaled anesthetic agents. With increasing complexity and duration of more extensive surgical procedures near the neck, nose, and mouth, the need arose for devices to isolate and control the airway. In 1878, Sir William Macewen was the first physician to intubate the trachea orally by a blind technique for the sole purpose of administering general anesthesia for an extensive operation on the tongue. Robert Miller and Robert Macintosh designed laryngoscopes in the early 1940s for anesthesiologists to visualize the larynx and intubate the patient's trachea under direct vision. The modern techniques to deal with the difficult airway came later with the introduction of special laryngoscope blades, the laryngeal mask airway, and fiberoptic laryngoscopes and bronchoscopes in recent decades.

It took almost 100 years after the first use of ether anesthesia for the specialty of anesthesiology to become well established during World War II. The new discipline had the usual problems of achieving recognition, but by 1935 leaders in the field were forming the early Departments of Anesthesiology at Bellevue Hospital in New York City, the University of Wisconsin, and the University of Iowa. Specialization in medicine depends primarily on advances in medical science and technology, professional preferences, and economic considerations. Some physicians are drawn to specialties because they offer defined responsibilities, more control over their practices, prestige, and potential income. This is true of both anesthesiology and otolaryngology.

As research in the medical sciences has continued to grow and new clinical applications of these findings are introduced, our medical knowledge base has expanded, creating new specialties and new subspecialties within established medical disciplines. Within anesthesiology, some of the earliest subspecialties were critical care medicine, pediatric anesthesia, neuroanesthesia, cardiac anesthesia, and thoracic anesthesia. In each of these subspecialties, like thoracic anesthesia, the medical knowledge of the fields of pulmonary medicine and thoracic surgery must be combined and coordinated with the knowledge and the special techniques of the anesthesiologist in order to provide safe care of the patient undergoing highly complex chest surgery. Certainly, this is also true of one of the newest subspecialties highlighted in this excellent textbook, *Anesthesiology and Otolaryngology*. This book takes it even one step further with its combined authorship of the chapters by both surgeons and anesthesiologists, who point out the key clinical material for their own and the other subspecialists who are often sharing the patient's airway during complex head and neck surgery. Both specialties emphasize the key coordination and communication that are needed for successful outcomes. In 2002, Pronovost and the Johns Hopkins group showed that clinical outcomes in critically ill patients were improved by having intensive care units staffed by well-trained intensivists, and I am certain that outcomes have been and will be improved with the growing role of subspecialists like those described in this text.

My successful career as a subspecialist in cardiac, thoracic, and vascular anesthesia is due to the excellent mentors I had during medical school, residency, and fellowship training, and early on as an attending anesthesiologist. These mentors served me as "wise and trusted counselors" as first described by Homer in *The Odyssey* about the character, Mentor. They were motivated to help me, to share knowledge and experience, and to demonstrate their commitment to caring for the patients. They showed me the benefits of subspecialization and helped me apply my knowledge of cardiovascular medicine to the cardiac surgical operating room, leading to the development of cardiac anesthesia and improved care for the patients with heart disease undergoing new cardiac operations. I hope that my example, in some small way, led the editors of this outstanding book to share their experiences with their students and mentees.

During my 15 years as chairman of the Department of Anesthesiology at the Mount Sinai Hospital and School of Medicine, I was able to introduce the subspecialties of anesthesiology in order to improve our clinical, educational, and research missions in coordination with the various surgical departments. Thus, today, it is my great pleasure to see this new book edited by Levine, Govindaraj and Demaria, Jr., and authored by authorities at Mount Sinai, that highlights the further developments in coordinated perioperative care for otolaryngology surgery. The highly specialized area of anesthesia for head and neck surgery around and in the airway provides extensive information for all anesthesiologists caring for patients undergoing all types of surgical procedures. This book brings together a segment of anesthetic and surgical practice not readily available from other sources. It should become the standard textbook for teaching in these subspecialities, lead to improved care, and, hopefully, even save lives by its demonstration of best practices for complex surgical procedures.

San Diego, CA, USA

Joel A. Kaplan

Foreword

The major theme of this important textbook is stated very clearly in the first chapter namely, "... the importance of teamwork ... with a special emphasis on effective communication." Both anesthesiologists and otolaryngologists share a common goal, which is to achieve the best possible outcome for the patient, and that outcome is very dependent on teamwork and communication. As a young otolaryngology resident at Boston University School of Medicine, I will never forget my encounter with a senior anesthesiologist at a major Boston teaching hospital. I was assisting my attending in a composite cancer resection of the head and neck and happened to notice that the blood in the field appeared somewhat dark and possibly not well oxygenated. The year was 1974, when routine use of blood gas monitoring was not available and the operating field was separated from the anesthesia delivery equipment by an "ether screen." I leaned over to speak to the anesthesiologist with some trepidation and expressed my concern. The answer was rather straightforward, "Don't tell me how to give anesthesia, and I won't tell you how to do surgery!" A few other choice words were expressed by my senior attending, but they are not appropriate for this Foreword. Fortunately, for me, the rest of my encounters with my anesthesia colleagues during and after residency have been exemplary. My mentors during residency taught me by example that respect, collegiality, and good communication are essential to ensure good surgical outcomes for my patients.

John C. Snow, MD, former Chair of the Department of Anesthesiology at Boston University, states in the first edition of Anesthesia in Otolaryngology and Ophthalmology (1972), "... there is the problem of a strong competition between the anesthesiologist and the surgeon in a small area of the body. In particular, surgery of the mouth and throat involves the airway itself, with the added risk of respiratory obstruction by secretions, blood, loose tissue, or instruments." Since the 1970s, the challenge of sharing the airway of course has become much more complicated with the advent of lasers and the emergence of new endoscopic technology, ventilating, and imaging systems. Given the incredible importance of teamwork, it is the responsibility of the anesthesiologist and otolaryngologist as the proverbial "Captains of the Ship" to collaboratively provide leadership and education for members of the entire perioperative team. Communication therefore must start well before the induction of anesthesia in all cases, but particularly in difficult airway situations. Preoperative photographs of the pathologic condition and status of the airway should always be present to share with the team before the patient arrives in the operating room. Important comorbidities such as gastroesophageal reflux, chronically infected secretions, reactive airway conditions, and sleep apnea need to be discussed preoperatively. A detailed written checklist of preparations for the surgical procedure should be part of the planning process. My typical checklist for laser endoscopy in the airway will include choosing and checking the functionality of the equipment, preoperative medications, position of the patient, endotracheal tube type and size or Venturi jet system, and of course, safety precautions for the patient and the operating team.

When the patient signs the permission for surgery, the team of otolaryngologists and anesthesiologists assume the role of guardian. I am not a particularly religious man; however, I do believe we have just made a sacred contract with the patient to provide the best possible surgical outcome. An understanding and insight into the entire anesthesia experience is an extremely important part of the surgeon's responsibility to the patient. Each individual patient has some unique requirements demanding careful planning and consideration prior to surgery. Attention to details and adhering to a checklist make the physicians better guardians for the patient. I am happy to tell the patient in the preoperative area, particularly in a difficult airway situation, that fortunately we have an experienced, talented anesthesia and nursing team specially trained to deal with the particular problem of the patient and that we work as a team. This statement has a very calming influence on the patient.

It is customary in a Foreword to tell the readers what is unique about the text and the contribution the book makes to the existing literature on the subject. The authors have succeeded in putting together for the first time an outstanding reference for both otolaryngologists and anesthesiologists practicing in the modern era with the state-of-the-art technology. The basic science chapters on anatomy, physiology, and pharmacology set the stage for the clinical chapters coauthored by both specialists, with unique considerations for both surgery and anesthesia management. "Insights" and "Pearls" are pointers provided by and for each other as would be expected in a truly team approach to care. This very comprehensive textbook in addition discusses acute and chronic pain management and postoperative care as well as the often not discussed topic of medical-legal concerns and patient safety. *Anesthesiology and Otolaryngology*, written and edited by Doctors Levine, Govindaraj, and DeMaria, Jr. who are leaders in their fields, is a landmark textbook that is essential reading for otolaryngologists and anesthesiologists dealing with patients requiring surgery of the upper aerodigestive system and head and neck. It will certainly be a most valuable addition to my medical library.

Albany, NY, USA

Stanley M. Shapshay

Preface

The book *Anesthesiology and Otolaryngology* is a novel and innovative collaboration of otolaryngologists and anesthesiologists predominately from their respective departments at the Icahn School of Medicine at Mount Sinai, one of the busiest otolaryngology services in the United States. Expert teams of anesthesiologists and otolaryngologists have been brought together to coauthor most of this text.

The book is divided into three main sections. The introductory chapters cover essential material for both specialties, including gross and radiographic anatomy, basic physiology, pharmacology of anesthetic and adjunctive medications, and preoperative assessment, optimization, and monitoring. In this section of the book we also include a chapter regarding oxygen delivery systems. This chapter serves as a comprehensive review for the anesthesiologists and an overview for the otolaryngologist of the basics of the anesthesia machine, oxygen delivery systems, and jet and other ventilator modalities.

The chapters in the main body of the text are collaboratively authored by experts in both specialties to provide a unique perspective on the management of patients presenting for a full range of otolaryngologic procedures. In each of these chapters information is presented and organized in the order in which it would occur; namely preoperative, intraoperative, and postoperative considerations. The otolaryngologist's perspective is presented first, followed by the anesthesiologist's perspective. Special consideration has been given to difficult airway management and pediatric otolaryngologic procedures because these topics represent some of the most challenging situations encountered by both specialists. The chapter on airway emergencies and the difficult airway, for example, serves as a key reference for the practicing clinician on the details and considerations in acute airway management. In no other setting is it as critical for both the anesthesiologist and otolaryngologist to work as an efficient team. This chapter discusses airway devices and techniques germane to both specialists, including awake intubation techniques, extubation protocols, fiberoptic bronchoscopy, and video and rigid laryngoscopy.

Each chapter in the main body of the book concludes with *Pearls* and *Insights*, special bulleted sections where key "take home" points are emphasized. *Clinical Insights* are written by each specialist for readers of the other specialty. *Insights* highlight salient concepts that are critical for a complete understanding of the topic from the other specialist's perspective. *Clinical Pearls*, on the other hand, are written by each specialist for readers from their own specialty. *Pearls* serve as a list of "tricks of the trade" and best practices from experts in the field.

The book concludes with special chapters regarding postoperative care, acute and chronic pain management, and medico-legal issues. Considering the prevalence of otolaryngologic procedures that are performed in the office setting, there is also a chapter regarding the logistics of managing an office-based surgical practice.

Anesthesiology and Otolaryngology represents the first text to bring together collaborative teams of anesthesiologists and otolaryngologists coediting and coauthoring the same topics

from their own point of view. In total, this text is meant to serve as a reference for each specialist, including practicing physicians, trainees, and allied health providers. It is our intent that this collaborative approach will provide you with the knowledge and perspectives necessary to improve your care for the operative otolaryngologic patient.

New York, NY, USA

Adam I. Levine Satish Govindaraj Samuel DeMaria, Jr.

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Working Side by Side: The Important Relationship Between Anesthesiologists and Otolaryngologists

Adam I. Levine, Samuel DeMaria, Jr., and Satish Govindaraj

The relationship between anesthesiologists and otolaryngologists dates back to the beginnings of modern anesthetic practice. Indeed, the earliest use of inhaled anesthetics was for head and neck and intraoral procedures. The first reported public demonstration of ether anesthesia in 1846 was administered for the excision of a vascular neck lesion. This monumental event was later memorialized (the Bullfinch amphitheater of the Massachusetts General Hospital was renamed the "Ether Dome") and recognized as America's greatest contribution to nineteenth century medicine. Incidentally and somewhat humorously, it was also the first case delayed by an anesthesiologist and the first reported case of intraoperative awareness. Not only was Dr. Morton late to the event due to a technical issue with a new ether anesthetic delivery device, but the patient reported that although he was comfortable and pain free, he was awake during the surgery.^[1]

A century later, the anesthesiologist, Dr. Samuel Rochberg wrote in the Archives of Otolaryngology:

The anesthesiologist and the otolaryngologist encounter many mutual problems. They observe many ties and much overlapping in their specialties. To achieve good results, they must understand each other's aims before, during and after every surgical procedure... In conclusion, I should like to reiterate the necessity for greater cooperation and understanding between otolaryngologists and anesthesiologists. Then they will achieve better anesthesia and finer surgical results, with fewer complications ^[2].

A.I. Levine (🖂)

Dr. Rochberg was ahead of his time in being aware of the relevance of interdisciplinary teamwork in the operating room. Despite this longstanding and historically rooted collaboration, little has been written regarding the importance of the relationship between otolaryngologists and anesthesiologists specializing in otolaryngologic surgery.

Due to the proximity of the surgical site to the airway and the complex problems encountered therein, otolaryngologists and anesthesiologists are specialists who literally work side by side and share many common concerns and considerations. Therefore, they benefit greatly by having an advanced understanding of each other's practice.

Insight into the practice of anesthesiology affords otolaryngologists certain advantages: (1) an appreciation of how the surgical plan impacts the anesthetic plan, (2) knowledge of how a given anesthetic affects the surgical environment and the patient's medical condition, and (3) how a patient's medical conditions impact intraoperative management. This allows the otolaryngologists to more effectively communicate their surgical needs to anesthesiology colleagues. Also, an understanding of anesthetic considerations allows otolaryngologists to function as expert assistants to the anesthesiology team during rare and critical events and to appreciate that the idealized surgical state may not be in the best interest of the patient.

Similarly, a thorough knowledge by the anesthesiologist of the otolaryngologist's practice affords them certain advantages: (1) knowledge of the scope of pathology allows for effective preoperative assessment, optimization and anesthetic planning, (2) understanding of the logistics and range of procedures allows for successful preparation for anticipated challenges and complications, and (3) the impact of the anesthetic management on the surgical environment is recognized as an area where anesthetic technique can affect surgical outcome. A working knowledge of one another's specialty is but one piece of a successful collaboration on the parts of both specialists. Although many surgical environments benefit from collegial and collaborative relationships, the unique and intimate otolaryngologic environment mandates such collaboration for optimal patient outcomes.

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Teamwork and Patient Safety

The reader will notice that a major theme of this textbook is the importance of teamwork amongst the two specialties with a special emphasis on effective communication. The Institute of Medicine (IOM) estimates that at least 1.5 million Americans are injured by medical errors annually. Although the IOM has stressed the need for a comprehensive improvement in both patient safety and efficiency in all medical disciplines ^[3], high error rates with serious consequences are most likely to occur in fast pace, critical environments like the intensive care units, emergency departments, and operating rooms. Remarkably, 63% of all sentinel events were found to be directly attributable to breakdowns in communication in these high intensity settings. As such, the Joint Commission has now made improving the effectiveness of communication among caregivers a major patient safety goal.

The importance of communication and effective team work initially arose from high-risk fields such as the nuclear and aviation industries. Due to the error rate in health care, experts have looked to these high reliability organizations for answers. Research from these industries has shown that a majority of disasters have been related to failures in teamwork, including problems in interpersonal communication and leadership. Similarly medical errors and communication breakdown occur in a complex clinical milieu for a variety of reasons. Lingard et al. showed that 30% of communications in the OR failed to convey the intended information properly ^[4]. Thirty-six percent of these failures (approximately 10% of all OR communications) resulted in negative effects on processes, including inefficiency, team tension, resource waste, workarounds, delays, and procedural errors. The surgical environment in particular has a number of factors that make it susceptible to communication and teamwork failure, such as fatigue, stress, high workload, production pressure, and the hierarchal health-care system.

In response to these and other similar findings in health care, team training education including crisis resource

1	Table 1.1 Key points of CRM				
	Points regarding decision making	Points regarding teamwork			
	and cognition	and resource management			
	Know the environment	Exercise leadership and			
	Anticipate and plan	followership			
	Use all available information	Call for help early			
	and cross check	Communicate effectively			
	Prevent or manage fixation errors	Distribute the workload			
	Use cognitive aids	Mobilize all available			
		resources for optimum			
		management			

management (CRM) and TeamSTEPPS[®] principles have emerged. While the details of these programs are beyond the scope of this introductory chapter, the basic principles can be found in Table 1.1.

An emphasis on the collaboration between anesthesiologists and otolaryngologists and a working knowledge of the complexities of the environment and each other's specialty can be found throughout this text because it is of paramount importance. This book's major goal is to educate and empower the reader so he or she can be a knowledgeable and effective leader and member of the otolaryngologic surgical team.

References

- Fenster JM. Ether day: the strange tale of America's greatest medical discovery and the haunted men who made it. New York: HarperCollins; 2001.
- Rochberg S. Anesthesiology and otolaryngology. Arch Otolaryngol. 1949;49(1):53–62.
- Leavitt M. Medscape's response to the Institute of Medicine Report: crossing the quality chasm: a new health system for the 21st century. Med Gen Med. 2001;3(2):2.
- Lingard L, Espin S, Whyte S, et al. Communication failures in the operating room: an observational classification of recurrent types and effects. Qual Saf Health Care. 2004;13(5):330–4.

Gross and Radiographic Anatomy

Puneet Pawha, Nancy Jiang, Katya Shpilberg, Michael Luttrull, and Satish Govindaraj

Introduction

The purpose of this chapter is to review the anatomy of the head and neck that is relevant to both the otolaryngologist and anesthesiologist. The chapter provides the otolaryngologist with an overview of imaging technologies and a concise anatomic review, while also providing the anesthesiologist with a working knowledge of head and neck anatomy and an ability to interpret normal and pathologic head and neck radiographic imaging. Although the intention of this chapter is not meant to supplant the importance of consultation with otolaryngology and radiology colleagues, the ability to interpret head and neck radiographic imaging is critical for the anesthesiologist caring for patients with head and neck pathology. The chapter is divided into two anatomic sections, the airway and the deep neck spaces. Each section includes a discussion of basic anatomy, related pathology, helpful radiological imaging studies, and innervation with related nerve blocks for those contemplating regional anesthesia of the head and neck. The chapter concludes with (Appendix A), a series of CT scans and a MRI that highlights the normal anatomy and a summary of the abbreviations used for each figure throughout the chapter (Appendix B).

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Principles of Computed Tomography and Magnetic Resonance Imaging

Computed Tomography

Computed tomography (CT) plays a crucial role in head and neck imaging. It is the study of choice for osseous, non-marrow replacing lesions as well as chondroid lesions and is indispensable in evaluating the temporal bone and the paranasal sinuses. The skull, face, and spine are most efficiently assessed for fractures with CT. In CT, photons emanating from a collimated X-ray beam pass through the patient and are picked up by detectors. Image contrast is based on the differential attenuation or absorption of the X-ray beam by various tissues, which increases with greater atomic number, density, and/or number of electrons. The least attenuating structures such as air appear black, while the most attenuating ones such as bone and metal appear white. The scale for CT absorption generally ranges from -1000 to +1000 CT numbers termed "Hounsfield units" with 0 assigned to water and -1000 to air. Fat ranges from -40 to -100, soft tissues from +20 to +40, hematomas from +50 to +80, and calcifications are greater than +150.

The X-ray beam is rotated over many different angles during a CT scan in order to get differential absorption patterns through a single slab of the patient's body. A final absorption value for each pixel within a CT slice is obtained by mathematical analysis called "projection reconstruction". Different reconstruction algorithms or kernels can be used to highlight a particular tissue with CT. Images from a given algorithm may be displayed with different window widths and levels in order to accentuate contrast differences between tissues. CT technology has been evolving over several generations in an effort to reduce scanning time and improve image

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quality. One of the latest advances in CT technology is the TE. T1 multi-detector system that can acquire multiple slices per short T

rotation, producing higher resolution scans five to eight times faster than traditional CT technology.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) is based on the ability of protons within the body to absorb and emit radiowave energy when placed within a strong magnetic field. A small number of protons within the scanned tissues align with the main magnetic field and are subsequently displaced from their alignment by application of radiofrequency gradients. When the radiofrequency gradient is terminated, the protons realign with the main magnetic field, releasing a small pulse of energy that is detected, localized, and processed by a computer algorithm. MR analyzes such tissue characteristics as hydrogen (proton) density, T1 and T2 relaxation times, and blood flow. The two major MR settings for conventional spin-echo sequences selected by the operator are "time of repetition" (TR) and "time to echo" (TE). Proton densityweighted sequences, which produce the best soft tissue contrast, are obtained by selecting a long TR and a short

TE. T1-weighted sequences are obtained by selecting a short TR and a short TE. Shortening of T1 such as by fat, proteinaceous material, melanin, methemoglobin, and gadolinium results in a higher signal on T1-weighted imaging. T2-weighted sequences use a long TR and long TE. Prolongation of T2 such as by free water produces high signal on T2-weighted imaging.

MR has advanced to rapid-imaging breath-hold techniques, minimizing motion-related artifacts. Gradient recalled echo (GRE) sequences readily reveal blood products, iron and calcium deposition as low-signal foci of susceptibility. Diffusion-weighted imaging allows detection of processes that restrict motion of water protons into the extracellular space such as infarcts, abscesses, and cellular neoplasms, which bright signal with corresponding low signal on apparent diffusion coefficient (ADC) maps. Inversion-recovery sequences suppress signal from tissues with short T1 relaxation times such as fat and accentuate those with long TR relaxation times such as many pathologic lesions that have high water content. Various other fat-suppression techniques can also be employed in MRI. Intravenous contrast can be given for both CT and MR in order to enhance differences between lesions and surrounding tissues, to characterize lesions by their enhancement patterns, to better delineate anatomy, and demonstrate vessel patency.

The Airway

Sinonasal Anatomy

The sinonasal region is composed of the nose, the nasal cavity structures and the four paranasal sinus groups: the frontal, ethmoid, sphenoid, and maxillary sinuses (Fig. 2.1).

The nasal cavity extends cephalocaudally from the cribriform plate to the hard and soft palates and is bound laterally by the nasal walls. The nasal septum cleaves the nasal cavity in half and is composed of a cartilaginous anterior portion and a bony posterior portion (Fig. 2.2).

The bony portion of the septum is formed by the perpendicular plate of the ethmoid bone superiorly and the vomer inferiorly. The nasal septum is often deviated to one side and bone spurs can protrude from the apex of the deviated septum (Fig. 2.3).

Three pairs of bony projections called the superior, middle, and inferior nasal turbinates arise from the lateral nasal wall and extend medially into the nasal cavity toward the nasal septum. Occasionally, a supreme turbinate is also present. The middle turbinate can be pneumatized (concha bullosa) and this may be associated with deviation of the adjacent nasal septum in the opposite direction. The intervening air channels surrounding the nasal turbinates are, respectively, called the superior, middle, and inferior nasal meati. The superior and middle meati function as components of the paranasal sinus drainage pathways discussed later in this section. The inferior meatus receives drainage from the lacrimal sac via the nasolacrimal duct, which is located in the anterior aspect of the inferior nasal turbinate. The olfactory recess of the air channels extend superiorly to the cribriform plate and occlusion of these air spaces can present with anosmia.

The frontal sinuses are formed within the frontal bone and are the last sinus to fully develop. They abut the orbital roof inferiorly and the anterior cranial fossa posteriorly. The degree of frontal sinus pneumatization is not uncommonly asymmetric, and there may be accessory frontal air cells present. Drainage of the frontal sinuses occurs through the frontal recess. The frontal recess is a complex hour-glass

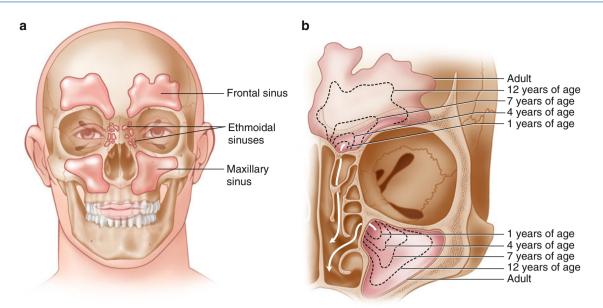


Fig. 2.1 (a) The frontal, ethmoid, and maxillary sinuses are shown here in relation to the facial skeleton. The sphenoid sinus (not shown) is located posterior to the ethmoid sinuses in the majority of cases.

(**b**) Frontal and maxillary sinus development occurs over many years until a person reaches adulthood at the age of 18 years

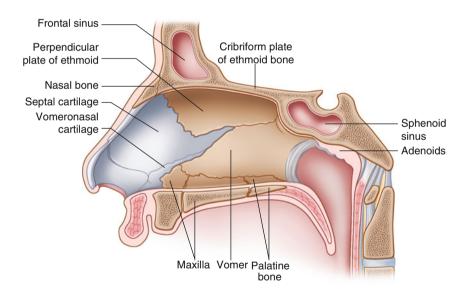


Fig. 2.2 The nasal septum is composed of both cartilage and bone. The superior bony septum is attached to the skull base. Note the posterior location of the sphenoid sinus in the sinonasal cavity

shaped space with variable anatomy that connects the frontal sinus to the anterior ethmoidal sinus. The boundaries of this space include the following: medially, the sagittal attachment of the middle turbinate to the skull base; laterally, it is bounded by the lamina papyracea of the orbit; posteriorly, the space is bounded by the anterior extension of the bulla ethmoidalis and anterior ethmoid artery and anteriorly, it is bounded by the agger nasi cell. Agger nasi cells are the most anterior ethmoid air cells and they lie anterior, inferior, and lateral to the frontal sinus. Its posterior wall forms the anterior wall of the frontal recess. The agger nasi cell is an important surgical landmark because the roof of the cell forms the anterior floor of the frontal recess. Additional anterior ethmoid air cells with various relationships to the frontal recess can be found near the agger nasi region, which are called frontal sinus cells and have been classified according to Kuhn (Table 2.1 and Fig. 2.4)^[1].

The ethmoid sinuses originate from the ethmoid bone and are bound by the cribriform plate and fovea ethmoidalis superiorly, the lamina papyracea laterally, and the nasal cavity medially. They are present at birth and are divided into anterior and posterior ethmoidal cells. The basal lamella of the middle turbinate, which is the attachment of the middle turbinate to the lateral nasal wall and skull base, is what separates the anterior and posterior ethmoid cells. The anterior ethmoid cells drain through the middle meatus and the posterior cells drain through the superior meatus.

Anatomically, the ethmoid sinuses display the most variation (Fig. 2.5). The ethmoid bulla, which is usually

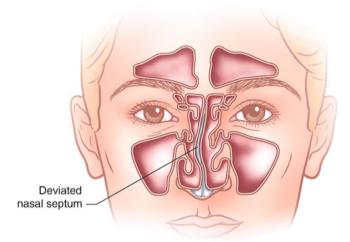


Fig. 2.3 The nasal septum is deviated to the right side causing obstruction of the right nasal cavity

the largest anterior ethmoid air cell, has the most constant anatomical relationship to its surrounding structures and so it serves as an important surgical landmark. The ethmoid bulla is found above the infundibulum and its lateral and inferior surface help to form the superior aspect of the hiatus semilunaris. Other named ethmoid cells include the agger nasi cell, which was discussed earlier, the Haller cell, and the Onodi cell. Haller cells are found along the inferomedial orbital rim and are seen in 10% of the population. These cells may become clinically significant when inflamed, as they can potentially obstruct the osteomeatal complex. The Onodi cell is the most posterior ethmoid air cell and projects superiorly over the sphenoid sinus. Preoperative identification of the Onodi cell is of particular importance because this variant can form a border of the optic canal and internal carotid artery posteriorly, structures which normally abut the sphenoid sinus. During transphenoidal sinus surgery, failure to recognize the presence of an Onodi air cell can lead to injury to these structures.

The sphenoid sinuses arise within the sphenoid bone and can have highly variable degrees of pneumatization. The sinus is often divided by a single sagitally oriented septum in the midline, but the position and number of septations can vary and the sphenoid sinuses are often asymmetric. The

Table 2.1 Kuhn's classification of frontal sinus cells

Frontal sinus cell type	Description
Туре І	A single cell in the frontal recess above the agger nasi cell
Type II	A group of cells in the frontal recess above the agger nasi cell
Type III	A single cell in the frontal recess that protrudes into the frontal sinus
Type IV	An isolated cell within the frontal sinus

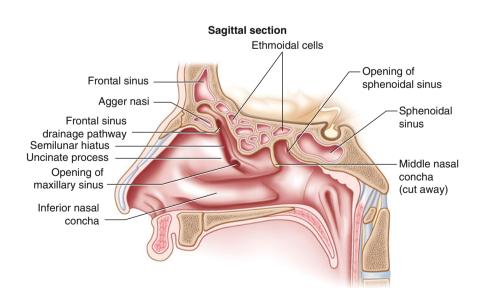
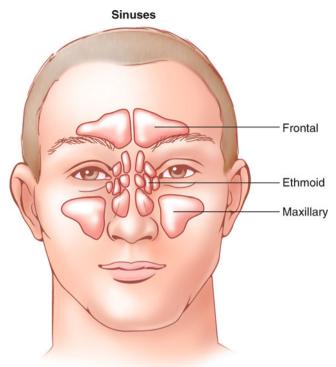


Fig. 2.4 The frontal sinus drainage pathway lies between the agger nasi and variable ethmoid air cells. Each of these cells must be removed during an endoscopic frontal sinus procedure



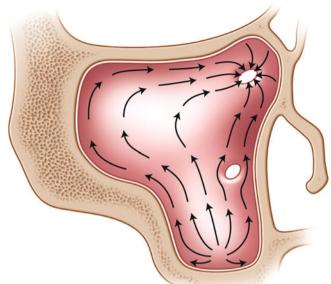


Fig. 2.6 The cilia of the maxillary sinus move mucus to the natural ostium of the sinus located antero-superior in the medial wall of the maxillary sinus. Any surgical opening of the maxillary sinus must incorporate the natural ostium. Note how transport bypasses a nonfunctional opening inferior to the natural ostium

Fig. 2.5 Position of the ethmoid sinuses

sphenoid sinus is surrounded by the sella turcica above, the roof of the nasopharynx below, the clivus posteriorly, and the ethmoid sinus anteriorly. Secretions within the sphenoid sinus are drained via paired sphenoid ostia, which join the sphenoethmoidal recess and lead into the superior meatus.

The maxillary sinuses are formed within each maxillary bone lateral to the nasal cavity. Superiorly, the roof of the maxillary sinus is formed by the orbital floor. Posterior to the maxillary antrum lies the retromaxillary fat and pterygopalatine fossa. Drainage of the maxillary sinus progresses superomedially via cilia, toward the maxillary ostium and infundibulum, which drain to the posterior portion of the hiatus semilunaris of the middle meatus (Fig. 2.6).

At this point it is worth noting that the maxillary, anterior ethmoid and the frontal sinuses all share a common drainage pathway via the middle meatus. Therefore, mucosal inflammatory disease within the middle meatus and/or adjacent structures can directly result in opacification of each or all of these sinuses. Due to the importance of these structures in the development of (and endoscopic treatment for) sinus disease, the area formed by the middle meatus as well as the structures forming the maxillary, ethmoid, and frontal sinus drainage pathway can collectively be referred to as the ostiomeatal unit.

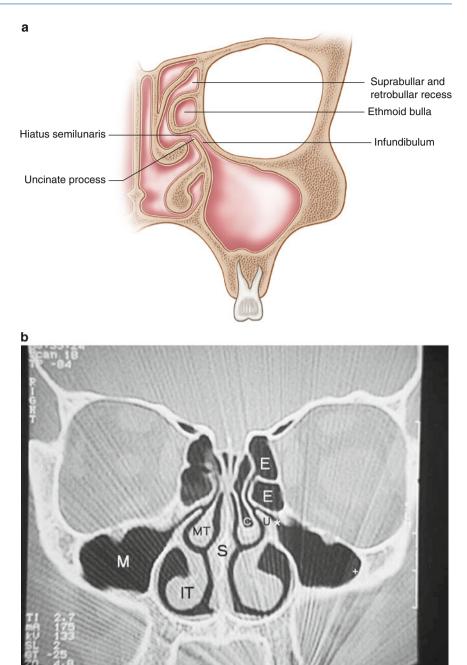
The most important components of the ostiomeatal unit are best visualized on coronal CT images and include the frontal sinus ostium and recess, uncinate process, infundibulum, ethmoid bulla, and the hiatus semilunaris (Fig. 2.7)^[2].

The uncinate process is a superomedially oriented projection of bone, originating from the posteromedial border of the nasolacrimal duct. It forms a free border that serves as the anterior border of the hiatus semilunaris. On CT scan, it can be seen as a superior projection of the medial maxillary sinus wall. The infundibulum serves as the main drainage conduit of the frontal, anterior ethmoid, and maxillary sinuses. It is bounded anteriorly by the uncinate process and medially by the frontal process of the maxilla and lamina papyracea. It is in continuity with the frontal recess anteriorly and connects the maxillary and anterior ethmoid ostia with the hiatus semilunaris. The hiatus semilunaris represents the air channel defined by the ethmoid bulla superiorly, uncinate process anteriorly and inferiorly, bony orbit laterally, and the middle meatus medially ^[2].

Pathologies of the sinonasal region include olfactory dysfunction, congenital anomalies, manifestations of systemic disease, allergy, acute and chronic sinusitis, and both benign and malignant neoplasms. The most common benign neoplasm is inverted papilloma and the most common malignant neoplasm is squamous cell carcinoma (Fig. 2.8).

Other malignant neoplasms include adenocarcinoma, adenoid cystic carcinoma, lymphoma, mucosal melanoma,

Fig. 2.7 (a) schematic of left sinonasal cavity showing the relationship between the uncinate process, hiatus semilunaris, and infundibulum. (b) coronal CT showing the same structures. *MT* middle turbinate, *M* maxillary sinus, *S* septum, *U* uncinate process, *IT* inferior turbinate, *E* ethmoid cell, *C* concha bullosa



esthesioneuroblastoma, and sinonasal undifferentiated carcinoma. Juvenile nasopharyngeal angiofibroma is a benign, vascular tumor that can be locally aggressive. The epicenter of this lesion is the sphenopalatine foramen with adjacent extension to the nasopharynx, pterygopalatine/infratemporal fossae, and skull base (Fig. 2.9).

Radiologic evaluation of the sinonasal and skull base structures is best performed with thin section spiral CT, utilizing multiplanar reformations. MRI can be useful as a problem solving tool for abnormal findings found on CT and in the setting of infiltrating pathologies such as malignancy to characterize disease extent, perineural spread, and intracranial involvement. Noncontrast CT examinations are usually more than sufficient to evaluate for acute and chronic sinonasal inflammatory disease (Fig. 2.10).

Contrast-enhanced examinations are beneficial in the assessment for adjacent intracranial pathology or in the setting of certain malignancies. Thin section spiral CT acquisitions paired with three-dimensional surgical planning software (such as BrainLab) can also be performed and serves as a powerful intraoperative tool during functional endoscopic sinus surgery.

Upper Aerodigestive Tract

Oral Cavity (Fig. 2.11)

The oral cavity represents the most anterior aspect of the upper aerodigestive tract, and many of its structures can be evaluated by direct physical examination. The anterior limit



Fig. 2.8 Inverted papilloma. Coronal CT demonstrates a mass widening the left maxillary sinus ostium (*asterisk*) with a nasal component (n) and a maxillary antrum component (a). Obstructed secretions also contribute to maxillary sinus opacification

of the oral cavity is the vermilion border of the lips. The posterior boundary of the oral cavity, which separates it from the oropharynx, includes the circumvallate papillae, anterior tonsillar pillars, and the junction of the hard and soft palates. The mylohyoid muscle and inferior alveolar ridge form the inferior border, and the buccal mucosa comprise the lateral oral cavity margins. The oral cavity can be further divided into subsites: upper lip, lower lip, upper alveolar ridge, lower alveolar ridge, anterior two-thirds of tongue (oral tongue), retromolar trigone, floor of mouth, buccal mucosa, and hard palate. These subsites are helpful for describing the location of an oral cavity lesion more precisely. Also, malignancies that arise from one subsite vary in their behavior compared to a different subsite and recognition of the appropriate subsite allows for proper prognostic evaluation and treatment. For example, oral tongue cancers that are more than 4 mm thick have a greater than 20% rate of occult cervical lymph node metastasis and such patients should undergo an elective neck dissection whereas in the floor of mouth, lesions more than 1.5 mm have a similar risk and require an elective neck dissection^[3].

Important deep neck spaces associated with the oral cavity include the sublingual and submandibular space. Together, they can also be referred to as the space, with the intervening mylohyoid muscle separating them into individual spaces. The sublingual space is located superior to the mylohyoid muscle, and it contains the sublingual glands and ducts, deep portion of the submandibular gland, submandibular (Wharton's) duct, anterior aspect of the hyoglossus muscle, lingual artery and vein, lingual nerve, glossopharyngeal nerve (CN IX), and the hypoglossal nerve (CN XII). The submandibular space is located inferior to the mylohyoid

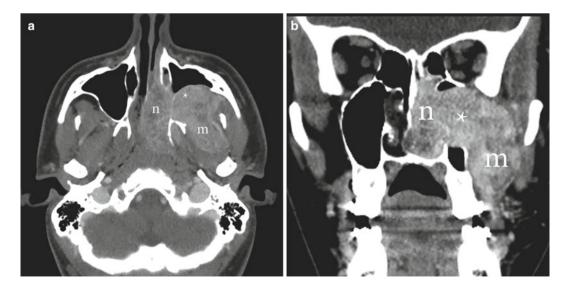


Fig. 2.9 Juvenile nasopharyngeal angiofibroma. Axial (**a**) and coronal (**b**) contrast-enhanced CT (CECT) images demonstrate an avidly enhancing mass extending from the nasal cavity (n) into the masticator space (m) via an enlarged pterygopalatine fossa (*asterisk*)

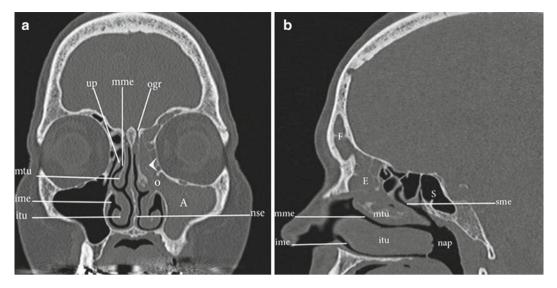


Fig. 2.10 Osteomeatal complex obstruction. Coronal CT with bone window (**a**) demonstrates a left osteomeatal complex obstructive pattern of sinus opacification; arrow points to the demineralized uncinate process; the maxillary antrum (A) and ostium (o) are opacified. Sagittal reconstruction (**b**) demonstrates opacification of the frontal sinus (F)

and anterior ethmoid air cells (E). Normal uncinate process (up, **a**); nasal septum (*nse*, **a**), inferior turbinate (*itu*, **a**, **b**), inferior meatus (*ime*, **a**, **b**), middle turbinate (*mtu*, **a**, **b**), middle meatus (*mme*, **a**, **b**), olfactory groove (*ogr*, **a**), sphenoid sinus (*S*, **b**), nasopharynx (*nap*, **b**), superior meatus (*sme*, **b**)

muscle and its contents include the superficial portion of the submandibular gland, anterior belly of the digastric muscle, facial artery and vein, inferior loop of the hypoglossal nerve (CN XII), submandibular and submental lymph nodes, as well as the submental space (midline fat-containing region between the anterior bellies of the digastric muscles). The facial artery and vein serve as useful landmarks for identifying the submandibular gland and its contents. The facial artery lies deep to the gland, while the facial vein is located immediately superficial and lateral to the gland. Therefore, extraglandular lesions abutting the submandibular gland will often be separated from the gland itself by the intervening facial vein, while intrinsic submandibular pathologies will displace the vein outward. It is important to recognize that there is no fascial boundary separating the posterior aspects of the submandibular and adjacent sublingual spaces from the adjacent parapharyngeal space (PPS). Furthermore, the mucosal surfaces of the oral cavity do not have a fascial border and are contiguous with the oropharyngeal mucosa. This becomes important in the setting of primary cancer, which can easily spread into adjacent structures.

Many different benign and malignant pathologies of the oral cavity can occur and the exhaustive list is too long to discuss in this chapter (Fig. 2.12).

However, two disease entities worth mentioning here is Ludwig's angina and angioedema, as both can cause acute airway obstruction and may require emergent airway management by the anesthesiologist or otolaryngologist. Ludwig's angina is an infection that involves the submental, sublingual, and submandibular spaces and can be lifethreatening due to airway obstruction. The usual source is odontogenic and the infection is generally polymicrobial. Silaloliths are stones located within the ducts of the submandibular and parotid glands that may cause obstruction of salivary flow with secondary infection of the gland (Fig. 2.13).

Severe cases of sialadenitis may mimic a case of Ludwig's angina. Patients will present with painful edema of the floor of mouth and neck and palpation of the floor of mouth is often described as feeling "woody." Initial treatment includes securing the airway with either endotracheal intubation or tracheostomy. Acute angioedema also causes localized edema and this can occur anywhere along the upper aerodigestive tract (oral cavity, pharynx, larynx). The pathophysiology of angioedema involves the release of vasoactive mediators that cause vasodilatation and capillary and venule leakage, leading to interstitial edema. Different types of angioedema include hereditary, drug-induced, and allergic. Involvement of the tongue or larynx can lead to airway obstruction and such patients will require emergent airway management.

The importance of radiologic evaluation of the oral cavity with CT, MRI, and CT/PET lies in their ability to evaluate for the origin and extent of oral cavity disease, accompanying invasion of adjacent structures and the presence or absence of metastatic disease. These findings are often difficult or impossible to discern by direct physical inspection alone and can be critical pieces of information for appropriate clinical management and cancer staging.

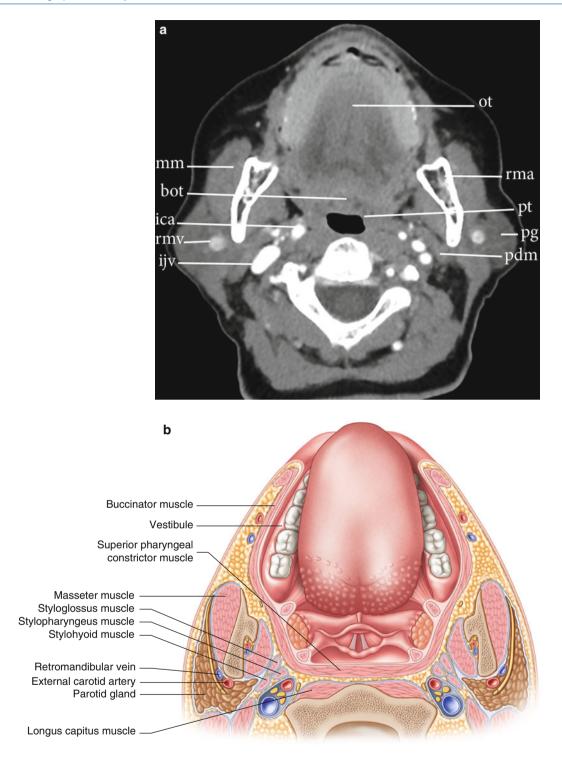


Fig. 2.11 A radiographic and schematic cross section through the oral cavity. The abbreviation list for normal structures is located in Appendix B at the end of the chapter

Nasopharynx

The nasopharynx is located in the superior aspect of the suprahyoid (SH) neck and represents the superior-most

aspect of the visceral compartment. It is bordered by the choanae of the nasal cavities anteriorly; the prevertebral muscles and clivus posterosuperiorly, and the eustachian tube and Fossa of Rosenmüller laterally. The inferior

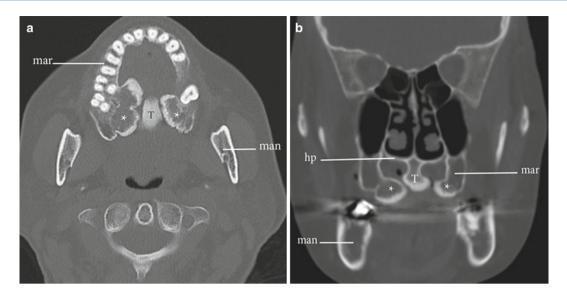


Fig. 2.12 Tori maxillaris and palatinus. Axial (**a**) and coronal (**b**) CT images with bone windows demonstrate exostoses projecting from the maxilla (*asterisk*) and hard palate (torus palatinus, T). Maxillary alveolar ridge (*mar*, **a**, **b**), mandible (*man*, **a**, **b**), hard palate (hp, **b**)

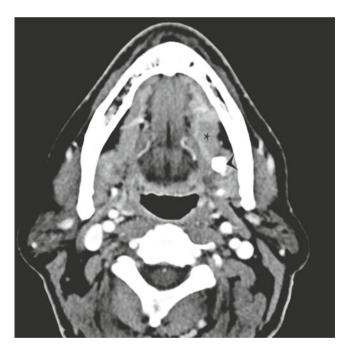


Fig. 2.13 Submandibular sialolith. Axial contrast-enhanced CT demonstrates a large left submandibular sialolith (*arrowhead*) with associated ductal dilatation (*asterisk*)

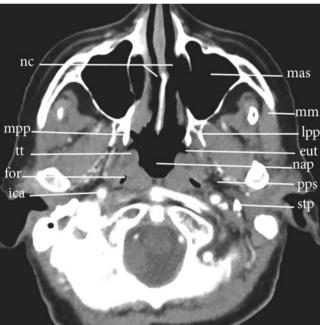


Fig. 2.14 Radiographic image at level of nasopharynx. Abbreviations are located in Appendix B at end of chapter

boundary is the oropharynx, which is separated from the nasopharynx by an invisible horizontal line extending posteriorly from the hard palate (Fig. 2.14).

The contents of the nasopharynx include nasopharyngeal mucosa, minor salivary glands, the adenoids, pharyngobasilar fascia (which serves as the primary attachment of the pharynx to the skull base), the superior constrictor muscles, and the sinuses of Morgagni (through which the levator palatini muscle and torus tubarius pass). The sinuses of Morgagni can act as a natural conduit through the posterosuperolateral aspect of the nasopharynx and allow pathology to spread from the pharyngeal mucosal space, through the pharyngobasilar fascia and into the skull base. The posterior and lateral margins of the nasopharynx are enclosed by the middle layer of the deep cervical fascia (MLDCF), which encases the outer margins of the pharyngobasilar fascia and separates the nasopharynx from the parapharyngeal, perivertebral, and retropharyngeal spaces (RPS).

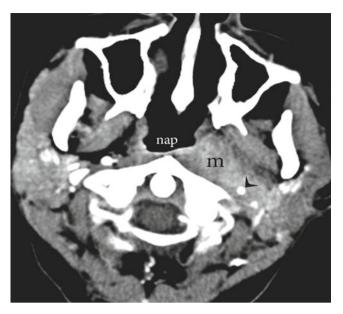


Fig. 2.15 Nasopharyngeal carcinoma. Axial CECT demonstrates a left nasopharyngeal mass (*m*) with extension to the left retropharyngeal space. The mass surrounds and laterally displaces the left internal carotid artery (*black arrow*). Nasopharynx (*nap*)



Fig. 2.16 A radiographic image at the level of the oropharynx. Appendix B at the end of the chapter has the list of abbreviations for each structure highlighted

Pathologies encountered within the nasopharynx include both benign and malignant lesions. Bengin lesions include Thornwaldt cysts and teratomas and the most common malignant neoplasm in this area is nasopharyngeal carcinoma (Fig. 2.15).

Soft tissue asymmetry is a commonly encountered finding on imaging, particularly in the fossa of Rosenmüller and tonsillar regions. Evaluation of the adjacent fat planes and correlation with direct physical inspection by the referring physician can be invaluable in distinguishing benign asymmetry from a true mass. On CT and MRI, particular attention should also be paid to the adenoidal soft tissues, located along the posterior wall of the nasopharynx, as these tissues should gradually regress in size with advancing age. While prominence of the adenoids is considered a normal finding in children and younger adults, a similarly prominent appearance in a 30- to 40-year old could represent residual adenoids, recent upper respiratory tract infection, lymphoid hyperplasia, or even lymphoma.

Oropharynx

The oropharynx represents the suprahyoid (SH) visceral compartment posterior to the oral cavity, and its components are visualized as the posterior structures during open mouth examination. The oropharynx is bound by the nasopharynx and soft palate superiorly, circumvallate papillae, and anterior tonsillar pillars anteriorly, superior and middle constrictor muscles posteriorly and the larynx and hypopharynx inferiorly. The anatomic boundary between the larynx and oropharynx is formed by the epiglottis and glossoepiglottic fold, while the pharyngoepiglottic fold forms the boundary between the hypopharynx and oropharynx (Fig. 2.16). It should also be noted that the circumvallate papillae divides the tongue into the oral tongue (anterior two thirds of the tongue) from the base of tongue (posterior one third of the tongue).

Components of the oropharynx include the pharyngeal mucosa, minor salivary glands, soft palate and uvula, base of tongue, lingual tonsils, palatine (faucial) tonsils, the superior and middle constrictor muscles, and minor salivary glands. The oropharynx also contains the anterior tonsillar pillars (mucosal fold overlying the palatoglossus muscle) and the posterior tonsillar pillars (mucosal fold overlying the palatopharyngeus muscle). The oropharynx is divided into the following subsites when describing malignancies in this area: soft palate, tonsils, base of tongue, and posterior pharyngeal wall. Recent literature has shown that there is an increase in the incidence of oropharyngeal squamous cell carcinoma and that this is due to an increase in HPV-16-related cancer of the oropharvnx^[4]. The strongest association between HPV infection and oropharyngeal carcinoma has been demonstrated in tonsillar cancer.

The oropharynx is vulnerable to many potential disease processes, including infections, benign neoplasms, and malignancies. Infections often seen in the oropharnx include tonsillitis and peritonsillar abscess (Fig. 2.17).

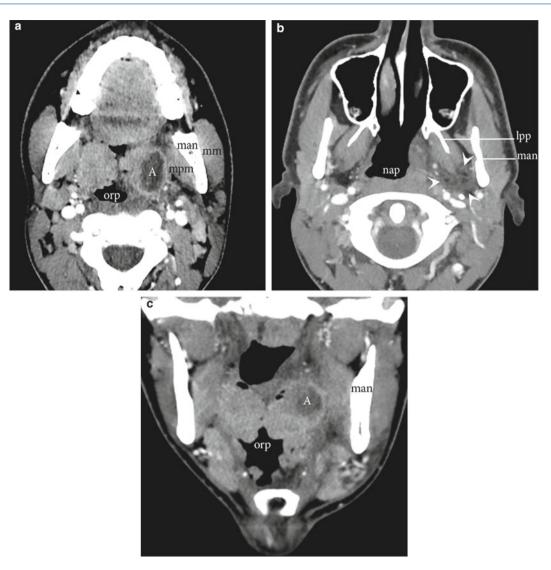


Fig. 2.17 Peritonsillar abscess. Axial (a, b) and coronal (c) CECT images demonstrate a rim-enhancing left-sided peritonsillar abscess (A) in this child with odynophagia and fever. There is resultant narrowing of pharynx and "kissing" appearance of the enlarged tonsils. There

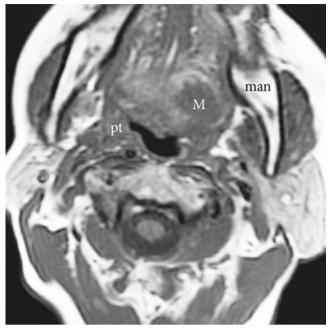
Benign neoplasms include papillomas and pleomorphic adenomas and malignant neoplasms are most commonly squamous cell carcinoma (Figs. 2.18 and 2.19). Evaluation for palatine tonsil and tonsillar wall pathology is a very common indication for cross-sectional head and neck imaging, and assessment of these structures is especially important.

Hypopharynx

The hypopharynx is the inferior continuation of the nasopharynx and oropharynx and extends from the level of the hyoid bone and vallecula inferiorly to the level of the cricopharyngeus muscle and inferior margin of the cricoid cartilage (Fig. 2.20).

is infiltration of left parapharyngeal fat (*white arrows*). Medial pterygoid muscle (*mpm*, **a**), mandible (*man*, **a**, **b**, **c**), masseter muscle (*mm*, **a**), oropharynx (*orp*, **a**, **c**), lateral pterygoid plate (*lpp*, **b**), nasopharynx (*nap*, **b**)

Its contents include the pharyngeal mucosa, minor salivary glands, and the inferior pharyngeal constrictor muscles. The hypopharynx is further divided into subsites, including the pyriform sinuses, posterior hypopharyngeal wall, and the postcricoid area. The pyriform sinuses form the recesses lateral to the aryepiglottic folds and medial to the thyroid cartilage and thyrohyoid membrane. The inferior tip of each pyriform sinus is referred to as the pyriform apex or recess and is located at the level of the true vocal cord. Posteriorly, the pyriform sinuses are bounded by the lateral aspect of the posterior hypopharyngeal wall, a continuation of the dorsal oropharyngeal wall extending from the hyoid bone and vallecula to the level of the cricoarytenoid joints. The postcricoid area is also known as the pharyngo-esophageal junction. It forms the anterior wall of the inferior hypopharynx and



scm pyr ijv

Fig. 2.18 Base of tongue SCC: axial post-contrast T1-weighted MRI demonstrates a rim-enhancing mass (M) at the left base of tongue. Right palatine tonsil (*pt*), mandible (*man*)

Fig. 2.20 Radiographic and schematic image at level of hypopharynx

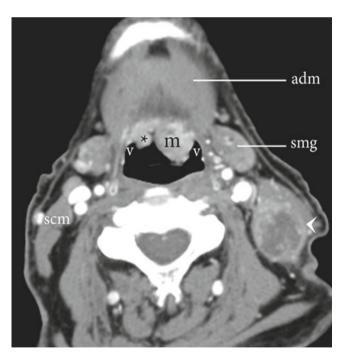


Fig. 2.19 Lingual tonsillar SCC: axial CECT demonstrates an enhancing mass originating from the left lingual tonsil (*m*) with a left cervical lymph node metastasis (*arrow*). Valleculae (*v*), right lingual tonsil (*asterisk*), anterior belly of digastric muscle (*adm*), submandibular gland (*smg*), sternocleidomastoid muscle (*scm*)

covers the area spanning the cricoarytenoid joints to the level of the cricopharyngeus muscle and inferior margin of the cricoid cartilage. Pathologies found within the hypopharynx include neoplasms (squamous cell carcinoma and minor salivary gland tumors) and retention cysts (Fig. 2.21).

Larynx

The larynx resides in the visceral compartment of the infrahyoid (IH) neck and is important in airway maintenance, protection against aspiration and phonation. It is divided into supraglottic, glottic, and subglottic regions (Fig. 2.20).

The cricoid, thyroid, and arytenoid cartilages form the structural foundation of the laryngeal soft tissues. The thyroid cartilage is an inverted V-shaped structure that progressively, but variably ossifies with age. It is composed of two alae, which merge in the midline and form a small superior indention called the superior thyroid notch. Posterior projections arising from the thyroid cartilage are called the cornua and serve as attachments to the thryohyoid ligament and hyoid bone superiorly and the articulation with the cricoid cartilage (cricothyroid joint) inferiorly. The cricoid cartilage is located inferior to the thyroid cartilage and forms a

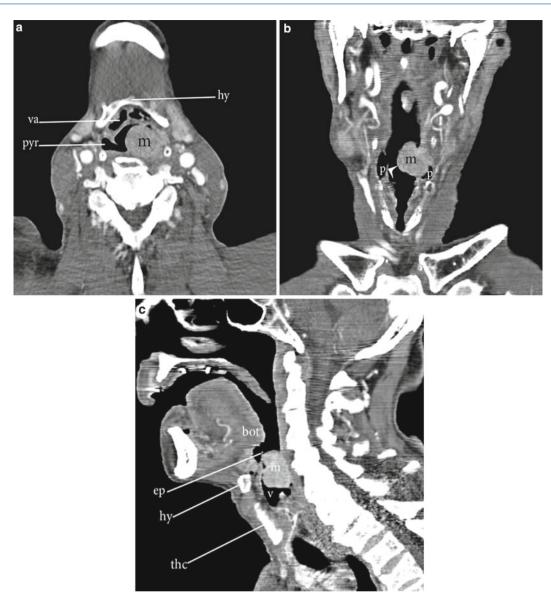


Fig. 2.21 Hypopharyngeal SCC: axial (**a**), coronal (**b**) and sagittal (**c**) CECT demonstrates an enhancing mass (m) along the left posterior hypopharyngeal wall, extending into the left pyriform sinus (p).

Laryngeal vestibule (v, sagittal); epiglottis (*white arrow*). Hyoid bone (*hy*, **a**, **c**), pyriform sinus (*pyr*, **a**), vallecula (*va*, **a**), thyroid cartilage (*thc*, **c**), base of tongue (*bot*, **c**), epiglottis(*ep*, **c**)

complete ring around the endolarynx and trachea. It has two portions, the anterior arch and posterior lamina. The arytenoid cartilages are the smallest of the laryngeal cartilages and represent the triangular or "comma" shaped structures perched laterally on top of the midline, posterior cricoid cartilage lamina (Fig. 2.22).

The supraglottic larynx is bordered superiorly by the oropharynx and extends cephalocaudally from the glossoepi-glottic and pharyngoepiglottic folds to the level of the laryngeal ventricles below (Fig. 2.23).

Laterally, it is bound by the pyriform sinuses (which are hypopharyngeal components) and is separated from these

structures by the lateral aspects of the aryepiglottic folds (i.e., marginal zones). It contains the epiglottis, vestibule, false vocal cords, pre-epiglottic space, paraglottic space, aryepiglottic folds, and arytenoid cartilage. The mobile epiglottic cartilage is located superiorly and protectively covers the laryngeal airway during swallowing. It is sometimes further divided into SH (free margin) and IH (fixed portion) components. The base of the epiglottis is called the petiole, which is attached to the thyroid cartilage by means of the thyroepiglottic ligament. The hypoepiglottic ligament serves as the attachment of the epiglottis to the hyoid bone and is covered by the mucosal glossoepiglottic fold. The fat containing pre-epiglottic space is located between the hyoid bone and epiglottis. Inferiorly, it merges with the paired paraglottic spaces. The laryngeal ventricle is the air space separating the false vocal cords above and true vocal cords below. The aryepiglottic folds represent the lateral margins of the supraglottic larynx and are formed by soft tissues projecting from the superior aspect of the arytenoid cartilage to the inferolateral aspect of the epiglottis (Fig. 2.24).

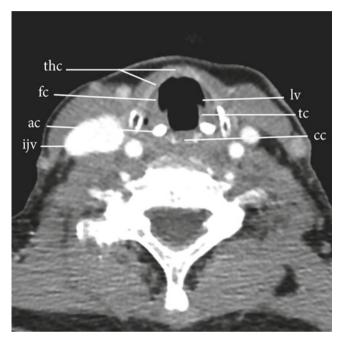


Fig. 2.22 Cross section at level of supraglottis at junction of false vocal cords and laryngeal ventricle

The glottic larynx extends from the apex of the laryngeal ventricles to a theoretical axial plane one centimeter inferior to the laryngeal ventricle apex. It contains the true vocal cords (glottis). The true vocal cords arise anteriorly from ventral projections off the arytenoid cartilage called the vocal processes. This anatomic relation is important because the cricoarytenoid joint is easily visible on CT and serves as the marker for the level of the true vocal cords. The vocal ligaments are paired fibrous bands that are located in the medial margins of the true vocal cords. The point at which the true vocal cords meet anteriorly is called the anterior commissure. The posterior commissure is also within the glottic larynx

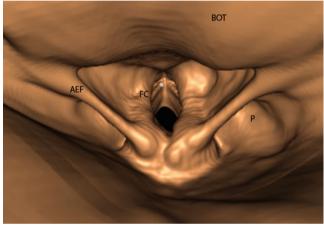


Fig. 2.23 Virtual laryngoscopy reconstructed from volumetric CT data, demonstrating normal anatomy. Aryepiglottic fold (*aef*), pyriform sinus (*p*), false cord (*fc*), true cord (*asterisk*), base of tongue (*bot*)

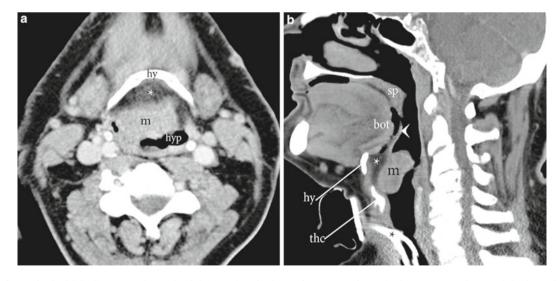


Fig. 2.24 Supraglottic SCC. Contrast-enhanced axial (**a**) and sagittal (**b**) CT demonstrates a mass (m) inseparable from the base of the epiglottis, protruding into the laryngeal vestibule and supraglottic airway. Axial images suggest early extension into the pre-epiglottic fat

(*white asterisk*) and airway narrowing. Epiglottis (*white arrow*); tracheostomy tube (*black asterisk*). Hyoid (*hy*), thyroid cartilage (*thc*), soft palate (*sp*), base of tongue (*bot*). hypopharynx (*hyp*)

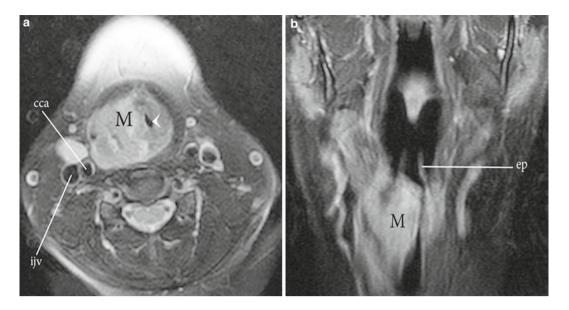


Fig. 2.25 Laryngeal SCC. Axial (**a**) and coronal (**b**) fat suppressed T2-weighted MR images demonstrate a hyperintense right supraglottic and glottic mass (M), compressing the airway (*arrow*). Common carotid artery (*cca*, **a**), internal jugular vein (*ijv*, **a**), epiglottis (*ep*, **b**)

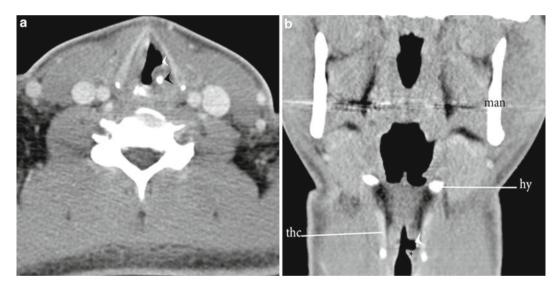


Fig. 2.26 Vocal cord paralysis. Axial (**a**) and coronal (**b**) CECT demonstrate medialization of the left vocal cord (*asterisk*) with associated ipsilateral ventricle enlargement (*white arrow*), compatible with

left vocal cord paralysis. The left arytenoid cartilage is medially rotated (*black arrow*). Thyroid cartilage (*thc*, **b**), hyoid bone (*hy*, **b**), mandible (*man*, **b**)

and characterizes the mucosal surface anterior and medial to the cricoid and arytenoid cartilages. Normally, the anterior and posterior commisures measures up to 1 mm in thickness. The subglottic larynx extends inferiorly from the glottic larynx to the lower edge of the cricoid cartilage. In children, the narrowest portion of the airway is the subglottis, while in adults, it is at the level of the true vocal cords (glottis).

The conus elasticus and the quadrangular membrane are two fibrous membranes in the larynx that are of clinical significance. The conus elasticus extends from the cricoid cartilage to the true vocal cords and superiorly, its free edge forms the vocal ligaments. The quadrangular membrane extends from the aryepiglottic fold to the ventricular fold (false vocal cord). Both structures serve to provide structural integrity to the larynx, and they are also important barriers in the spread of malignancy.

Radiologic evaluation of the larynx is usually performed to assess for hoarseness, laryngeal mass (such as squamous cell carcinoma; Fig. 2.25), vocal cord paralysis (recurrent laryngeal nerve injury; Fig. 2.26), and trauma. Spiral CT is usually the radiologic test of choice for evaluating the larynx, although MRI is also useful (particularly in assessing extent of cancer

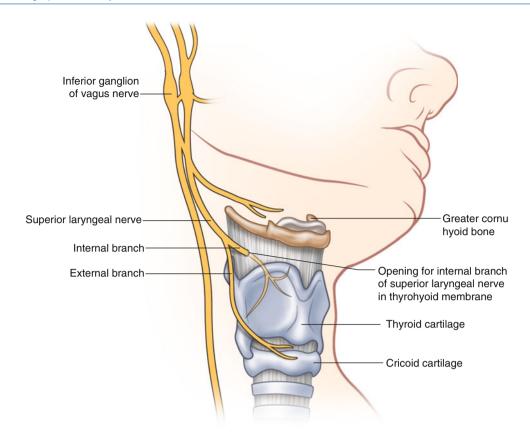


Fig. 2.27 The course of the superior laryngeal nerve once it leaves the vagus nerve

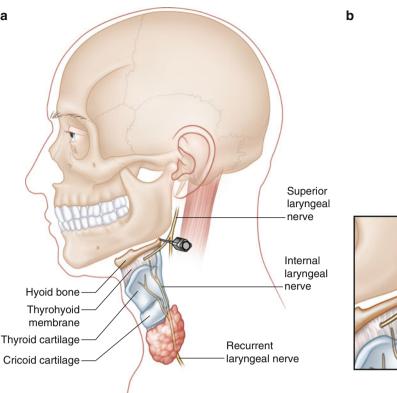
involvement and invasion into adjacent tissues). Evaluation of laryngeal pathology can be aided by thin section imaging (1 mm axial images) for a more thorough evaluation of the glottis and surrounding structures. Overall, CT and MRI can be useful in identifying pathology within physical exam blind spots including the pre-epiglottic and paraglottic spaces.

Superior Laryngeal Nerve Block

The laryngeal mucosa is very sensitive and the slightest irritation can cause a very strong cough and gag reflex. Therefore, superior laryngeal nerve blocks can be very useful for many procedures that manipulate the larynx. In difficult airway situations that require endoscopic intubation in an awake patient, performing a superior laryngeal nerve block will help to provide comfort to the patient and reduce these reflexes. This nerve block is also useful in laryngoplasty procedures where only local anesthesia is given. In these procedures, it is preferable to have the patient awake so that the surgeon can get instant feedback on the patient's voice quality.

The superior laryngeal nerve is a branch of the vagus nerve. After branching from the vagus nerve, it further divides into an internal and external branch. The internal branch of the superior laryngeal nerve runs anteriorly and pierces the thyrohyoid membrane to provide sensory fibers from the upper surface of the vocal cords to the base of tongue. The external branch of the superior laryngeal nerve runs inferiorly along the posterolateral aspect of the thyroid cartilage to provide motor fibers to the cricothyroid muscle and inferior constrictor muscle (Fig. 2.27). To provide local anesthesia, the superior laryngeal nerve can be blocked at the internal branch as it enters the thyrohyoid membrane. To perform this nerve block, first palpate for the hyoid bone, the greater cornu of the hyoid bone, and thyroid cartilage. Then insert the needle perpendicular to the skin, halfway between the hyoid bone and thyroid cartilage and approximately 1-1.5 cm anterior to the greater cornu of the hyoid bone (Fig. 2.28). Advance the needle until resistance is felt as the needle pierces the thyrohyoid membrane. At this point, 2-3 ml of local anesthetic, generally 2% lidocaine, can be injected [5].

Fig. 2.28 The surgical technique and injection location for a superior laryngeal nerve block. A more detailed description of the procedure is later described





Deep Spaces of the Neck (Fig. 2.29)

Neck Overview and Cervical Fascia

Traditionally, anatomists and clinicians have divided the neck into triangles to help to aid in the physical exam evaluation and to help describe pathologies of the neck. The sternocleidomastoid muscle, which runs obliquely from the mastoid process and occipital bone to the sternum and clavicle, divides the neck into anterior and posterior triangles. Therefore, the boundaries of the anterior triangle include the posterior border of the sternocleidomastoid muscle, the midline of the neck, and the mandible. The anterior triangle can be further divided into the submental, diagastric, carotid, and muscular triangles and the posterior triangle can be subdivided into the occipital and supraclavicular triangles. The borders of the submental triangle are the anterior belly of the digastrics muscle, the midline of the neck, and the hyoid bone. The diagastric (also called the submandibular) triangle is bordered by the anterior and posterior bellies of the diagstric muscle and the mandible. The carotid triangle is bordered by the sternocleidomastoid muscle, the posterior belly of the digastric muscle, and the omohyoid muscle. The muscular triangle is bordered by the omohyoid muscle, the sternoceidomastoid muscle, and the midline of the neck.

The occipital triangle is bordered by the sternocleidomastoid muscle, the trapezius muscle, and the omohyoid muscle. The supraclavicular triangle is bordered by the sternocleidomastoid muscle, the omohyoid muscle, and the clavicle ^[6].

Although helpful clinically, the triangles of the neck are not as useful radiographically to describe anatomical structures and pathologies. Instead, the neck is divided in the SH and IH neck based on the easily visualized hyoid bone on imaging, and the neck is then further divided into deep neck spaces, which is helpful to use when looking at crosssectional imaging.

The SH neck spans from the skull base to the hyoid bone, excluding orbit, sinuses, and the oral cavity. The IH neck extends from below the hyoid bone into the superior mediastinum. Three layers of deep cervical fascia cleave the neck into anatomic spaces. There are lateral paired spaces of the neck and midline posterior spaces. The lateral paired spaces include the parapharyngeal, buccal, masticator, parotid, and posterior cervical space (PCS). The midline posterior spaces consist of anterior cervical, posterior cervical, visceral, retropharyngeal and perivertebral spaces (PVS).

The superficial cervical fascia (SCF) is a primarily fatty layer of relatively loose connective tissue that lies between the dermis and the deep cervical fascia. It covers the head, face, and neck and contains the platysma muscles, muscles of facial expression, and portions of the anterior external

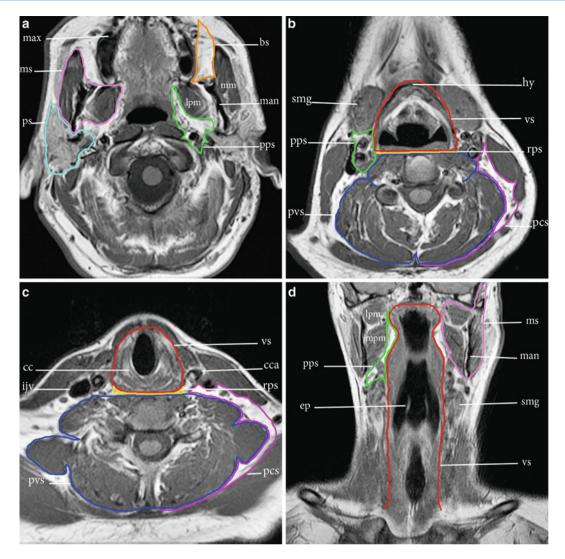


Fig. 2.29 Deep spaces of the neck are outlined on serial axial T1-weighted MR images. Parotid space (*ps*), masticator space (*ms*), parapharyngeal space (*pps*), buccal space (*bs*), visceral space (*vs*), retropharyngeal space (*rps*), perivertebral (*pvs*), posterior cervical space

(*pcs*), hyoid bone (*hy*), submandibular gland (*smg*), cricoid cartilage (*cc*), internal jugular vein (*ijv*), common carotid artery (*cca*), epiglottis (*ep*), mandible (*man*), maxilla (*max*), lateral pterygoid muscle (*lpm*), master muscle (*mm*)

jugular veins. Subcutaneous nerves, venules, and lymphatics are also within the SCF. In the scalp, the SCF encompasses the frontalis muscle.

The superficial layer of deep cervical fascia (SLDCF) completely encircles the neck, attaching posteriorly to the vertebral spines and ligamentum nuchae, caudally to the manubrium and laterally to the clavicles and scapulae. SLDCF surrounds the masticator and parotid spaces (PS) as well as part of the carotid sheath in the SH neck and invests the strap, sternocleidomastoid and trapezius muscles in the IH neck.

The median layer of the deep cervical fascia (MLDCF) originates from the skull base in the SH neck, where it is associated with tensor and levator velli palatini muscles and the superior pharyngeal constrictors. The MLDCF extends

caudally behind the sternum, fuses with the fibrous pericardium, and is prolonged out along the adventitia of great vessels. The MLDCF defines the deep margin of pharyngeal mucosa in the SH neck and circumscribes the visceral space (VS) in the IH neck. It is associated with the strap muscles and contributes to the carotid sheath in both the SH and IH neck. Visceral fascia in the IH neck and buccopharyngeal fascia in the SH neck are alternative terms used to describe the MLDCF.

The deep layer of deep cervical fascia (DLDCF) originates from cervical vertebral spinous processes and ligamentum nuchae and extends from the skull base to the coccyx. It surrounds the PVS, investing the splenius capitis, levator scapulae, and scalene muscles. DLDCF contributes to the carotid sheath in both the SH and IH neck. The prevertebralor dorsal portion of DLDCF covers the anterior surface of longus capitis and colli muscles. The alar or ventral portion of DLDCF separates the RPS from the so-called danger space by providing the lateral and posterior wall to the RPS and the lateral wall to the danger space. A small, sagitally oriented fascial sheet that separates the retropharyngeal and danger spaces from the more lateral PPSs is known as cloisonsagittale. Sibson's fascia is the portion of DLDCF that extends laterally from the transverse process of the seventh cervical vertebrae, covering the dome of the pleura, attaching to the medial aspect of the first rib and separating the neck from the thorax. Tensor-vascular-styloid fascia is located between the SLDCF and DLDCF, immediately contiguous with the buccopharyngeal fascia portion of the MLDCF. It contains the ascending palatine artery and vein and divides the PPS into pre-styloid and poststyloid compartments^[7].

Cervical Plexus Nerve Block

The cervical plexus transmits sensory input from the neck. Cervical plexus blocks can be useful in surgical procedures including neck dissections, parathyroidectomy and thyroidectomy. It has been shown to reduce intraoperative general anesthetic requirements and post-operative analgesic use ^[8].

The cervical plexus forms from spinal nerves C2, C3, and C4 and emerge between the anterior and middle scalene muscles. From here, they divide to form two plexi, the superficial cervical plexus and the deep cervical plexus. The deep cervical plexus curves around the lateral border of the anterior scalene muscle to traverse inferiorly and medially, providing motor fibers to the neck and posterior scalp. The superficial plexus nerve fibers are sensory fibers that innervate the anterior and posterior cutaneous tissues of the neck. These fibers continue laterally from the scalene muscles to emerge superficial to the sternocleidomastoid muscle at its posterior border. The superficial plexus can be blocked at the point where it emerges from the SCM. To perform this nerve block, first place the patient in a supine position with their head turned away from the side that is being blocked. Then palpate for the posterior border of the SCM and locate the point that is midway between the mastoid process and the clavicle. At this point, inject 5 ml of local anesthetic with the needle directed superiorly, along the posterior border of the SCM. Then inject another 5 ml of anesthetic inferiorly. Alternatively, ultrasound guidance can be used to find the plane between the SCM and scalene muscles and the injection is placed in the intramuscular plane. Tran et al. in a prospective, randomized trial did not find any differences in success rate with either technique ^[9].

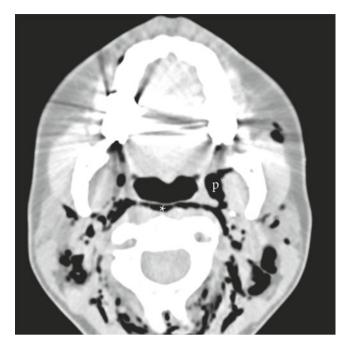


Fig. 2.30 Axial CT, displayed with a "lung window" to highlight air, demonstrates extensive emphysema within the subcutaneous and deep neck spaces in this patient who also had extensive pneumomediastinum. Air fills and clearly delineates the left parapharyngeal space (p) and retropharyngeal space (*asterisk*)

Lateral Paired Spaces

Parapharyngeal Space

The parapharyngeal space (PPS) begins at the level of the inferior jugular foramen and the carotid canal at the skull base and extends down to the greater cornu of the hyoid bone. It is a pyramidal shaped space, with the base of the pyramid at the level of the skull base and the apex at the greater cornu of the hyoid bone (Fig. 2.30).

All three layers of deep cervical fascia contribute to the PPS. The PPS is divided by the tensor veli palatini fascia and tendon (or tensor-vascular-styloid fascia) into pre-styloid and poststyloid compartments^[10]. The pre-styloid compartment of the PPS is located lateral to the pharynx and posteriomedial to the masticator space (MS). Contents of the pre-styloid compartment include the tail of the parotid gland, minor salivary glands, internal maxillary artery, ascending pharyngeal artery, pharyngeal venous plexus, and branches of the mandibular division of the trigeminal nerve. The poststyloid compartment of the PPS is also known as the carotid space, and this space continues inferiorly into the IH neck as the carotid sheath. It encompasses cranial nerves nine through twelve, cervical sympathetic chain, glomus bodies, internal jugular vein, common carotid artery in its IH aspect and the internal carotid artery in its SH aspect (Figs. 2.31 and 2.32).



Fig. 2.31 Glomus vagale tumor. Axial CECT demonstrates an avidly enhancing mass (*asterisk*) occupying the retro-styloid compartment of the left parapharyngeal space, displacing the internal (*white arrow*) and external (*black arrow*) carotid arteries anteriorly, compressing and laterally displacing the internal jugular vein (open arrow) and pushing the pharyngeal wall medially. Sternocleidomastoid muscle (*scm*), oropharynx (*OrP*), floor of mouth (*FoM*)



Fig. 2.32 Second branchial cleft cyst. Axial CECT demonstrates a fluid density mass (*asterisk*) on the right side of the neck displacing the submandibular gland (smg) anteriorly and the sternocleidomastoid muscle (scm) posteriorly. The carotid vessels (*arrow*), within the retrostyloid compartment of the parapharyngeal space, are displaced medially

Tumors of the pre-styloid compartment of the PPS are most commonly salivary gland neoplasms (which arise from both the tail of the parotid gland and minor salivary glands) and neoplasms of the post-styloid compartment of the PPS are usually neurogenic lesions (schwannoma, paraganglioma, neurofibroma). When imaging the PPS with MR, TI precontrast sequences are important for depicting high velocity flow voids of paragangliomas. CT and MR arteriograms and venograms are useful in evaluating for vascular pathology.

Masticator Space

The masticator space (MS) is located anterolateral to the PPS in the SH neck and is surrounded by the SLDCF. The medial fascial slip runs along the deep surface of the pterygoid muscles, inserting just medial to foramen ovale, while the lateral slip covers the surface of masseter muscle, attaching to the zygomatic arch. The craniocaudal extent of the MS is from high parietal calvarium to the mandibular angle. The masseter space contains the ramus and condyle of the mandible, the third division of the fifth trigeminal cranial nerve (V3) and muscles of mastication (masseter, temporalis, medial, and lateral pterygoids). The MS encompasses foramina ovale and spinosum and contains the temporomandibular joint.

Pterygopalatine and Infratemporal Fossa

The pterygopalatine and infratemporal fossa are located within the MS. The infratemporal fossa is the portion of the MS deep to the zygomatic arch and superficial to the pterygomaxillary fissure, while the pterygopalatine fossa is medial to the pterygomaxillary fissure.

The pterygopalatine fossa is located anterior to the pterygoid plates and dorsal to the posterior maxillary sinus wall. The anatomic importance of the pterygopalatine fossa lies in the fact that it serves as the junction of numerous foramina and spaces within the skull base, orbit, nasal cavity, and upper neck. These include the sphenopalatine foramen (connecting to the posterolateral aspect of the superior nasal meatus), pterygomaxillary fissure (extending to the MS), inferior orbital fissure (communicating with the superior orbital fissure and orbit), greater and lesser palatine foramina/canals (leading into oral cavity), foramen rotundum (leading to the cavernous sinus and skull base), and the vidian canal (serving as a conduit into the skull base and middle cranial fossa).

The MS can be easily imaged with both CT and MR. If a MS mass is identified, a search for perineural spread of tumor on V3 is mandatory and is best performed with contrastenhanced multiplanar MRI. The imaging field of view should be extended from the level of the lateral pons (including foramen ovale and Meckel's cave) to the mental foramen of the mandible.

Trigeminal Nerve Blocks

The trigeminal nerve (cranial nerve V) supplies sensory innervation to the head and neck and it also provides motor function to the muscles of mastication. The nerve has an intimate relationship with the pterygopalatine and infratemporal fossa and its branches serve as targets for regional nerve blocks in the head and neck. Therefore, anatomy of the trigeminal nerve will be reviewed here in detail.

The trigeminal nerve originates from several brainstem nuclei, which converge in the trigeminal (Gasserian) ganglion located in Meckel's cave. Meckel's cave is a dural invagination found within the petrous portion of the temporal bone. The postganglionic fibers give rise to the ophthalmic (V1), maxillary (V2), and mandibular (V3) nerves. Branches of the trigeminal nerve serve as targets for many potential regional nerve blocks (Fig. 2.33) ^[11]. Blockage of the trigeminal ganglion is also possible, but is reserved for treatment of refractory trigeminal neuralgia and will not be further discussed here ^[12–14].

The ophthalmic nerve (V1) enters the orbit through the superior orbital fissure. Just prior to entering the orbit, it branches into the nasociliary, lacrimal, and frontal nerves. The nasociliary nerve branches into the anterior and posterior ethmoidal nerves. The anterior ethmoidal nerve enters the nasal cavity through the anterior ethmoidal foramen to provide sensation to the anterior nasal cavity mucosa. It then terminates as the nasal branch and exits through the nasal bones to provide sensation to the external nose. The posterior ethmoidal nerve exists through the posterior ethmoidal foramen to provide sensation to the posterior ethmoid air cells and the sphenoid sinus. The lacrimal nerve courses through the orbit to provide innervation to the lacrimal gland, located in the lateral and superior aspect of the orbit. The frontal nerve passes through the orbit and exits as its terminal branches, the supraorbital and supratrochlear nerves, which are of interest because they serve as easily accessible nerves for regional anesthesia.

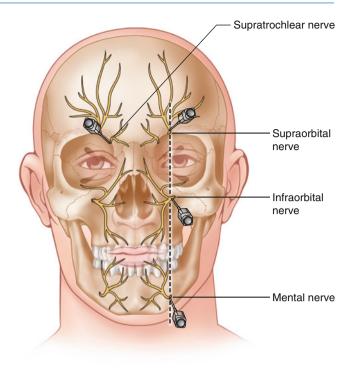


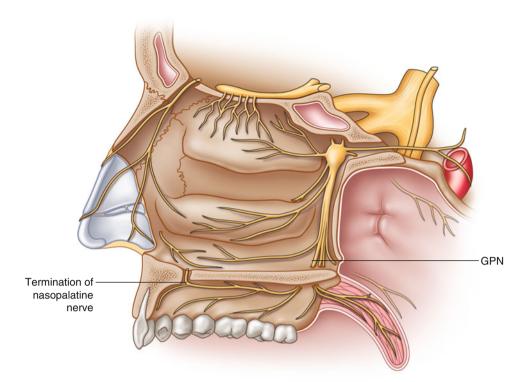
Fig. 2.33 The end branches of the trigeminal nerve are derived from all three divisions of the trigeminal nerve: supratrochlear/supraorbital (V1), infraorbital (V2), and mental (V3) nerves

The supraorbital nerve exits the orbit through the supraorbital notch (or foramen), which can be palpated approximately 2.5 cm lateral to the midline of the face along the superior orbital rim in the upper border of the orbit ^[15]. The supraorbital notch can also be found by palpating the area of the supraorbital rim directly above the pupil when looking straight ahead. The supraorbital nerve allows for sensory input from the upper eyelid, forehead, and scalp.

The supratrochlear nerve exits the orbit approximately 1 cm medial to the supraorbital notch at the orbital rim ^[14]. It provides innervation to the medial portion of the upper eyelid and lower forehead.

A supraorbital and/or supratrochlear nerve block is often useful for surgery involving the lower forehead and upper eyelid. To perform this nerve block, first palpate and mark the supraorbital notch. The supraorbital nerve can be blocked by injecting 2–3 ml of local anesthetic at the supraorbital notch. Care must be taken to avoid direct injection into the bony notch/foramen, as this can cause pressure-induced or needleinduced nerve injury ^[15]. The supratrochlear nerve can be blocked by injecting 2–3 ml of local anesthetic at the orbital rim approximately 1 cm medial to the supraorbital notch ^[15].

The maxillary nerve exits the skull base through the foramen rotundum to enter the pterygopalatine fossa, where it divides into several branches. The zygomatic branch, which further divides into the zygomaticotemporal and zygomaticofacial nerve, innervates the skin over the temple and **Fig. 2.34** The greater palatine nerve (GPN) is derived from the maxillary division of the trigeminal nerve (V2) and can be accessed via a transoral route through the greater palatine foramen located at the junction of the hard and soft palate.



zygomatic arch. Pterygopalatine branches from V2 travel to the pterygopalatine ganglion and the postganglionic fibers form the greater and lesser palatine nerves. The greater palatine nerve gives off branches to innervate the inferior nasal cavity mucosa and then exits through the greater palatine foramen to provide sensation to the hard palate. The lesser palatine nerve also provides branches to the nasal cavity mucosa and then travels through the lesser palatine foramen to innervate the soft palate and tonsils. The infraorbital nerve, another branch of V2, travels through the pterygopalatine fossa to enter the infraorbital canal of the maxillary bone where it gives off several branches to innervate the upper teeth, palate, and nasal cavity mucosa. It then exits the maxilla through the infraorbital foramen and terminates into several branches that innervate the lower eyelids, cheek, and upper lip.

The infraorbital nerve and greater palatine nerve are both amenable to nerve blocks. Bilateral infraorbital nerve blocks are often useful in upper lip surgery, nasal work such as closed reduction of a nasal fracture, as well as dental work on the maxillary teeth. Greater palatine nerve blocks are useful for both dental procedures and endoscopic sinus surgery. Used in combination with general anesthesia, this nerve block helps to provide both local anesthesia and vasoconstriction for the endoscopic sinus surgeon. The vasoconstriction is due to the spread of the local anesthetic into the pterygopalatine fossa, where the vasocontstricting agent in the anesthetic (i.e., epinephrine) acts on the internal maxillary artery and its terminal branches ^[16].

The infraorbital nerve block can be performed from a transcutaneous or intraoral approach ^[15, 17]. The infraorbital nerve can be located by palpating for the infraorbital foramen, which is located 2.5 cm from the midline of the face, just inferior to the orbit ^[17]. To perform the nerve block transcutaneously, insert the needle 1 cm inferior to the foramen and advance in the direction of the foramen. Keeping the needle directed upward and lateral to the foramen to avoid passing the needle through the foramen and into the orbit, inject 2-4 ml of local anesthetic ^[15]. Prior to injection, make sure to pull back on the syringe to confirm that the needle is not in a vessel, as the facial artery is also in this area. In the transoral approach, the needle is inserted into the superior buccal groove and directed superiorly and laterally until the tip of the needle is felt under the skin by the finger palpating for the foramen^[17].

The greater palatine nerve block is performed transorally. The greater palatine foramen can be found just anterior to the junction of the hard and soft palate, medial to the maxillary second or third molar (Fig. 2.34) ^[18]. To perform this nerve block, first place a 45° bend in the needle approximately 2.5 cm from the tip of the needle. The needle tip, once in the foramen, should feel as though it has fallen into a space. The needle should then be advanced no further than the length of the bent portion and 2 ml of local anesthetic should be given ^[19]. If the needle is in the correct position, there should be moderate resistance when injecting the local anesthetic and blanching of the hard palate should be seen.

The mandibular nerve, which is the largest of the three branches of the trigeminal nerve, exits the skull base through the foramen ovale. It then enters the infratemporal fossa and divides into anterior and posterior divisions. The anterior division of V3 supplies motor innervation to the muscles of mastication. The posterior division provides sensory innervation through three branches: the auriculotemporal nerve, lingual nerve, and inferior alveolar nerve. The inferior alveolar nerve further divides into its terminal branch, the mental nerve. The auriculotemporal nerve provides sensory input from the skin over the pre-auricular area, the lingual nerve provides sensation from the anterior two-thirds of the tongue, and the inferior alveolar nerve innervates the mandibular teeth, buccal mucosa, gingiva, and mandible.

The inferior alveolar nerve is commonly blocked for dental procedures as it enters the mandible through the mandibular foramen. In the direct intraoral approach for nerve blocking, the mandibular foramen can be found approximately midway between the dental arches when the mouth is wide open. A divot in the mucosa of this area, called the pterygotemporal depression, is the site for needle penetration. The surrounding landmarks include the coronoid notch of the mandible anteriorly and the pterygomandibular fold posteriorly. The pterygomandibular fold is a vertical band of mucosa that can be found posterior to the last mandibular molar tooth and extends superiorly to where the hard and soft palates meet. The needle should be advanced 2-2.5 cm beyond the buccal mucosa overlying the pterygotemporal depression and the local anesthetic can then be injected ^[20].

The terminal branch of the inferior alveolar nerve, the mental nerve, is also often a target for local nerve block. As the inferior alveolar nerve emerges through the mental foramen, it becomes the mental nerve, which innervates the chin and lower lip. To perform this nerve block, the mental foramen can be located by finding the intersection between a vertical line running through the pupil looking straight ahead and a horizontal line midway between the upper and lower borders of the mandible. The mental nerve can be blocked intraorally or from the external skin. In the intraoral approach, the needle is placed between the two premolar teeth immediately below the tooth root apices and 2-3 ml of anesthetic is then injected [21]. In the extra-oral approach, the mental foramen can be found with the landmarks described earlier. The canal angles medially and inferiorly, so approach the foramen by entering the skin 0.5 cm lateral and superior to the foramen and angle the needle toward the foramen. Again, 2-3 ml of local anesthetic is then injected ^[15].

Buccal Space

The buccal space (BS) is a primarily fat-filled space of the mid-face forming padding of the cheeks. It has no definite fascial boundaries and is bordered by the buccinator muscle medially, muscles of facial expression laterally, masseter and pterygoid muscles, mandible and parotid gland posteriorly and the retromaxillary fat pad superiorly. The parotid duct traverses the BS, piercing it at the level of the maxillary second molar. Accessory parotid glands can be present in the BS. The BS also contains lymph nodes.

Either CT or MR can be used for evaluation of the BS with particular attention to asymmetry of fat density or T1 signal intensity as masses can be subtle, especially if adjacent to the mandible or maxilla. Dental amalgam may obscure BS disease on both CT and MR. The majority of BS pathology is nodal disease or extension from adjacent structures and spaces.

Parotid Space

The parotid space (PS) is invested in the SLDCF and is located lateral to the PPS in the SH neck. The PS extends from the external auditory canal and mastoid tip superiorly to below the angle of mandible inferiorly. It is lateral to the PPS and posterior to the MS. The upper PS is separated from the parapharyngeal space by the posterior belly of the digastric muscle. The parotid tail is the inferior-most aspect of the PS, defined by the superficial plastysma and deep sternocleidomastoid muscles as well as the PPS. The PS contains the parotid gland, external carotid artery, retromandibular vein, and the extracranial portion of the seventh (facial) cranial nerve. The superficial lobe of the parotid gland represents $\sim 2/3$ of the PS while the deep lobe projects in the PPS. Approximately 20-30 lymph nodes are found in each parotid gland, serving as first order drainage for the external auditory canal, pinna and the surrounding scalp. Parotid glands become replaced with fat with advancing age.

Space occupying lesions of the PS can displace the PPS from lateral to medial with widening of stylomandibular gap, particularly if located within the deep lobe of the parotid. Both CT and MR can readily image the PS. The normal intraparotid facial nerve is not visible on imaging, and the relationship of a mass lesion to the estimated facial nerve plane, lateral to retromandibular vein, must be assessed. Contrast-enhanced T1 fat-saturated multiplanar MR is optimal in assessment for perineural spread of tumor via CN VII. Contrast-enhanced CT with thin slices is generally recommended if inflammation or infection of the PS is suspected (Fig. 2.35); the gantry can be angled to avoid dental amalgam. Small superficial lobe PS lesions require no imaging with needle aspiration generally being sufficient for diagnosis (Fig. 2.36).

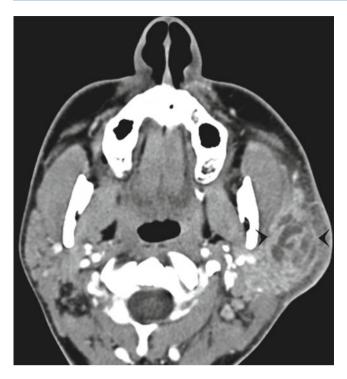


Fig. 2.35 Axial CECT demonstrates a multi-loculated fluid collection (*arrows*) within the enlarged left parotid gland, compatible with parotitis with abscess formation. There are surrounding inflammatory changes, including infiltration of subcutaneous fat and thickening of the overlying skin.

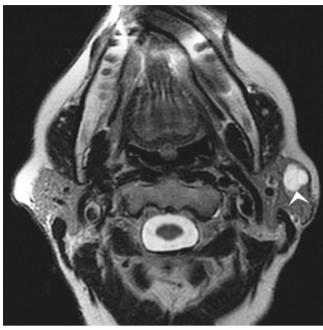


Fig. 2.36 Pleomorphic adenoma of the partotid gland. T2-weighted MRI demonstrates a well circumscribed hyperintense mass (*arrow*) with a bosselated contour, within the left parotid gland, typical of a pleomorphic adenoma

Posterior Cervical Space

The posterior cervical space (PCS) begins at the mastoid tip in the SH neck and extends to the level of the clavicles with most of its volume contained within the IH neck. It is superficial to the PVS and posterolateral to the carotid space. It is located between the SLDCF and DLDCF with all three layers of the carotid sheath contributing to its anteromedial border. The occipital triangle, bounded by the sternocleidomastoid and trapezius muscles and the inferior belly of omohyoid muscle, constitutes the majority of the PCS. The PCS contains primarily fat as well as the eleventh (accessory) cranial nerve and spinal accessory lymph nodes (level V). A segment of the brachial plexus emerging from the anterior and middle scalene gap passes through the PCS, including the dorsal scapular nerve supplying the rhomboid and levator scapulae muscles.

Mass lesions of the PCS displace the carotid space anteromedially, elevate the sternocleidomastoid muscle, and flatten deeper PVS structures. Most lesions of the PCS arise from the spinal accessory nodal chain (Fig. 2.37).

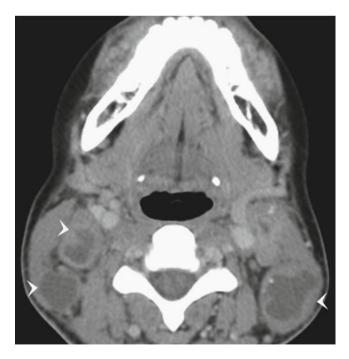


Fig. 2.37 Axial CECT demonstrates bilateral necrotic lymph nodes (*arrows*) within the posterior cervical space

Visceral Space

The visceral space (VS) is a cylindrical space located in the IH neck, extending from the hyoid bone into the superior mediastinum. It is surrounded by the MLDCF and is the largest space of the IH neck. It is medial to the paired anterior cervical spaces, anteromedial to the PPS and anterior to the RPS. The VS contains the thyroid, parathyroids, the trachea, the esophagus, recurrent laryngeal nerves, and portions of the sympathetic trunk. Most authors consider the VS to consist of two subdivisions: an anterior pretracheal (containing the trachea) and posterior retrovisceral, which communicate freely between the levels of thyroid cartilage and the inferior thyroid artery.

The pharyngeal mucosal space is within the VS and contains nasopharyngeal, oropharyngeal, and hypopharyngeal mucosal surfaces, minor salivary glands, lymphatics and pharyngeal constrictor muscles.

A lesion is considered to be arising from the VS if its center is located medial to the PPS with medial to lateral displacement of parapharyngeal fat and disruption of normal mucosal and submucosal architecture. If a malignant neoplasm is suspected, the entire neck should be evaluated for lymphadenopathy for staging purposes and the field of view should be extended to the level of the carina. The VS can be successfully imaged with both contrast-enhanced CT or MR. Knowledge of clinical assessment of the region of concern is crucial as many lesions can be visualized by the referring clinician. Contrast-enhanced fat-saturated MR adds value if there is suspicion of skull base invasion or perineural tumor spread. CT performed with bone algorithm or set to bone window is helpful in delineating the extent of skull base invasion. A common pitfall in interpreting images of the VS is mistaking normal asymmetry such as that resulting from variable amounts of lymphoid tissue or from contained fluid, particularly frequent in the lateral pharyngeal recess, for a space occupying lesion.

A thyroid mass lesion displaces the carotid space laterally and the trachea and the esophagus to the opposite side of the neck (Fig. 2.38).

Ultrasound is the first best single approach to thyroid lesions, which may be used to guide needle aspiration biopsy. Iodine-131 study is performed if the biopsy yields differentiated thyroid carcinoma, which can be followed by MR for locoregional staging, particularly to assess paratracheal lymph nodes. CT is often not recommended as iodinated contrast load may delay iodine-based nuclear medicine therapy. Parathyroid or tracheoesophageal groove mass lesions displace the thyroid lobe anteriorly, carotid space



Fig. 2.38 Thyroid goiter. Axial CECT demonstrates marked enlargement and heterogeneity of the right thyroid lobe (M), with leftward deviation and mild narrowing of the trachea (T). The left thyroid lobe is also mildly enlarged

anterolaterally and longus colli muscle posteriorly and may originate from parathyroid glands, paratracheal nodes or recurrent laryngeal nerves. Parathyroid lesions can occur in both normally located and ectopic parathyroids that can be situated in the mediastinum and can be evaluated with nuclear medicine scintigraphy (sestamibi) or multi-phase CT. A cervical tracheal mass lesion displaces the thyroid laterally and the esophagus posteriorly, while an esophageal lesion displaces both the trachea and thyroid gland anteriorly. In this setting an intraluminal tracheal mass may present with signs and symptoms similar to tracheal stenosis and secondary airway compromise (Fig. 2.39).

Retropharyngeal Space

The retropharyngeal space (RPS) extends from the clivus to the level of T3 vertebra. It is located posterior to the pharyngeal mucosal space in the SH neck and to the VS in the IH neck and is medial to the carotid space. Anteriorly, it is bounded by the MLDCF and posteriorly and laterally by the alar fascia portion of the DLDCF. The median raphe divides the RPS into two halves. Since the superior-most nasopharyngeal portion of the RPS is narrow, the path of the least resistance for spread of lesions, particularly abscesses, is inferiorly towards the mediastinum.

While the SH portion of the RPS contains both lymph nodes and fat, no lymph nodes are present in its IH aspect. Retropharyngeal lesions in the SH neck may originate from



Fig. 2.39 Tracheal stenosis. CT 3D reconstruction of the airway, shown in an AP orientation, demonstrates short segment tracheal stenosis (*arrows*) in this patient with a history of prolonged intubation

lymph nodes and mimic a PPS lesion. MR is more sensitive than CT for detecting retropharyngeal lymphadenopathy. The lateral group of retropharyngeal lymph nodes, known as nodes of Rouviere, is more often visible on imaging than the medial group. An extranodal RPS mass lesion in either the SH or IH neck is posterior to the pharyngeal mucosal space and anterior to prevertebral muscles, which it flattens as it enlarges. Extranodal edema, infection or tumor typically fills the RPS from side to side and is best evaluated with contrastenhanced CT (Fig. 2.40). Non-abscess retropharyngeal fluid exerts a minimal mass effect and demonstrates no wall enhancement.

Perivertebral Space

The perivertebral space (PVS) is located posterior to the RPS, surrounding the spine in both the SH and IH neck and extending from the skull base to the level of the clavicles. The PVS can be separated further by the firm attachment of DLDCF to transverse processes of cervical vertebrae into the anterior prevertebral and posterior paraspinal spaces.

The prevertebral portion of the PVS is posterior to the RPS throughout its extent, posteromedial to the parapharyngeal space and medial to the anterior aspect of the PCS. It contains longus colli, capitis and scalene muscles, vertebral bodies, vertebral artery and vein, brachial plexus roots and the phrenic nerve. The paraspinal portion of the PVS is deep to the PCS and transverse processes of cervical vertebrae. It comprises posterior vertebral elements as well as levator scapulae and paraspinal muscles.

The *danger space* is a potential space between the prevertebral and RPS extending from the skull base to the diaphragm. It is so called because it serves as a pathway for the spread of cranial and cervical infections into the middle and lower mediastinum. Disease processes that invades this space can spread rapidly. The posterior border of the danger space is the prevertebral fascia and the anterior border is the alar fascia. The inferior aspect of the RPS communicates with the danger space at the level of T3. The danger space is indistinguishable from the RPS on imaging.

Most PVS lesions originate in vertebral bodies. The anterior aspect of the DLDCF blocks pharyngeal pathology from accessing the PVS and redirects PVS tumor or infection into the epidural space via neural foramina. Cervical spine MR is the best tool to assess for epidural disease.

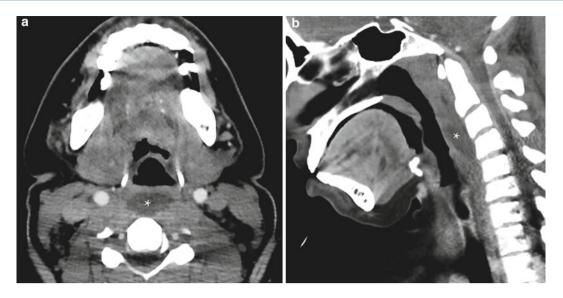


Fig. 2.40 Retropharyngeal abscess. Axial (a) and sagittal (b) CECT demonstrates a discrete fluid collection (*asterisk*) occupying the retropharyngeal space in this young patient with fever and odynophagia, compatible with an abscess

Appendix A

Normal CT anatomy slices cephalad to caudad (Figs. 2.41, 2.42). See separate abbreviation list.

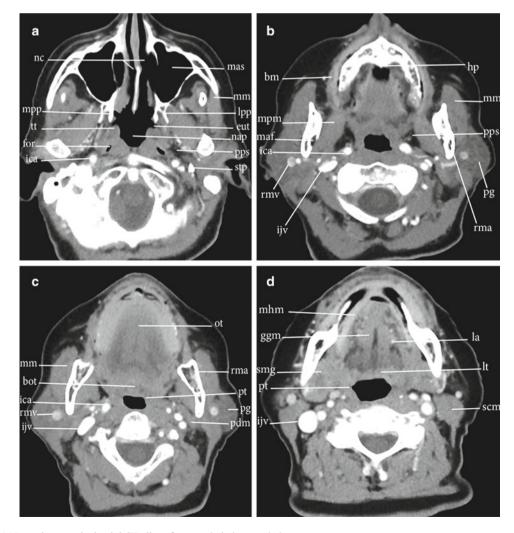


Fig. 2.41 (a-h) Normal anatomical axial CT slices from cephalad to caudad

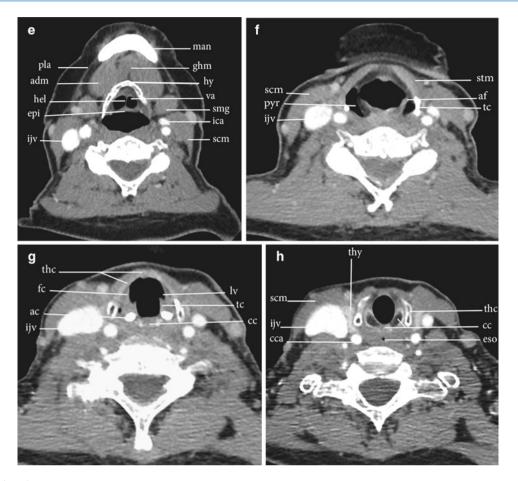


Fig. 2.41 (continued)

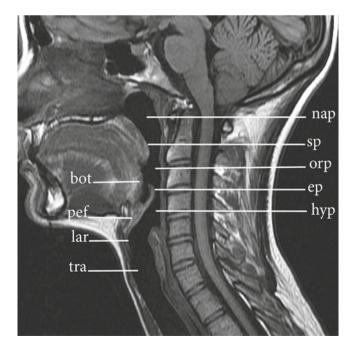


Fig. 2.42 Normal anatomical sag MRI

Appendix B

Adm	Anterior belly of digastric muscle
Ac	Arytenoid cartilage
Af	Aryepiglottic fold
Bm	Buccinator muscle
Bot	Base of tongue
Cc	Cricoid cartilage
Cca	Common carotid artery
Ep	Epiglottis
Eso	Esophagus
Eut	Eustacian tube (orifice)
For	Fossa of Rosenmuller
Fc	False cord
Ggm	Genioglossus muscle
Ghm	Geniohyoid muscle
Hel	Hyoepiglottic ligament
Нр	Hard palate
Ну	Hyoid bone
Нур	Hypopharynx
Ica	Internal carotid artery
Ijv	Internal jugular vein
Lar	Larynx
Lpm	Lateral pterygoid muscle
Lpp	Lateral pterygoid plate
Lt	Lingual tonsils
Lv	Laryngeal ventricle
Man	Mandible
Maf	Mandibular foramen
Mas	Maxillary sinus
Max	Maxilla
Mhm	Mylohyoid muscle
Mm	Masseter muscle
Mpm	Medial pterygoid muscle
Мрр	Medial pterygoid plate
Nap	Nasopharynx
Nc	Nasal cavity
Orp	Oropharynx
Ot	Oral tongue
Pdm	Posterior belly of digastric muscle
Pef	Pre-epiglottic fat
Pg	Parotid gland
Pla	Platysma muscle
Pps	Parapharyngeal space
Pt	Palatine tonsils
Pyr	Pyriform sinus
Rma	Ramus of mandible

D	
Rmv	Retromandibular vein
Scm	Sternocleiomastoid muscle
Smg	Submandibular gland
Stm	Strap muscle
Sp	Soft palate
Stp	Styloid process
The	Thyroid cartilage
Thy	Thyroid
Тс	True cord
Tra	Trachea
Tt	Torus tubarius
Va	Vallecula
Neck spaces (I	MR images)
Bs	Buccal space (orange)
Ms	Masticator space (pink)
Pcs	Posterior cervical space (purple/magenta)
Pps	Parapharyngeal space (green)
Pvs	Paravertebral space (dark blue)
Ps	Parotid space (light blue)
Rps	Retropharyngeal space (yellow)
Vs	Visceral space (red)

References

- Hurley DB, Javer AR, Kuhn FA, Citardi MJ. The endoscopic management of chronic frontal sinusitis associated with frontal sinus posterior table erosion. Am J Rhinol. 2000;14:113–20.
- Laine FJ, Smoker WR. The ostiomeatal unit and endoscopic surgery: anatomy, variations, and imaging findings in inflammatory diseases. AJR Am J Roentgenol. 1992;159:849–57.
- Jalisi S. Management of the clinically negative neck in early squamous cell carcinoma of the oral cavity. Otolaryngol Clin North Am. 2005;38:37–46.
- Lin DT, Cohen SM, Coppit GL, Burkey BB. Squamous cell carcinoma of the oropharynx and hypopharynx. Otolaryngol Clin North Am. 2005;38:59–74.
- 5. Stockwell M, Lozanoff S, Lang SA, Nyssen J. Superior laryngeal nerve block: an anatomical study. Clin Anat. 1995;8:89–95.
- Bielamowicz SA, Storper IS, Jabour BA, Lufkin RB, Hanafee WN. Spaces and triangles of the head and neck. Head Neck. 1994;16: 383–8.
- Som PM and Curtin HD. Head and Neck Imaging: Fifth Edition. St. Louis, MO Mosby; 2011.
- Andrieu G, Amrouni H, Robin E, et al. Analgesic efficacy of bilateral superficial cervical plexus block administered before thyroid surgery under general anaesthesia. Br J Anaesth. 2007;99:561–6.
- de Tran QH, Dugani S, Finlayson RJ. A randomized comparison between ultrasound-guided and landmark-based superficial cervical plexus block. Reg Anesth Pain Med. 2010;35:539–43.
- Som PM and Curtin HD. Head and Neck Imaging: Fifth Edition. St. Louis, MO Mosby; 2011.
- Suresh S, Voronov P. Head and neck blocks in children: an anatomical and procedural review. Paediatr Anaesth. 2006;16:910–8.
- Perkin GD. Trigeminal Neuralgia. Curr Treat Options Neurol. 1999;1:458–65.

- 13. Green MW, Selman JE. Review article: the medical management of trigeminal neuralgia. Headache. 1991;31:588–92.
- 14. Zakrzewska JM. Trigeminal neuralgia. Prim Dent Care. 1997;4: 17–9.
- Salam GA. Regional anesthesia for office procedures: part I. Head and neck surgeries. Am Fam Physician. 2004;69:585–90.
- Wormald PJ, Athanasiadis T, Rees G, Robinson S. An evaluation of effect of pterygopalatine fossa injection with local anesthetic and adrenalin in the control of nasal bleeding during endoscopic sinus surgery. Am J Rhinol. 2005;19:288–92.
- Molliex S, Navez M, Baylot D, Prades JM, Elkhoury Z, Auboyer C. Regional anaesthesia for outpatient nasal surgery. Br J Anaesth. 1996;76:151–3.

- Chrcanovic BR, Custodio AL. Anatomical variation in the position of the greater palatine foramen. J Oral Sci. 2010;52:109–13.
- Douglas R, Wormald PJ. Pterygopalatine fossa infiltration through the greater palatine foramen: where to bend the needle. Laryngoscope. 2006;116:1255–7.
- Khoury JN, Mihailidis S, Ghabriel M, Townsend G. Applied anatomy of the pterygomandibular space: improving the success of inferior alveolar nerve blocks. Aust Dent J. 2011;56:112–21.
- Whitworth JM, Kanaa MD, Corbett IP, Meechan JG. Influence of injection speed on the effectiveness of incisive/mental nerve block: a randomized, controlled, double-blind study in adult volunteers. J Endod. 2007;33:1149–54.

Basic Pharmacology of Anesthetic Agents

Wagdy Sidrak, Adam I. Levine, and Samuel DeMaria, Jr.

Introduction

Anesthesiologists prescribe, prepare, and administer medications during the perioperative period to facilitate patient safety while optimizing surgical conditions. The vast majority of these medications, unfortunately, have significant and potentially life-threatening side effects. Comprehension of these concepts can facilitate and impact operating conditions for the otolaryngologist, improve operating room efficiency, and help to prevent surgical complications. Therefore, it is of paramount importance for the otolaryngologist to have a working knowledge of the agents administered by anesthesiologists while they operate.

Basic Pharmacologic Principles

Pharmacodynamics

Pharmacodynamics describes the mechanism of action of a drug and its effects on the body at the receptor level. Most drugs exert their action by binding to a receptor and acting as agonists, antagonists, or both. The *potency* of a drug is the relative amount of drug needed to produce a given effect. It is proportional to the affinity of a drug to its receptors. *Efficacy* is the maximal or ceiling effect of a drug, which is reached after saturation of receptor binding sites. An understanding

and application of basic pharmacodynamic principles allows for optimal anesthetic management of otolaryngology procedures. For instance, knowing that the cough reflex is inhibited by activation of opioid mu receptors facilitates its suppression and thus minimizes "bucking" and improves patient tolerance of the presence of an endotracheal tube. Alternatively, it can be suppressed at the axonal level with blockade of the internal laryngeal branch of the superior laryngeal nerve by local anesthetic injection.

Pharmacokinetics

Pharmacokinetics describes the movement of a drug throughout the body and involves absorption, distribution, metabolism, and elimination. Intravenous administration provides the most predictable route of drug absorption and is therefore used in most circumstances. Drugs given orally may undergo first-pass metabolism in the liver (via the portal circulation) and thus have variable bioavailability. The volume of distribution relates to the apparent volume in the body into which a drug is contained. Drugs that are more highly bound in peripheral tissues have a larger volume of distribution than drugs that are more concentrated in the blood. Highly polar drugs, such as neuromuscular blocking agents, are limited to the extracellular fluid compartment, whereas those that are more lipid soluble, such as synthetic opioids and propofol, can have very large volumes of distribution. Drugs that circulate in the plasma can also have variable ionization and a degree of protein binding, both of which further limit their distribution in the body.

Intravenous Anesthetic Drugs

Anesthetic agents often follow a two- or three-compartment pharmacokinetic profile. After administration of a bolus intravenous dose, most of the drug is distributed to highly perfused organs (i.e., brain, lungs, heart, kidneys, liver)

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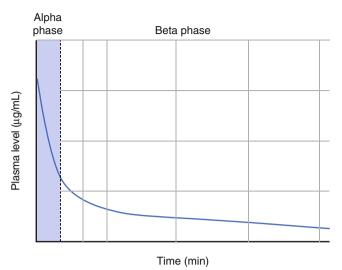


Fig. 3.1 Plasma concentration curve of a typical anesthetic agent

first, and is rapidly taken up by the lipophilic tissues in the central nervous system (CNS) where most anesthetics exert their effects. At this point, the plasma concentration of drug is greatly decreased by rapid redistribution (the α -phase or half-life). The α -half-life is responsible for the rapid termination of many anesthetic effects. Over the next minutes to hours, the drug that was taken up by the CNS slowly re-equilibrates with the plasma and the remaining drug in the blood is redistributed to the less perfused peripheral tissues (muscle, skin, fat, bone), where it is slowly absorbed. This slow absorption as well as subsequent metabolism is the β elimination phase (Fig. 3.1). Large bolus doses or multiple frequent doses will result in a prolonged anesthetic effect since termination will largely be dependent on β elimination. This pharmacokinetic model explains the rapid onset and offset of many commonly used anesthetic agents.

During a continuous intravenous infusion, a loading bolus dose of a drug is administered to rapidly achieve a therapeutic concentration at the effector sites (receptor site). This is then followed by a continuous infusion to maintain the plasma concentration and therefore the effect, with the goal being to sustain a stable therapeutic plasma level. This avoids the use of intermittent boluses, which would result in significant fluctuations in plasma drug levels. After discontinuation of the drug infusion, saturated peripheral compartments act as depots for the drug to slowly diffuse back into circulation. Using computer-modeled data, this phenomenon can be described in terms of a calculated context-sensitive half-time (i.e., the time required for the concentration of a drug to decrease by 50% after a prolonged infusion). Drugs with a relatively short context-sensitive half time, or those that do not increase over long administrative times such as

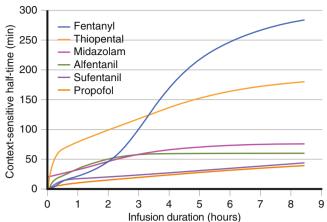


Fig. 3.2 Context-sensitive half-times of intravenous anesthetic agents as a function of duration of infusion. Reprinted with permission from ^[1]

remifentanil or propofol may be more useful in ambulatory cases or cases in which extubation is planned (Fig. 3.2). Drugs with longer context-sensitive half-times or those affected by administration duration (fentanyl) require more careful timing and weaning of the anesthetic (which can be challenging in more unpredictable operations and with variable patient pharmacokinetics).

Drug dosing is primarily based on patient body weight. Drugs which have higher lipid solubility (e.g., benzodiazepines, barbiturates) are dosed based on total body weight, whereas hydrophilic drugs, which have a tendency to remain in the intravascular space, are dosed based on ideal body weight. Many intravenous anesthetic agents are also titrated to a certain effect, such as vital signs, neuromuscular monitor data, or bispectral index ("BIS" score), which can simplify dosing in patients with nonideal pharmacokinetic profiles.

Clearance describes the volume of blood from which a drug is eliminated from the body per unit time. Classically, most drugs obey first-order kinetics, wherein a constant fraction of a drug is eliminated per unit of time, independent of the plasma concentration. After four half lives, approximately 95% of a drug is eliminated from the body and the drug is considered to be cleared entirely. The elimination half-life is only clinically applicable to drugs that obey single-compartment pharmacokinetics, yet most anesthetics are lipophilic drugs which follow multi-compartment pharmacokinetics. The kidneys and liver are the primary organs of drug elimination. The kidneys eliminate water-soluble compounds, many of which are byproducts of oxidationreduction (Phase I, cytochrome reactions) and/or conjugation (Phase II) reactions in the liver. The hepatic extraction ratio varies between drugs and also determines their rate of biotransformation in the liver.

Opioids

Opioids are the most commonly used analgesic compounds in the perioperative care of surgical patients. As a component of anesthetic care, their use attenuates the hemodynamic and hormonal stress responses to noxious stimuli. Their effects arise from mimicry of endogenous endorphins, enkephalins, and dynormins at opiate receptors located throughout the body. Analgesic effects result from binding of receptors in the central and peripheral nervous systems. Opioids can be classified as naturally occurring, semisynthetic, and synthetic.

Activation of mu, delta, and kappa receptors located in the brainstem and spinal cord (dorsal horn) results in analgesia by decreasing neurotransmission (through an inhibitory G protein), thereby suppressing ascending nociceptive signals. In addition to analgesia, mu receptor activation results in many other effects, including: euphoria, ventilatory depression, constipation, miosis, bradycardia, pruritus, urinary retention, cough suppression, nausea, muscle rigidity, and hypothermia.

Opioids are effective in providing hemodynamic stability during the perioperative period. In normovolemic patients without critical illness, there is minimal effect on cardiac contractility and blood pressure with usual doses, although dose-dependent bradycardia does occur. For this reason, high-dose opioids have been used as induction agents in patients with heart failure and coronary artery disease and have a long record of safety and efficacy. They are also useful in blunting detrimental hemodynamic responses to laryngoscopy, surgery, and postoperative pain.

The most feared consequence of opioids is respiratory depression. Opioids result in a decrease in respiratory rate as well as a decrease in respiratory responses to hypercarbia and hypoxia. They also increase the $PaCO_2$ and apneic threshold (i.e., the highest $PaCO_2$ at which one remains apneic). Pain, on the other hand, is a stimulus to breath and often leads to hyperventilation, tachycardia, and hypertension. Balancing this can be a challenge. Patients receiving relatively high doses of opioids are often arousable and will breathe if instructed. Due to the risk of increased $PaCO_2$ leading to cerebral vasodilation, opioids should be used cautiously in patients with traumatic brain injury as they may cause intracranial hypertension as well as sedation, making neurological assessment more difficult.

Posttranslational modifications as well as different CNS distributions can lead to idiosyncratic responses to specific opioid agonists ^[2]. Patients receiving chronic opioid therapy develop tolerance to analgesia and respiratory depression through activation of adenylyl cyclase. Significant tolerance to miosis and constipation does not occur. If possible, patients taking narcotics chronically should have them continued throughout the perioperative period ^[3]. Oral medications can be taken on the morning of surgery and transdermal fentanyl patches should be left on the patient during nonmajor surgery,

or they may be replaced with similar doses intravenously. A wide range of doses for opiates exist and they should be tailored to patient responsiveness, generally starting low and titrating upwards as needed.

Naturally Occurring Opioids

Morphine

Morphine is the prototypical opioid agonist. Following intravenous administration, onset of action is 5-10 min, peak analgesic effect occurs in 15-30 min, and elimination halflife is between 1.7 and 3.3 h. The duration of action is 3–4 h. It is therefore useful in postoperative patients, where an extended duration of analgesia is required. Hepatic phase II conjugation results in active metabolites, morphine-3glucuronide and morphine-6-glucuronide, which are then eliminated in the urine. The accumulation of these metabolites in patients with renal failure can result in prolonged effects. There is a dose-dependent histamine release with morphine, which can lead to a decrease in blood pressure or bronchospasm with the administration of high doses. Therefore, morphine should be avoided in patients with asthma or other reactive airways disease, coronary artery disease, aortic stenosis or any patient that would not tolerate tachycardia or hypotension.

Codeine

Codeine is another naturally occurring opioid with strong cough-suppressant properties. Its analgesic effects result from biotransformation in the liver to morphine (10%). Hydrocodone is a codeine derivative that is combined with acetaminophen in the preparation Vicodin[®] (Hydrocodone 7.5 mg/acetaminophen 750 mg).

Semi-synthetic Opioids

Semisynthetic opioids result from structural modification to the morphine compound and include heroin, hydromorphone, hydrocodone, oxycodone, oxymorphone, and the partial-agonist buprenorphine.

Hydromorphone

Hydromorphone (Dilaudid[®]) is 5-10 times as potent as morphine and is a useful alternative in patients requiring higher opioid doses (e.g., chronic pain, poor pain control). Its peak effect is 20-30 min after intravenous administration and has

a duration of action of 2-3 h. Usual dosing for postoperative pain is 1.5 mg every 2-4 h (equi-analgesic effect to 10 mg morphine). In the immediate postoperative period, higher and more frequent doses may be required.

Oxycodone

Oxycodone is orally administered with similar potency to morphine and is useful in postoperative analgesia (5–30 mg every 4 h). Its preparations include Percocet (oxycodone 5 mg with 325 mg acetaminophen) and timed release oxycodone (Oxycontin[®]).

Synthetic Opioids

Completely synthetic opioids, with the exception of meperidine, are more potent opioid agonists and are commonly used intraoperative analgesics.

Fentanyl

Fentanyl is approximately 100 times more potent then morphine. Its high lipid solubility promotes rapid passage through the blood-brain barrier, giving it an onset time within 30 s and a peak effect in 3-5 min. A loading dose (2-8 mcg/kg) administered 3 min before laryngoscopy is useful in blunting the hemodynamic response. It undergoes significant firstpass metabolism in the lung and quickly redistributes to other tissues (such as muscle and fat), leading to a short duration of action (30-60 min). It eventually slowly mobilizes back into the circulation and is eliminated by the liver, with an elimination half-time of 3-4 h. Fentanyl is useful in providing intraoperative analgesia in either intermittent boluses every 30 min or as an infusion (0.5-5.0 mcg/kg/h), although its context-sensitive half-time rises sharply after approximately 90 min. A transdermal fentanyl delivery system is also available for the treatment of chronic pain and a transmucosal lollipop is available for analgesia and sedation in children.

Sufentanil

Sufentanil is a fentanyl analog and is also commonly used intraoperatively. It is 5–10 times as potent as fentanyl and has a similar pharmacodynamic and pharmacokinetic profile. It has a slightly shorter elimination half-life due to a smaller volume of distribution. Loading doses range between 0.25 and 2 mcg/kg and are followed by a maintenance dose of 0.3– 1.5 mcg/kg/h, either as an infusion or intermittent boluses.

Alfentanil

Due to its small volume of distribution and highly nonionized fraction (90%), alfentanil is a synthetic opioid with a rapid onset and relatively rapid offset. Alfentanil is 1/5th as potent as fentanyl, but is particularly useful in situations where fast and intense, yet brief analgesia is required. A 15 mcg/kg IV bolus is effective in blunting the hemodynamic consequences of intubation. An infusion of 25–150 mcg/kg/h can be used to maintain intraoperative analgesia, although its context-sensitive half-time may be prolonged. Its effect is terminated by redistribution and its elimination half-life is 1.5 h.

Remifentanil

Remifentanil is a popular, yet expensive fentanyl analog with a reported potency that is equal to fentanyl. It is unique in that its context-sensitive half-time is constant at 3-4 min, independent of infusion duration. It is predictably metabolized by nonspecific plasma and tissue esterases and its metabolism is independent of the liver or kidney, patient comorbidities, or genetics. A loading dose of 0.5-5 mcg/kg followed by an infusion of 0.05-2 mcg/kg/min can be useful intraoperatively in procedures which are stimulating but result in a relatively low amount of postoperative pain, such as sinus endoscopy or blunting the hemodynamic response to laryngoscopy and intubation. When needed, a longer-acting opioid, regional anesthesia, or local anesthesia infiltration can be administered prior to awakening to provide postoperative pain relief. Taking advantage of its antitussive properties, a low-dose remifentanil infusion can be continued for a more smooth emergence and extubation, without significantly delaying emergence ^[4, 5]. By reliably inhibiting the sympathetic response to surgery, it is also often used in cases where controlled hypotension is employed to improve operating conditions (Chap. 13)

Meperidine

Meperidine was the first synthetic opioid created and has a potency that is one-tenth that of morphine. It is used for post-operative analgesia (50–100 mg IV, IM) and small doses (12–25 mg) are effective in suppressing postoperative shivering via binding of kappa opioid receptors. Its metabolite, norme-peridine is neurotoxic, can induce seizures, and requires the liver and kidneys for its elimination. The use of meperidine in patients taking monoamine oxidase inhibitors may lead to the development of the potentially lethal serotonin syndrome or serotonin toxicity (Libby-Zion Case). Like morphine, meperidine is also associated with histamine release which may be mitigated by slow injection. Unique among the opioid

agonists, meperidine has a direct myocardial depression and has atropine-like activity that may cause tachycardia. Due to its side effect profile, it is typically not a first-line agent.

Methadone

Methadone is a long-acting synthetic opioid with similar potency to morphine that is indicated for the treatment of opioid withdrawal and management of chronic pain. It also has antagonist activity at glutaminergic *N*-methyl d-aspartate (NMDA) receptors. It is equipotent to morphine but carries a plasma half-life of 13–100 h, and is usually dosed once to twice daily. It can prolong the QTc interval.

Naloxone

Naloxone is an antagonist of all opioid receptors and is used to reverse cases of narcotic overdose. It is typically titrated in 40–80 mcg increments every 1–2 min until normalization in respiratory rate occurs. Abrupt reversal with large doses of naloxone may create a rebound surge in catecholamines, leading to hypertension, tachyarrythmias, seizures, and pulmonary edema. Its peak effect occurs in 1–2 min and duration of action is approximately 2 h. In the case of large overdose or use of long-acting opioids, an infusion may need to be started (3–10 mcg/h) as the opioid's effect may last beyond naloxone's antagonism.

Induction Agents and Hypnotics

Propofol

Propofol is y-aminobutyric acid (GABA) receptor agonist which produces a deep state of unconsciousness within 30 s of administration of an induction dose (1.5-2.5 mg/kg). It causes a dose-dependent respiratory depression, with apnea occurring in approximately 70-96% of patients after an induction dose, and provides excellent intubating conditions. Its relatively short context-sensitive half-time makes it a good choice for sedation in the operating room and intensive care unit. Propofol's effects wane after redistribution approximately 5-10 min after a bolus dose, and it is ultimately eliminated by the liver (and presumably lung) with a half-life of 4–7 h. Recovery is typically faster with propofol induction compared with other available agents. Use of propofol is associated with a decreased incidence of postoperative nausea and vomiting ^[6] and is associated with a faster discharge from the PACU. Use of a total intravenous anesthesia (TIVA) technique with propofol and remifentanil may decrease intraoperative and postoperative

bleeding, as well as reduce the incidence of coughing on emergence and extubation in sinus endoscopy cases (Chap. 13) ^[7-10].

Propofol causes arterial and venous dilatation resulting in hypotension and, by impairing the baroreceptor reflex tachycardia, decreases cardiac output. Its use should be judicious in hypovolemic and elderly patients, who may have exaggerated responses to these side effects. It decreases cerebral metabolic rate for oxygen (CMRO₂), thus decreasing cerebral blood flow and intracranial pressure. Pain on injection, which is worse when smaller veins are used, can be mitigated by addition of lidocaine to the solution or given prior to propofol injection ^[11].

Barbiturates

Barbiturates are GABA receptor agonists that cause a dosedependent depression of the reticular activating system. They are highly useful as bolus injections for induction of anesthesia (thiopental 3-5 mg/kg or methohexital 1.5 mg/kg), but their use in infusions is limited as a result of saturation of the peripheral compartment leading to prolonged sedation. Thiopental, which will likely no longer be available in the United States, and methohexital were the two most commonly used agents in this class. Barbiturates are useful in rapid-sequence induction as patients are typically unconscious within 30 s. Awakening after a bolus is typically after 5–10 min and is due to redistribution (thiopental's β half-life is 11.7 h), drowsiness may last for several hours due to prolonged metabolism. Infusions and large boluses will delay emergence. Lingering pyschomotor impairment is longer with thiopental than methohexital.

Barbiturates cause depression in both tidal volume and respiratory rate in a dose-dependent manner. Induction doses often cause apnea. Protective airway reflexes are not as depressed as with propofol, so patients are more likely to "buck" or cough during airway instrumentation in the absence of a muscle relaxant. Barbiturates cause venodilation and are the most negative inotropes of the intravenous agents, leading to hypotension and a decrease in cardiac output. These effects are exaggerated in hypovolemic patients and patients with impaired cardiac function, but hypotension is not as severe as with propofol—secondary to less baroreceptor depression with resultant compensatory tachycardia. Barbiturates decrease CMRO₂ and decrease mean arterial pressure (MAP), leading to decreases in cerebral blood flow and intracranial pressure.

Thiopental is highly alkaline in solution and intra-arterial injection leads to severe vasospasm that may progress to tissue ischemia and permanent damage. It will also precipitate when mixed with acidic drugs such as neuromuscular blocking agents. Its hepatic metabolism and enzyme induction contraindicate its use in acute intermittent porphyria.

Benzodiazepines

Benzodiazepines have a number of effects on the central nervous system including sedation, anxiolysis, muscle relaxation, anterograde amnesia, and anticonvulsant effects. In high enough concentrations, unconsciousness develops and these agents can be used for induction of general anesthesia. They are GABA agonists and are primarily used as premedicants due to their anxiolytic and amnestic effects.

The most commonly used benzodiazepines are midazolam, diazepam, and lorazepam. Midazolam is the shortestacting and most frequently used. It is highly lipid soluble after injection and has an onset time of less than 1 min. As a premedication, (0.5-2 mg IV), it has a strong calming effect on patients with minimal risk for respiratory depression. Continued small boluses or an infusion (0.25-1 mcg/kg/min) can be used for intraoperative sedation in less stimulating procedures. Induction doses (0.1-0.3 mg/kg) have an increased frequency of respiratory depression but hemodynamic effects are minimal. Duration of unconsciousness after an induction dose is 15-30 min, but arousal is associated with a greater "hangover" (i.e., residual sedation) than propofol or thiopental, making it less suitable for outpatient surgery. Because of its relatively short context-sensitive half-time, midazolam is the benzodiazepine that is most suitable for infusions and is often used in intensive care units for this purpose. An active metabolite, 1-hydroxymidazolam can accumulate in prolonged infusions in patients with renal failure.

Diazepam and lorazepam are intermediate and long-acting benzodiazepines, respectively. Unlike midazolam, they are available in tablet formulations. When given intravenously, both are associated with pain on injection. Diazepam has several active metabolites which may accumulate with repeated doses or infusions.

Elderly patients and patients with preexisting cardiac or pulmonary dysfunction are more sensitive to the effects of benzodiazepines. Respiratory depression is potentiated by administration of other respiratory depressants, such as opioids. Both midazolam and diazepam have active metabolites that may accumulate in patients with renal failure. Chronic use of all benzodiazepines is associated with tolerance to anxiolytic effects, so preoperative doses may need to be increased accordingly.

An advantage of benzodiazepines is the availability of a specific reversal agent, flumazenil. Flumazenil competes with benzodiazepines for binding at the GABA receptor. Doses of 0.2 mg are given every 1 min until adequate reversal is achieved, up to a maximum dose of 1 mg. Patients on chronic benzodiazepines, tricyclic antidepressants, or patients with a history of seizures are at risk for withdrawal seizures when given flumazenil, and these risks should be weighed against benefits in this specific instance. Flumazenil has a half-life of only 1 h, so repeated doses may be necessary depending on the benzodiazepine administered.

Etomidate

Etomidate is an induction agent with minimal hemodynamic effects that is frequently used in patients with congestive heart failure, coronary artery disease, or hypovolemia. Induction with etomidate (0.2–0.6 mg/kg IV) is associated with minimal changes in blood pressure, heart rate, or cardiac output. Like several other anesthetics, it acts by a GABA-linked mechanism. Induction with etomidate has a similar onset and duration to propofol and barbiturates, but is associated with less respiratory depression. Indeed, patients may not become apneic after an induction dose. Etomidate also decreases CMRO₂, leading to a decrease in cerebral blood flow and intracranial pressure.

A worrisome side effect of etomidate is its inhibition of the adrenocortical stress response ^[12]. It inhibits 11 β -hydroxylase in the adrenal cortex, leading to suppression of cortisol production for 4–8 h after induction and longer after infusions or repeated dosing. Although there is little evidence correlating this with an increase in mortality, this fact has limited its use as an infusion for sedation or maintenance of anesthesia. Other side effects associated with etomidate are pain on injection, myoclonus, and an increased incidence of postoperative nausea and vomiting.

Ketamine

Ketamine is an NMDA receptor antagonist with a similar chemical structure to phencyclidine (PCP). It provides sedation, as well as deep analgesia and amnesia (making it the most "complete" intravenous anesthetic), while maintaining spontaneous respiration and upper airway tone, but not protective reflexes (patients are still vulnerable to aspiration). Patients receiving ketamine may appear conscious, in a catapleptic state ("dissociative anesthesia"), due to an inhibition of thalamocortical pathways.

Ketamine differs from other general anesthetics in its cardiovascular effects since its administration causes a release of endogenous catecholamines and central sympathetic stimulation. It is the only general anesthetic agent which increases blood pressure and heart rate, making it useful in the hypovolemic patient (whether relative or absolute). Its maximal cardiovascular stimulation occurs approximately 4–5 min after administration. It does, however, have negative inotropic effects and can decrease blood pressure in the catecholamine-depleted patient. It should be used judiciously in patients with coronary artery disease, as its hemodynamic effects lead to increased myocardial oxygen demand.

Ketamine has many advantageous respiratory effects. At low doses, patients maintain spontaneous respiration with minimal respiratory depression. Apnea may occur at induction doses (1–2 mg/kg intraveneously or 4–8 mg/kg intramuscularly) or if an opioid is simultaneously administered. Ketamine stimulates salivation, which may increase the risk of aspiration and laryngospasm; this can be blunted by co-administration of an anticholinergic such as glycopyrrolate. It is also a potent bronchodilator and is the induction agent of choice in patients in active bronchospasm. Ketamine does increase pulmonary vascular resistance and should be avoided in patients with pulmonary hypertension or right-sided heart failure.

In the central nervous system, ketamine is the only intravenous anesthetic that increases cerebral oxygen consumption, blood flow and may increase ICP. Ketamine must be avoided in patients with traumatic brain injury and increased intracranial pressure. A major unwanted side effect of ketamine is the occurrence of vivid nightmares and hallucinations, which may persist into the postoperative period (up to 24 h after use). Similarly, the drug can increase the incidence of emergence delirium. Administration of a benzodiazepine prior to ketamine can reduce these effects. Myoclonus and purposeless movements of the extremities are also occasionally observed with ketamine.

Ketamine has a relatively fast onset time (30-60 s), with emergence occurring after approximately 30 min. It can be applied adjunctively with other anesthetics in low doses (0.5 mg/kg bolus followed by 1-2 mg/kg/h) as it carries an opioid-sparing effect in the perioperative period and is highly useful in opioid-dependent patients.

Dexmedetomidine

Dexmedetomidine is a highly selective $\alpha 2$ adrenergic receptor agonist that is used as a sedative in the operating room and intensive care unit. This $\alpha 2$ agonism occurs in the locus ceruleus and spinal cord to produce sedation, analgesia, and anxiolysis. It provides reliable hypnosis, yet patients remain easily arousable and capable of following commands. Dexmedetomidine appears to provide unconsciousness that has more similar characteristics to natural sleep than other anesthetics, and its mild respiratory depressant effects seem to mimic those that occur during normal sleep (small decreases in tidal volume). The respiratory response to hypercapnia is maintained, although the risk of upper airway obstruction does increase. Since dexmedetomidine is a sympatholytic agent, typical cardiovascular effects include bradycardia and a reduction of MAP. Hypotension is more marked when loading doses are administered. Reducing or eliminating the loading dose (0.5-1 mcg/kg over 15 min) appears to provide satisfactory hemodynamics. The drug may decrease the incidence of postoperative myocardial infarction, similar to clonidine, another $\alpha 2$ agonist, and it is useful in attenuating increased heart rate and plasma catecholamines during emergence from anesthesia^[13].

Due to its relatively mild respiratory depressant effect when used alone, dexmedetomidine has been suggested as a

useful sedative for fiberoptic intubation. The ability to easily arouse patients makes it well suited for monitored anesthesia care as well. Its use is approved for infusions (0.2–0.7 mcg/ kg/h) lasting less than 24 h, although it appears to be safe for much longer than this without any rebound hypertension ^[14]. It is also useful as a transitional sedative from the operating room to intensive care unit when patients are expected to be extubated within 24 h.

Neuromuscular Blocking Agents

Neuromuscular blocking agents are frequently used since many otolaryngologic procedures involve operating on or near vital structures, where patient movement could be catastrophic. Similarly, even slight patient movement in small operating fields or under surgical microscopes can greatly increase the technical difficulty of procedures. From the perspective of the anesthesiologist, muscle relaxants can provide marked facilitation for intubation and subsequent mechanical ventilation by inhibiting airway reflexes. On the contrary, many operations on the head and neck (tympanomastoidectomy: Chap. 11; parotidectomy: Chap. 14; and radical neck dissections: Chap. 15) require peripheral nerve monitoring and the use of neuromuscular blocking (NMB) agents is contraindicated. The use of NMB agents is so commonplace in the practice of anesthesiology that it is imperative that the use or avoidance of NMB be discussed amongst the anesthesiologists and otolaryngologist prior to the start of the procedure.

It is important to note that muscle relaxants should only be administered as part of a balanced anesthetic technique in the presence of an adequate depth of unconsciousness and analgesia. Patient movement may be an indicator of insufficient depth of anesthesia and muscle relaxants may mask this. They should be used with caution in hemodynamically unstable patients requiring a "light" degree of anesthesia as this could present an increased risk of anesthesia awareness.

Neuromuscular inhibitors act by blocking synaptic transmission at nicotinic acetylcholine receptors at the neuromuscular junction. There are two classes of muscle relaxants: depolarizing and non-depolarizing neuromuscular blockers.

Depolarizing Neuromuscular Blocking Agents

Succinylcholine

Succinylcholine is the only depolarizing agent used in modern clinical practice. Its chemical structure is composed of two acetylcholine molecules linked together by acetyl methyl groups. It acts by binding to postsynaptic nicotinic

 Table 3.1
 Adverse effects of succinylcholine

Adverse effects	Notes
Bradyarrythmias	Caused by inhibition of muscar- inic acetylcholine receptors. More common in pediatric patients due to higher vagal tone or with repeated dosing
Myalgias	Caused by fasiculations. Self-limited. Incidence decreased with non-depolarizing muscle relaxants and lidocaine
Hyperkalemia	Risk of malignant tachyarryth- mias. Risk is increased in patients with burns (>24 h to 1 year after burn), traumas, denervation injuries, sepsis, prolonged immobilization (>24 h), and myopathies
Increased intracranial pressure	May be secondary to fasicula- tions. Inhibited by pre-administra- tion of non-depolarizing muscle relaxant
Increased intraocular pressure	Likely due to contraction of extraocular muscles
Malignant hyperthermia	Succinylcholine (as well as halogenated inhaled anesthetics) has been identified as triggers for malignant hyperthermia
Prolonged paralysis	In patients with genetically atypical pseudocholinesterase, impaired function of pseudocho- linesterase (liver failure, cholinesterase inhibitor drugs), or reduced synthesis of pseudocho- linesterase (liver failure)

acetylcholine receptors at the neuromuscular junction, causing muscle fasciculation. Its muscle relaxant effect is likely attributed to continued occupation of the acetylcholine receptor, preventing subsequent depolarization of the motor end plate. Succinvlcholine is the fastest-acting muscle relaxant (onset time 30-60 s) and has the shortest duration of action (elimination half-life of approximately 3 min, duration of effect 5–10 min). It is therefore useful in emergent and rapid-sequence intubations (0.5-2.0 mg/kg IV) and its short duration of action allows the potential for patients to recover respiratory muscle strength in the case of a difficult or impossible intubation or ventilation. It can be used in cases requiring intubation where paralysis may be undesired during the surgical procedure, such as nerve dissection or when nerve monitoring is used. Succinylcholine has a fairly diverse range of adverse effects (Table 3.1).

Termination of action of succinylcholine is by diffusion away from the neuromuscular junction and subsequent prompt degradation by plasma pseudocholinesterases (butyrylcholinesterase). Patients with decreased pseudocholinesterase activity can have prolonged paralysis as a result of succinylcholine. Most commonly this is due to liver failure, malnutrition, advanced age, drug interactions, pregnancy, or a genetic mutation leading to impaired pseudocholinesterase activity. These conditions are fairly rare, however. Patients with myasthenia gravis, on the other hand, are more resistant to the actions of succinylcholine. It appears that doses of 0.5–0.6 mg/kg provide similar intubating conditions to 1.0 mg/kg ^[15, 16] with potential advantages of shorter apnea time, a reduced rise of serum potassium and decreased postoperative myalgias. Even smaller doses (0.1 mg/kg) can be used to relieve laryngospasm without causing apnea.

Administration of succinylcholine normally causes a transient increase in serum potassium level of 0.5 mEq/L. This is generally well tolerated in otherwise healthy patients, but patients with preexisting hyperkalemia or certain neuromuscular disorders may present an exaggerated hyperkalemic response resulting in malignant and potentially lethal cardiac arrhythmias. In patients with burns (>24 h from the injury), paraplegia (>1 week from the injury), prolonged immobilization, muscular dystrophy, stroke, and certain genetic neuromuscular disorders (e.g., Duchenne's muscular dystrophy) there is a proliferation of extrajunctional nicotinic acetylcholine receptors, leading to an exaggerated and potentially fatal hyperkalemic response to succinylcholine.

Large cumulative doses (>6 mg/kg) or extended exposure (>30 min) to succinylcholine may result in prolonged neuromuscular inhibition known as phase 2 block. Phase 2 block is a result of a conformational change in the acetylcholine receptor and resembles non-depolarizing muscle blockade. Similarly, it may be reversed with cholinesterase inhibitors, guided by appropriate neuromuscular monitoring.

Non-depolarizing Neuromuscular Blocking Agents

Non-depolarizing neuromuscular blocking agents are competitive inhibitors at the acetylcholine receptor in the neuromuscular junction. They are often used for tracheal intubation and are the agents of choice for intraoperative maintenance of paralysis. Classification is based on chemical structure (benzylisoquinolines or steroidal compounds) and duration of action.

Potency of non-depolarizing agents is based on their ED_{95} , or the average amount required to produce 95% reduction in twitch height. To ensure adequate intubating conditions in a majority of patients, a dose equivalent to approximately twice the ED_{95} is used. Dosing to provide surgical relaxation is more conservative and is guided by a peripheral nerve stimulator (to maintain approximately 80–95% depression of single-twitch height). With the

Table 3.2 Neuromuscular blocking drugs

Agent	ED ₉₅ (mg/kg)	Intubating dose (mg/kg IV)	Onset (min)	Duration (min)	Metabolism
Succinylcholine	0.3	1	0.75	5-10	Butrylcholinesterases
Mivacurium	0.08	0.15-0.2	1.5–2	12-20	Butrylcholinesterases
Atracurium	0.2	0.4	2–3	20-30	Hoffman/ester
Cisatracurium	0.05	0.15-0.2	3	20-30	Hoffman
Rocuronium	0.3	0.6	2	20-30	Biliary > renal
Vecuronium	0.05	0.1	2–2.5	20-30	Hepatic/renal/biliary
Pancuronium	0.07	0.1	3	60–90	Renal (80%)

exception of atracurium, drug potency is, paradoxically, inversely related to onset time. Onset of action can be accelerated by using increased doses (four times the ED_{05}), but at the cost of prolonged paralysis. This is particularly useful in the case of rocuronium. "Double dose" rocuronium (1.2 mg/ kg) has an onset time approaching that of succinvlcholine (60-90 s), and is the only non-depolarizing muscle relaxant approved for rapid-sequence induction by the FDA. It can be useful in cases requiring rapid-sequence induction of anesthesia where succinylcholine is not available or contraindicated. Duration of action is extended, however, to 60 min with this technique. One-tenth of the intubating dose of a non-depolarizing agent can be given to prevent fasiculations about 3 min prior to succinvlcholine administration, keeping in mind that the dose of succinylcholine will need to be increased by 50% in this case (although this small dose may increase the risk of muscle weakness and aspiration prior to induction).

Commonly used benzylisoquinolinium compounds include mivacurium, atracurium, and cisatracurium, while the steroidal compounds include rocuronium, vecuronium, and pancuronium. The combination of two drugs from the same structural class results in an additive effect, while mixing drugs from different classes will result in a synergistic response. Benzylisoquinolinium agents tend to cause a dosedependent histamine release (except cisatracurium), which can be attenuated by slow injection or divided dosing. Common dose ranges and clinical characteristics of nondepolarizing agents can be found in Table 3.2.

Benzylisoquinolinium Compounds

Mivacurium

Mivacurium, with a duration of action of approximately 20 min after an intubating dose, is the shortest acting nondepolarizing muscle relaxant. It is metabolized in the plasma by the same pseudocholinesterase (butyrylcholinesterase) that is responsible for succinylcholine's metabolism. Although it is not a trigger for malignant hyperthermia, patients who had a prolonged paralysis from succinylcholine should not receive mivacurium. Its production has been discontinued in the USA.

Atracurium

Atracurium is a benzylisoquinolone with an intermediate duration of action. Its metabolism is largely independent of hepatic or renal routes. It is metabolized by a combination of hydrolysis by nonspecific esterases and Hoffman degradation. Ester hydrolysis relies on a group of esterases found in the plasma that differ from the pseudocholinesterase enzyme that metabolizes succinylcholine. Ester hydrolysis is also responsible for degradation of esmolol and remifentanil. Hoffman degradation is a spontaneous breakdown in plasma at physiologic pH and temperature. Laudanosine is a byproduct of the Hoffman degradation of atracurium. It is a CNS excitant and a potential epileptogen, but its effect is not clinically significant except in high doses, long infusions, and liver and kidney failure.

Cisatracurium

Cisatracurium is one of the ten isomers of atracurium. It is four times as potent as atracurium and results in minimal release of laudanosine and histamine. Its elimination is by Hoffman degradation, making its more predictable pharmacokinetics useful in patients with hepatic or renal dysfunction.

Steroidal Compounds

Rocuronium

Rocuronium is the non-depolarizing relaxant with the fastest onset time. As mentioned earlier, twice the standard intubating dose can be a good alternative for succinylcholine in rapid-sequence intubations where use of succinylcholine

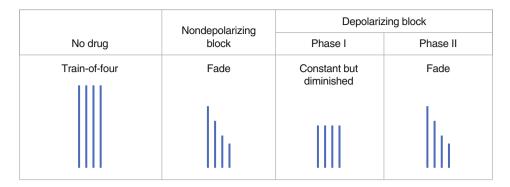


Fig. 3.3 Common neuromuscular monitoring patterns used for non-depolarizing and depolarizing blockade

may be deleterious. Elimination is biliary and renal (30%), so its action may be prolonged in renal failure or biliary tract obstruction.

Vecuronium

Vecuronium carries a similar pharmacokinetic profile to rocuronium, with the exception of a slower onset time. Elimination also takes place by biliary and renal routes.

Pancuronium

Pancuronium is a long-acting non-depolarizing agent. It has an onset of action in 3–5 min and provides blockade lasting 60–90 min. Administration results in a mild increase in heart rate and MAP, an effect mediated by both sympathomimetic and vagolytic properties. It is excreted primarily unchanged in urine so dose adjustments must be made in patients with a decreased glomerular filtration rate. If pancuronium is used as a defasicalating agent, it may prolong the duration of succinylcholine since it inhibits pseudocholinesterase activity.

Reversal of Neuromuscular Blockade

After administration of a non-depolarizing neuromuscular inhibitor, adequately gauging its metabolism, muscle recovery, and residual blockade can be difficult. Several studies have demonstrated the prevalence of residual paralysis ^[17]. Therefore, with the exception of those receiving very small doses, most patients require pharmacologic reversal prior to emergence and extubation. This is done by administration of an acetylcholinesterase inhibiting drug. These drugs act by inhibiting the breakdown of acetylcholine at the neuromuscular junction, thereby providing competition with agents for binding at the postsynaptic acetylcholine receptor.

Recovery from neuromuscular blockade may be assessed by several methods. Correlates of adequate recovery include: sustained head lift for 5 s, leg raise, ability to hold a tongue depressor between teeth, tidal volume >5 mL/kg, vital capacity >20 mL/kg, maximum negative inspiratory force <-25 cm H₂O, train of four ratio>0.9, and sustained tetanus at 100 Hz for 5 s. None of these, in isolation, reliably predicts complete reversal of neuromuscular blockade so efforts to pharmacologically reverse blockade should be used almost universally.

Reversal agents act to increase acetylcholine at both nicotinic and muscarinic receptors. This can lead to untoward and life-threatening parasympathetic stimulation. Marked bradycardia, bronchospasm, increased gastrointestinal peristalsis, hypersalivation, and pupillary constriction can all result from overriding parasympathetic stimulation. It is therefore imperative to concurrently administer an anticholinergic such as gylcopyrrolate or atropine to blunt unwanted muscarinic effects. Reliable reversal of neuromuscular blockade requires at least one twitch on train-of-four stimulation

Figure 3.3 shows common neuromuscular monitoring patterns used for non-depolarizing and depolarizing blockade. The most commonly used reversal agents are neostigmine and edrophonium. Both of these drugs have a quaternary amine structure, inhibiting passage through the blood-brain barrier. Neostigmine (0.04–0.08 mg/kg) has an onset time of 7–10 min and is given along with glycopyrrolate (0.01– 0.015 mg/kg) or atropine (0.015 mg/kg). Because glycopyrrolate has an onset time of 2–3 min, tachycardia is often seen when simultaneously administered with neostigmine. Edrophonium has an onset time of 1–2 min and is paired with a similarly fast-acting atropine.

There are many factors that can potentiate neuromuscular blockade. Inhaled anesthetics, antibiotics (except penicillins, metronidazole, and cephalosporins), local anesthetics, verapamil, magnesium sulfate, and tamoxifen all enhance neuromuscular blockade, while anticonvulsants, steroids, and azathioprine antagonize blockade. Hypothermia, acidosis and electrolyte disturbances including hypokalemia, hypermagnesemia, hypocalcaemia also augment paralysis. These should all be considered in cases of unanticipated weakness after reversal.

Local Anesthetics

Overview

Local anesthetic injection is most often used in infiltrative and regional anesthesia either as a stand-alone technique or in conjunction with sedation or general anesthesia. Local anesthetics can also be applied topically to mucous membranes and skin to provide anesthesia. In patients at risk for airway obstruction or those with difficult airways, regional anesthesia can permit patients to breathe spontaneously while maintaining muscle tone of upper and lower airways.

Local anesthetics have wide-ranging application in otolaryngology, from minor office procedures to more invasive open surgeries. Application of local anesthetic appears to decrease postoperative pain in tonsillectomy and myringotomy tube placement ^[18–20]. Lidocaine given intravenously, sprayed into the larynx, down the endotracheal tube, and even placed into the endotracheal tube cuff has been found to decrease the incidence of coughing during extubation as well as decreasing postoperative sore throat ^[21, 22].

Pharmacodynamics

Local anesthetics are sodium channel inhibitors that function by decreasing neural transmission of nocioceptive signals. The sodium-potassium ATPase is responsible for the creation of a resting membrane potential of -60 to -90 mV. Normally, following activation by excitatory signals at the synapse, activation of voltage-gated sodium channels leads to an action potential that travels down the axon. In some nerves, myelin sheaths provide an insulative layer surrounding the axon that prevents the electrical current from leaving. Impulses are then expedited down the axon by hopping from successive nodes of Ranvier (saltatory conduction). Local anesthetics function by binding to the inner aspect of neuronal sodium channels in order to inhibit such action potential propagation. It is believed that local anesthetics preferentially block small, myelinated, and high-frequency firing nerves. The clinical application of this blockade sensitivity may not be significant. Additionally, in large nerve bundles, sensory fibers are usually located more externally and are usually blocked before motor fibers. In general, blockade of sympathetic fibers occurs first, sequentially followed by pain and temperature, touch, and motor function. Differential blockade occurs depending on the drug used as well, with bupivacaine producing the greatest sensory blockade and etidocaine producing more pronounced motor block. The central nervous system and cardiovascular system are the sites where local anesthetic toxicity

predominates, and are a result of blockade of sodium channels in those organ systems.

Structure

Structurally, local anesthetics are composed of an aromatic ring and an amine group, linked centrally by either an ester or amide bond. This central portion is used to classify the two groups—esters and amides. Esters are metabolized to *p*-aminobenzoic acid by plasma cholinesterase, have a short half-life in the plasma (<1 min), and are more likely to elicit (rare) allergic reactions. Amides are degraded in the liver and have a half-life of 2–3 h. All local anesthetics are weak bases with a pKa of 7–9, existing mainly in the cationic form at physiologic pH.

Pharmacokinetics

Local anesthetics must first cross the cell membrane in the uncharged form before binding to the inner pore of the sodium channel. Local anesthetics with a pKa closer to physiologic pH are more likely to exist in the unprotonated, more hydrophobic form and therefore have a faster onset of action, with the exception of the fastest-acting agent, chlorprocaine. The speed of onset of local anesthetics is therefore inversely related to the pKa. Adding sodium bicarbonate to a local anesthetic solution in order to increase the unprotonated fraction may improve the onset of action. Potency of local anesthetics is related to their lipid solubility. Protein binding (to α -1 acid glycoprotein), peripheral vascular effects, and lipid solubility determine duration of action. Epinephrine (1:200,000) can be added to local anesthetic solutions to prolong the duration of action by causing local vasoconstriction and decreasing systemic absorption. Use of epinephrine is best avoided in peripheral nerve blocks (e.g., digital and penile blocks), patients with heart disease, and uncontrolled hypertension. Refer to Table 3.3 for pharmacological properties of individual local anesthetic agents.

Toxicity

The most likely adverse effects of local anesthetics are due to their excitatory effects on the central nervous system. Patients generally will first complain of lightheadedness or dizziness, followed by tinnitus and/or perioral numbness, become agitated, and in the worst cases progress to tonic-clonic seizures. It is therefore essential to have immediate access to resuscitation and intubation equipment in addition to anticonvulsants (midazolam 0.03–0.06 mg/kg, thiopental 1–2 mg/kg, or propofol 0.5–1 mg/kg) when performing nerve blocks.

Local anesthetic	pKa	Onset	Relative potency	Duration	Maximum dose ^a (mg/kg)
Esters					
Procaine	8.9	Slow	1	Short	11 (13)
Chlorprocaine	8.5	Fast	1	Short	10 (12)
Tetracaine		Slow	8	Long	1.5 (2)
Amides					
Prilocaine	7.9	Fast	2	Moderate	5 (7)
Lidocaine	7.9	Fast	2	Moderate	5 (7)
Mepivacaine	7.6	Fast	2	Moderate	3 (5)
Bupivacaine	8.1	Moderate	8	Long	2 (2.5)
Ropivacaine	8.1	Fast	6	Long	2 (2.5)
Etidocaine	7.7	Slow	6	Long	4

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^aFor subcutaneous infiltration. Dose in parenthesis is the maximum dose in mg/kg with the addition of epinephrine. When two different local anesthetics are used, the maximum safe dose should be assumed to be additive and not independent. 1% solution=10 mg/mL From ^[23], by permission of Oxford University Press

Hypoventilation worsens this toxicity by leading to acidosis and hypercarbia, increasing cerebral blood flow and decreasing local anesthetic binding to serum proteins.

Local anesthetics are class 1 antiarrythmic agents with depressant effects on cardiac conduction, hence the use of lidocaine to treat excitatory arrhythmias. Excessive inhibition of these channels, however, can lead to prolonged conduction and decreased inotropy. Local anesthetic overdose also leads to arteriolar dilation and hypotension. The exception to this trend is cocaine, which inhibits reuptake of norepinephrine and dopamine, leading to hypertension, increased inotropy, and tachyarrhythmias. Cocaine should be used cautiously in patients with cardiac disease as it can precipitate myocardial ischemia.

Bupivacaine has increased affinity for sodium channels and is more likely to produce severe cardiotoxic effects. Cardiovascular collapse from bupivacaine is particularly resistant to treatment and is often fatal. The use of 20% intralipid solution has been found to be effective as a rescue agent in cardiovascular collapse from bupivacaine that is unresponsive to traditional resuscitation protocols ^[24–26]. Ropivacaine is a stereoisomer of bupivacaine that has reduced affinity for cardiac sodium channels and is accordingly less cardiotoxic (although still more cardiotoxic than lidocaine). With all local anesthetics, aspiration prior to injection decreases the risk of intravascular injection.

Topical Application of Local Anesthetic

Besides use by injection in regional nerve blocks and tissue infiltration, local anesthetic agents can also be used topically. Local anesthetic sprays are available for application to the esophagus and larynx for endoscopic procedures. Several agents can be sprayed into the larynx and trachea prior to intubation to reduce the incidence of coughing during emergence from anesthesia (laryngotracheal anesthesia)^[21] and decrease the sympathetic response to tracheal intubation. The cricothyroid membrane may also be punctured with a small needle for the direct application of local anesthetic to the larynx and trachea. Local anesthetics can be delivered via nebulizer for endoscopy or awake intubation (Chap. 8).

Eutectic mixture of local anesthetics (EMLA) is a cream composed of 2.5% lidocaine and 2.5% prilocaine that may be applied to intact skin as a local anesthetic. Use is limited by onset time (requires 60 min) and risk of methemoglobinemia with prilocaine, to which newborns are more susceptible.

Inhaled Anesthetics

Inhaled anesthetics are a cornerstone in anesthesia practice and are the classic agents for maintenance of general anesthesia. They provide a relatively predictable depth of anesthesia and are easily and quickly titratable. Inhaled agents provide an element of hypnosis, analgesia, and skeletal muscle relaxation with relatively fast onset and offset and are therefore fairly "complete" anesthetics. The current generation of agents in use includes halogenated agents (isoflurane, sevoflurane, and desflurane) along with nitrous oxide (Table 3.4). Although halothane is still commercially available its use is limited and will not be discussed here.

Inhaled anesthetic agents' pharmacokinetics are unique from other agents used in anesthetic practice, as they are inert and rely primarily on the lungs for

Table 3.4	Properties of common	ly used inhalational agents
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Agent	MAC% ^a	Vapor Pressure (mm Hg at 20 °C)
Nitrous oxide	105	-
Isoflurane (forane)	1.2	240
Desflurane (suprane)	6.0	681
Sevoflurane (ultane)	2.0	160

^a minimum alveolar concentation in adults

absorption and elimination. After absorption by the lungs into the bloodstream, they are carried to the central nervous system where they exert their anesthetic effects. The level of agent can then be measured in the exhalation limb of the breathing circuit by as analysis techniques (spectrometry) and used to assess the depth of anesthesia. Inhaled agents are absorbed from alveoli until reaching equilibrium with the blood. At this point, inspired and expired concentrations of the anesthetic are approximately equal and the desired concentration has been reached.

The rapidity of onset and offset of each individual agent is determined by its solubility in the blood and brain. Agents that are less soluble in the bloodstream reach equilibrium more rapidly, leading to a faster onset. The "vessel-rich" organs (brain, heart, kidneys, digestive tract, and endocrine organs) receive approximately 75% of the cardiac output, leading to a faster equilibration of blood and tissue partial pressures than other tissues such as muscle and fat. It is therefore useful to recognize that agents with a lower blood:gas solubility (desflurane, nitrous oxide) have a faster onset than those which are more soluble in the blood (isoflurane). This effect can be overcome with more soluble agents by increasing the delivery of inhaled anesthetics in one of several ways: increased alveolar ventilation, increased concentration (partial pressure), decreasing circuit capacity/ absorption, and increased fresh gas flows.

The three factors which affect the uptake of inhaled anesthetics are the solubility, alveolar-to-venous partial pressure difference, and the cardiac output. The uptake equation describes the interaction of these factors:

Uptake = Solubility \times (PI – PA)/PB \times Cardiac Output

where PI=partial pressure of inspired gas; PA=partial pressure of alveolar gas; and PB=barometric pressure.

Somewhat counterintuitive is the fact that patients with impaired cardiac output have faster induction times than those with normal or increased cardiac outputs. This is a result of faster equilibration of alveolar and inspired partial pressures leading to faster saturation of the bloodstream.

The minimum alveolar concentration (MAC) is the concentration of volatile anesthetic agent in the lungs that

Table 3.5 Factors affecting minimum alveolar concentration (MAC)			
Increased MAC	Decreased MAC		
Children (from infancy to adolescence)	Premature infants and the elderly		
Hypernatremia	Hypothermia		
Cocaine or amphetamine	Pregnancy		
intoxication	Acute alcohol intoxication		
Chronic alcohol use	Opiates, benzodiazepines,		
MAO inhibitors	barbiturates, clonidine,		
Tricyclic antidepressants	dexmedetomidine		

From Adams, Jerome M.; Wolfe, John W. Pharmacology of inhalational anesthetics. In: Ehrenfeld JM, Urman RD, Segal S, editors. Anesthesia student survival guide: a case based approach. Springer; 2010

prevents movement to an abdominal incision in 50% of subjects. MAC levels vary between anesthetic agents and may be used as a measure of depth of anesthesia. 1.3 (times) MAC prevents movement in 99% of patients, 1.7-2 times MAC (MAC-BAR) suppresses adrenergic responses to noxious stimulus, and 0.3-0.5 MAC is generally thought to be sufficient to prevent awakening or awareness. Isoflurane is the most potent agent, with a MAC of 1.2%, followed by sevofluorane (2%), desflurane (6%), and nitrous oxide (105%). MAC values are higher in neonates and lowest in the elderly population, decreasing by approximately 6% per decade of life. MAC values are additive when more than one agent is used. Many other factors influence the MAC (Table 3.5). The mechanism of action of volatile anesthetics is still not entirely known, but is thought to occur by enhancing the function of inhibitory neurotransmitters and decreasing the functions of excitatory neurotransmitters in the CNS.

The same principles that apply to delivery of inhaled agents apply to recovery or "washout" of the anesthetic. Poorly soluble agents are removed from the blood more rapidly and thus result in a faster recovery from anesthesia. Increasing fresh gas flow leads to decreased rebreathing of anesthetic agent and also speeds up awakening. With increasing times under anesthesia, more volatile anesthetic becomes equilibrated into the vessel-poor groups (muscle, fat), which can act as a depot for anesthetics and delay recovery. Inhaled agents are primarily eliminated through the lungs. Metabolism is not thought to significantly affect pharmacokinetics and occurs in the liver (<1%). Sevoflurane metabolism is associated with increased levels of fluoride and compound A in the bloodstream, which carry a theoretical but not clinically significant nephrotoxicity risk in humans. This is increased with use of lower fresh gas flows, which decreases washout and increases the temperature of soda lime absorbent. Two liters per minute has been set as a mandatory minimum flow rate in Canada and Australia and some practitioners choose to use this as a minimum, leading to increased costs with the use of sevoflurane.

Respiratory Affects

Volatile anesthetics all produce a "rapid, shallow" breathing pattern, with a decrease in tidal volume and increase in respiratory rate. Minute ventilation is mildly depressed. The ventilatory responses to hypoxia and hypercarbia are blunted in a dose-dependent fashion. Hypoxic pulmonary vasoconstriction is conserved at lower doses of volatile agent (but is impaired at increasing doses above 1 MAC) allowing for their use in one-lung ventilation. Volatile agents are bronchodilators and increasing the concentration of potent inhaled anesthetic can be used as an adjunct to relieve bronchospasm with minimal decrease in FiO₂. Nitrous oxide directly increases pulmonary vascular resistance and should be avoided in patients with pulmonary hypertension.

Cardiovascular Effects

Potent inhaled anesthetics decrease MAP and cardiac contractility in a dose-dependent fashion. Impaired cardiac contractility occurs from alterations in myocyte calcium homeostasis. There is direct vasodilation followed by a compensatory increase in heart rate to maintain a relatively normal cardiac output. Nitrous oxide causes venoconstriction, mild tachcyardia, increased cardiac output, and maintenance of blood pressure. Inhaled anesthetics do cause prolongation of the QT interval but none of the agents discussed here increases the rate of ventricular dysrhythmias. The potent inhaled agents also have the benefit of mimicking cardiac myocyte ischemic preconditioning at the molecular level—using a potent inhaled agent for at least 5–10 min at >1 MAC improves the heart's ability to respond to ischemic and reperfusion injury ^[27, 28].

Inhaled Agents

Isoflurane

Isoflurane is the most soluble and most potent inhaled agent (MAC 1.2%, blood:gas partition coefficient 1.4), and therefore carries the slowest pharmacokinetics of the commonly used agents. Delivering a higher concentration can speed the induction of anesthesia, but awakening can be slow ^[6]. It should therefore be carefully decreased towards the end of the case to prevent prolonged awakening. The principal advantage to isoflurane is its low cost. A common strategy is to use isoflurane to maintain anesthesia for the beginning and majority of the case, and then switch to a faster agent such as desflurane towards the end of the procedure. Isoflurane also causes the greatest increase in heart rate of all inhaled agents at 1 MAC.

Sevoflurane

Sevoflurane (MAC 2.0%, blood:gas partition coefficient 0.65) is the least pungent of the halogenated agents, making it the most suitable for inhalation induction ^[29]. This can be useful in pediatric patients in whom intravenous access can be difficult, and is also used in patients where maintenance of spontaneous ventilation is desired. It is also commonly used in brief procedures where mask ventilation can be used to provide a deep general anesthetic without instrumentation of the airway. Sevoflurane does not cause an increase in heart rate.

Desflurane

Desflurane is the least potent and least soluble halogenated anesthetic (MAC 6%, blood:gas partition coefficient 0.42). It has the fastest onset and offset [30-33], allowing it to be more easily titrated to the desired depth, as well as decreasing the time to awakening after surgery. Its boiling point is near room temperature, 23.5 °C, and vapor pressure-660 also important in this discussion it therefore requires a special vaporizer (TEC 6) that actively heats the desflurane to 39.5 °C, ensuring a reliable and predictable level of delivery. It may be the most pungent inhaled agent and may have a higher frequency of cough and laryngospasm than other inhaled anesthetics [30]. A tachycardic and hypertensive response can be seen with high or rapidly increasing inspired concentrations. There is also a risk of carbon monoxide production when dry carbon dioxide absorbents are used with desflurane.

Nitrous Oxide

Nitrous oxide is another fast-acting inhaled agent that, due to its low potency, is often used in combination with other inhaled agents, intravenous agents, or narcotics. It is not pungent and can be used in combination with sevoflurane for mask induction of anesthesia. One of its main advantages is its lack of hemodynamic depression in vivo and its possible mild sympathomimetic effects. It is also believed to provide more analgesia than the other inhaled agents. Early studies estimated 30% nitrous oxide to be equi-analgesic to 10–15 mg morphine. A commonly used technique involves the combination of 60–70% nitrous oxide along with an opioid (nitrousnarcotic technique). The use of a shorter-acting opioid such as alfentanil, sufentanil, or remifentanil can make this technique particularly useful in procedures where rapid emergence is desired and brief periods of intense stimulation are encountered.

Nitrous oxide is much more soluble than nitrogen (34 times more soluble) in the blood. This allows it to enter closed air spaces in the body 34 times quicker then nitrogen can exit. This will lead to an increase in the volume of the enclosed air space. Therefore, its use should be avoided in patients with a pneumothorax, bowel obstruction, laparoscopy, and posterior fossa craniotomies, where there is increased risk for air embolization. It should also be discontinued at least 30 min prior to overlay graft placement in tympanic membrane surgery as an increased middle ear pressure may dislodge the graft.

Use of nitrous oxide does lead to a slightly increased incidence in postoperative nausea and vomiting, which is more pronounced in female patients ^[34]. This effect can be lessened by the concomitant use of propofol or neutralized with a prophylactic dose of ondansetron. It can be safely used in patients with a history of malignant hyperthermia, unlike the other volatile agents. Its inhibition of vitamin B-12 dependent enzymes can potentially cause megaloblastic anemia with prolonged use (>24 h). Nitrous oxide supports combustion, so it should be avoided in laser surgeries or any time airway fire is a real risk.

Autonomic Drugs

The autonomic nervous system (ANS) is responsible for maintenance of homeostasis and normal organ function. A more thorough review of the system can be found in Chap. 4, but a brief description follows below.

The Sympathetic Nervous System

Alpha-1 (a1) Adrenergic Receptors

Agonism of alpha-1 (α 1) adrenergic receptors results in constriction of smooth muscle, with the most relevant being vascular smooth muscle. Phenylephrine and norepinephrine are commonly used agonists and are often used to treat hypotension, especially when due to vasodilatory causes (e.g., sepsis, spinal anesthesia). Epinephrine and dopamine, when given at higher doses, also have α 1 agonist effects and the former is used in the treatment of hypotension secondary to low cardiac output states as well as anaphylactic shock. Phenylephrine is most specific to the α 1 receptor and the increase in blood pressure from phenylephrine may cause a reflex decrease in heart rate. Phenylephrine and oxymetazoline (Afrin), another α 1 agonist, are also useful for their vasoconstrictive effects on nasal mucosa and are often employed as nasal decongestants. The efficacy of phenylephrine as a nasal decongestant, however, has yielded inconsistent results in studies ^[35–37], is less efficacious ^[38], and is associated with more side effects than oxymetazoline ^[39]. Phenylephrine may be applied prior to nasal intubation in order to dilate the passageway and decrease the incidence of epistaxis, although it does carry a risk of significant hypertension ^[40]. Phenylephrine can also be added to local anesthetic injections in order to decrease systemic toxicity.

The major $\alpha 1$ antagonists include prazosin, phenoxybenzamine, and phentolamine. Prazosin is an antihypertensive that is also indicated for the treatment of prostatic hyperplasia due to its effects on urethral and prostatic $\alpha 1$ receptors. Phenoxybenzamine and phentolamine are used in the treatment of hypertension resulting from pheochromocytoma.

Alpha-2 (a2) Adrenergic Receptor

The alpha-2 (α 2) adrenergic receptor is a presynaptic receptor that is responsible for negative feedback, or inhibition, of the sympathetic response. Clonidine and dexmedetomidine are two α 2 antagonists that produce sedation, anxiolysis, and a decrease in blood pressure. Both of these agents decrease the inhaled anesthetic requirements. Abrupt discontinuation of clonidine prior to surgery can result in a rebound hypertension and is therefore discouraged. Clonidine may decrease the risk of myocardial ischemia and mortality in the perioperative period ^[41]. It may be used in lieu of β -blockers for this purpose in situations where they are contraindicated (bronchospasm, bradycardia).

Beta-1 (β1) Adrenergic Receptors

Beta-1 (β 1) adrenergic receptors are located in the SA node, AV node, and ventricular muscle of the heart and are responsible for increased myocardial chronotropy (heart rate) and inotropy (contractility). Agonism of β 1 receptors is commonly employed in the management of low cardiac output states such as acute heart failure and hypotension. Blockade of the β 1 receptor is the primary goal of β -blocker therapy. β -blockers are used in the management of hypertension, certain tachycardias, angina, aortic dissection, and chronic congestive heart failure. β -blocker drugs which are most specific for the β 1 receptor (atenolol, betaxolol, esmolol, acebutolol, and metoprolol) are preferred in patients at risk for bronchospasm. The use of β -blockers in high risk patients has been shown to decrease the incidence of perioperative myocardial ischemia ^[42]. Abrupt discontinuation of β -blockers can result in a rebound tachycardia and is another reason that their use should be continued throughout the perioperative period, as tolerated.

Drug	Mechanism of action	Infusion rate	Side effects
Phenylephrine	al receptor	0.1-1.0 mcg/kg/min	Reflex bradycardia
Norepinephrine	$\alpha > \beta 1$ receptor	0.05–0.5 mcg/kg/min	Increased myocardial oxygen consumption, potential vasoconstriction of blood supply to organs
Epinephrine	$\beta > \alpha$ receptor	0.01-0.5 mcg/kg/min	Palpitations, splanchnic ischemia
Vasopressin	V1 receptor	0.02–0.1 U/kg/h	Excessive vasoconstriction to end-organs
Dopamine	$D1 > \beta 1 > \alpha$ receptor	2-20 mcg/kg/min	Tachyarrhythmias
Dobutamine	β1>β2	2-30 mcg/kg/min	Tachycardia, ventricular ectopy, hypotension
Milrinone	Phosphodiesterase inhibition	0.375-0.75 mcg/kg/min	Hypotension, thrombocytopenia
Nitroglycerin	↑ Nitric oxide	0.5-8 mcg/kg/min	Methemoglobinemia, tolerance, headache, reflex tachycardia
Sodium nitroprusside	↑ Nitric oxide	0.5-8 mcg/kg/min	Thiocyanate toxicity, cyanide toxicity

Beta-2 (β2) Adrenergic Receptors

Beta-2 (β 2) receptors are located in bronchial smooth muscle, vascular smooth muscle supplying skeletal muscle, and in the walls of the bladder and uterus. The effect of the β 2 receptor is to produce relaxation of smooth muscle. β 2 agonists used in the treatment of asthma are albuterol and, in severe cases, epinephrine. β 2 agonists ritodrine and terbutaline are used as tocolytic agents in obstetrics as they provide uterine smooth muscle relaxation. There are many nonspecific β -blockers (block both β 1 and β 2 receptors) on the market, but specific β 2 antagonists do not play a role in the clinical setting. Labetalol and carvedilol are nonspecific β -blockers which have the additional effect of α 1 antagonism. Labetalol is available in intravenous form and is quite effective in the treatment of acute and chronic hypertension.

Table 3.6 Commonly administered vasoactive and vasodilator drugs

Autonomic Agents for Otolaryngologic Patients

There are many vasoactive drugs used in the perioperative management of otolaryngologic patients. Although there is a very large spectrum of drugs, only the most commonly used intravenous agents will be discussed here. Table 3.6 summarizes these drugs.

Ephedrine

Ephedrine is an indirect-acting sympathomimetic drug used in the treatment of hypotension. It acts to release stored norephinephrine from presynaptic vesicles which acts at α - and β -receptors to increase blood pressure through vasoconstriction (predominantly venous) and an increase in cardiac output through an increase in both heart rate and myocardial contractility. Small boluses (2.5–20 mg intravenously) are effective in the treatment of mild-moderate hypotension. After repeated doses, tachyphylaxis may be seen as norephinephrine stores are depleted from the presynaptic nerve terminals.

Epinephrine

Epinephrine is an endogenous catecholamine. At low doses (0.01–0.05 mcg/kg/min), epinephrine stimulates predominantly β receptors, causing an increase in cardiac contractility as well as skeletal muscle vasodilation and thus possibly decreasing diastolic blood pressure. Greater doses of epinephrine can be used for the treatment of hypotension, as α receptor agonism increases and vasoconstriction occurs. In addition to the uses listed above, epinephrine is an excellent bronchodilator and is the treatment of choice in anaphylaxis (300 mcg subcutaneously or intramuscularly) and racemic epinephrine can be given in an inhaled form to treat croup and upper airway edema causing stridor. Side effects include palpitations, arrhythmias, and impaired splanchnic circulation ^[43].

Norepinephrine

Norepinephrine is a potent α -adrenergic receptor agonist as well as a moderate β 1 agonist. It is a first-line vasopressor in the treatment of septic shock ^[44] and can also be useful in cardiogenic shock accompanied by vasodilation. It lacks β 2 agonist activity and is not effective in the treatment of bronchoconstriction. Side effects include increased myocardial oxygen consumption and vasoconstriction with theoretically decreased end-organ perfusion at higher infusion rates.

Vasopressin

Vasopressin is a hormone that is released by the posterior pituitary gland in response to reductions in plasma volume, painful stimulation, and increased serum osmolality. It is a potent vasoconstrictor that is used in the treatment of septic shock ^[45], refractory shock due to other circumstances, and cardiac arrest. It is believed to cause vasoconstriction by agonism at V1 receptors on vascular smooth muscle cells. Serum levels of vasopressin are thought to be relatively deficient in many forms of shock [46] and a dose of 0.02-0.1 U/kg/h or 0.01-0.04 U/min is used for its support of the blood pressure via vasoconstriction. Vasopressin has relatively little effect on pulmonary vascular resistance due to absence of V1 receptors in this vascular tree. It may be the drug of choice to treat significant hypotension during anesthesia in patients on angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blocking (ARBS) agents

Dopamine

Dopamine is a catecholamine that is used in the management of hypotension and septic shock [44]. At low doses (0.5-2 mcg/ kg/min), it is an agonist primarily on dopamine receptors. At this dose, it does not have much of a hypertensive effect but was postulated to improve renal blood flow and urine output through its effects on renal dopamine receptors. This has not been shown to affect the outcome of acute renal failure in clinical practice, however [47]. At moderate doses (2–10 mcg/ kg/min), dopamine stimulates beta adrenergic receptors to cause an increase in cardiac output and peripheral vasodilation. At even higher doses (10-20 mcg/kg/min), alpha receptor effects predominate, leading to increased systemic and pulmonary vasoconstriction. Its use is limited by the increased risk of tachyarrythmias, severe vasoconstriction, and increased cardiac filling pressures and myocardial oxygen consumption.

Dobutamine

Dobutamine is a synthetic beta receptor agonist with a greater affinity for β 1 than β 2 receptors. It acts to increase cardiac output (via increased contractility) and cause peripheral vasodilation. The net result is an increase in cardiac output with a relatively unchanged blood pressure and heart rate. Similar to dopamine, increased myocardial oxygen consumption limits its utility in patients with coronary ischemia. Tachyphylaxis can occur with long-term infusions.

Milrinone

Milrinone is bipyridine phosphodiesterase isozyme 3 inhibitor that is used in the acute management of heart failure. It improves myocardial contractility and is a peripheral vasodilator, causing a decrease in blood pressure. Although it may produce an improved hemodynamic profile and improvement in symptoms, morbidity may be increased, especially in the face of ischemic cardiomyopathy ^[48, 49]. Use of milrinone increases the risk of ventricular dysrhythmias.

Nitroglycerin

Nitroglycerin is commonly used in the management of hypertension, congestive heart failure, and angina. It acts by increasing nitric oxide (and thus increasing intracellular cGMP), causing vasodilation. Venodilation predominates, leading to decreases in blood pressure, preload to the heart, filling pressures, and myocardial oxygen demand. It also may redistribute coronary perfusion to ischemic areas, relieve coronary spasm, and decrease platelet aggregation. Its short half-life allows for easier titration when given intravenously. Pitfalls of nitroglycerin include reflex tachycardia, headache, inhibition of hypoxic vasoconstriction and increased $R \rightarrow L$ shunt, as well as tachyphylaxis, and methemoglobinemia with infusions of longer duration.

Sodium Nitroprusside

Sodium nitroprusside as another antihypertensive agent commonly used in the management of acute hypertension and hypertensive emergencies. It has a rapid onset and short halflife, making it ideal for use in intravenous infusions. In contrast to nitroglycerin, sodium nitroprusside is predominantly an arteriolar dilator. Adverse effects include thiocyanate toxicity (especially in patients with renal failure) and cyanide toxicity.

Practical Considerations

Control of hemodynamics is obviously one of the most important aspects of the perioperative care of patients. There are a number of medications that can be used to manipulate systemic vascular resistance, heart rate, and cardiac contractility. It is important to note that these medications should not be substituted as a treatment for the underlying causes of the hemodynamic changes or should only be used as temporizing measures if they are used for this purpose. For example, a patient who is hypertensive may have one of a number of causative factors (pain, anxiety, etc.) and these should be ruled out before treatment with an antihypertensive agent. On the other hand, patients with severe hypertension or underlying cardiovascular risk factors which may be life-threatening (i.e., aortic dissection, unstable angina) must be treated promptly prior to or while seeking out an underlying cause. There is concern regarding the use of vasoconstrictive therapies in free flap surgeries and their potential negative influence on flap blood flow and survival. Although this may not be supported by scientific data [50, 51], vasopressor use in these procedures should be very judicious nonetheless and a search for underlying sources of hypotension (e.g., bleeding) should be thoroughly performed. This is discussed in detail in Chap. 16, Head and Neck Cancer Surgery II: Reconstruction.

Catecholamines are frequently injected prior to incision either with local anesthetic agents or used alone in otolaryngologic surgeries. Injection of epinephrine in 1:100,000–1:200,000 concentrations (10 or 5 mcg/mL, respectfully) is commonly combined with local anesthetic agent to prolong the action of the local anesthetic and decrease its systemic toxicity by causing local vasoconstriction. The maximum recommended dose of epinephrine in adults is 200 mcg. Higher doses may cause hypertension, tachycardia, and arrhythmias ^[52]. The addition of epinephrine may not prolong analgesia in nasal injections and its use should be weighed against the risks ^[53, 54].

The Parasympathetic Nervous System

The parasympathetic nervous system controls most vegetative processes and can be summarized by the phrase "rest and ruminate." Preganglionic neurons are located in both the cranium (cranial nerves III, VII, IX, and X) and the sacrum (spinal cord segments S2–S4). Of the cranial nerves, the vagus (cranial nerve X) is responsible for the majority of systemic parasympathetic regulation, while the sacral spinal cord segments control the distal colon and genitourinary tract.

The vagus nerve and its branches are responsible for several important reflexes in the head and neck including the gag reflex, oculocardiac reflex, and efferent fibers of the carotid sinus reflex. It also innervates the muscles of the larynx. The glossopharyngeal nerve (cranial nerve IX) is responsible for the afferent reflexes of the baroreceptors of both the carotid sinus and the gag reflex. Intraoperative stimulation of these reflexes can cause massive parasympathetic responses resulting in bradycardia or asystole which should

Table 3.7 Anticholinergic drugs

Drug	Sedation	Antisialagogue	Heart rate
Atropine	+	+	+++
Glycopyrrolate	0	++	++
Scopolamine	+++	+++	+

be treated first by removal of the stimulus as well as by the use of the anticholinergic drugs atropine (faster onset) and glycopyrrolate.

Muscarinic Acetylcholine Receptors

Muscarinic acetylcholine receptors are located at the postsynaptic parasympathetic sites of the heart, smooth muscle, and glands. Agonism at these sites causes bradycardia, increased gastrointestinal motility, and increased secretions, respectively. Three commonly used anticholinergic agents in anesthetic practice are atropine, glycopyrrolate, and scopolamine. Their utility in the treatment of bradycardia and as antisialogogues and sedatives is summarized in Table 3.7.

Conclusion

A thorough working knowledge of the major pharmacologic agents employed in anesthesia for otolaryngologic procedures is of paramount importance to anesthesia providers and surgeons alike. While several texts are devoted entirely to anesthetic pharmacology, this chapter should be a primer on most of the commonly used agents.

References

- Hughes MA, Glass PS, Jacobs JR. Context-sensitive half-time in multicompartment pharmacokinetic models for intravenous anesthetic drugs. Anesthesiology. 1992;76(3):334–41.
- Pasternak G. Molecular biology of opiod analgesia. J Pain Symp Manage. 2005;29:S2–9.
- Mitra S, Sinatra RS. Perioperative management of acute pain in the opioid-dependent patient. Anesthesiology. 2004;101(1):212–27.
- Nho JS, Lee SY, Kang JM, et al. Effects of maintaining a remifentanil infusion on the recovery profiles during emergence from anaesthesia and tracheal extubation. Br J Anaesth. 2009;103(6): 817–21.
- Aouad MT, Al-Alami AA, Nasr VG, Souki FG, Zbeidy RA, Siddik-Sayyid SM. The effect of low-dose remifentanil on responses to the endotracheal tube during emergence from general anesthesia. Anesth Analg. 2009;108(4):1157–60.
- Gupta A, Stierer T, Zuckerman R, Sakima N, Parker SD, Fleisher LA. Comparison of recovery profile after ambulatory anesthesia with propofol, isoflurane, sevoflurane and desflurane: a systematic review. Anesth Analg. 2004;98(3):632–41.

- Hohlrieder M, Tiefenthaler W, Klaus H, et al. Effect of total intravenous anaesthesia and balanced anaesthesia on the frequency of coughing during emergence from the anaesthesia. Br J Anaesth. 2007;99(4):587–91.
- Hans P, Marechal H, Bonhomme V. Effect of propofol and sevoflurane on coughing in smokers and non-smokers awakening from general anaesthesia at the end of a cervical spine surgery. Br J Anaesth. 2008;101(5):731–7.
- Eberhart LH, Folz BJ, Wulf H, Geldner G. Intravenous anesthesia provides optimal surgical conditions during microscopic and endoscopic sinus surgery. Laryngoscope. 2003;113(8):1369–73.
- Wormald PJ, van Renen G, Perks J, Jones JA, Langton-Hewer CD. The effect of the total intravenous anesthesia compared with inhalational anesthesia on the surgical field during endoscopic sinus surgery. Am J Rhinol. 2005;19(5):514–20.
- Picard P, Tramèr MR. Prevention of pain on injection with propofol: a quantitative systematic review. Anesth Analg. 2000;90:963–9.
- Bloomfield R, Noble DW. Etomidate, pharmacological adrenalectomy and the critically ill: a matter of vital importance. Crit Care. 2006;10(4):161.
- Talke P, Chen R, Thomas B, et al. The hemodynamic and adrenergic effects of perioperative dexmedetomidine infusion after vascular surgery. Anesth Analg. 2000;90(4):834–9.
- Wunsch H, Kahn JM, Kramer AA, et al. Dexmedetomidine in the care of critically ill patients from 2001 to 2007: an observational cohort study. Anesthesiology. 2010;113(2):386–94.
- Naguib M, Samarkandi AH, El-Din ME, Abdullah K, Khaled M, Alharby SW. The dose of succinylcholine required for excellent endotracheal intubating conditions. Anesth Analg. 2006;102(1): 151–5.
- El Orbany MI, Joseph NJ, Salem MR, Klowden AJ. The neuromuscular effects and tracheal intubation conditions after small doses of succinylcholine. Anesth Analg. 2004;98:1680–5.
- Murphy GS, Brull SJ. Residual neuromuscular block: lessons unlearned. Part I: definitions, incidence, and adverse physiologic effects of residual neuromuscular block. Anesth Analg. 2010;111(1):120–8.
- Lawhorn CD, Bower CM, Brown Jr RE, et al. Topical lidocaine for postoperative analgesia following myringotomy and tube placement. Int J Pediatr Otorhinolaryngol. 1996;35(1):19–24.
- Bhananker SM, Azavedo L, MacCormick J, Splinter W. Topical lidocaine and oral acetaminophen provide similar analgesia for myringotomy and tube placement in children. Can J Anaesth. 2006;53(11):1111–6.
- Sun J, Wu X, Meng Y, Jin L. Bupivacaine versus normal saline for relief of post-adenotonsillectomy pain in children: a meta-analysis. Int J Pediatr Otorhinolaryngol. 2010;74(4):369–73.
- Minogue SC, Ralph J, Lampa MJ. Laryngotracheal topicalization with lidocaine before intubation decreases the incidence of coughing on emergence from general anesthesia. Anesth Analg. 2004; 99(4):1253–7.
- 22. Venkatesan T, Korula G. A comparative study between the effects of 4% endotracheal tube cuff lignocaine and 1.5 mg/kg intravenous lignocaine on coughing and hemodynamics during extubation in neurosurgical patients: a randomized controlled double-blind trial. J Neurosurg Anesthesiol. 2006;18:230–4.
- Covino BG. Pharmacology of local anaesthetic agents. Br J Anaesth. 1986;58(7):701–16.
- 24. Weinberg G, Ripper R, Feinstein DL, Hoffman W. Lipid emulsion infusion rescues dogs from bupivacaine-induced cardiac toxicity. Reg Anesth Pain Med. 2003;28(3):198–202.
- Rosenblatt MA, Abel M, Fischer GW, Itzkovich CJ, Eisenkraft JB. Successful use of a 20% lipid emulsion to resuscitate a patient after a presumed bupivacaine-related cardiac arrest. Anesthesiology. 2006;105(1):217–8.
- Litz RJ, Roessel T, Heller AR, Stehr SN. Reversal of central nervous system and cardiac toxicity following local anesthetic intoxi-

cation by lipid emulsion injection. Anesth Analg. 2008;106(5): 1575–7.

- Frässdorf J, Borowski A, Ebel D, et al. Impact of preconditioning protocol on anesthetic-induced cardioprotection in patients having coronary artery bypass surgery. J Thorac Cardiovasc Surg. 2009; 137(6):1436–42.
- De Hert SG, Van der Linden PJ, Cromheecke S, et al. Cardioprotective properties of sevoflurane in patients undergoing coronary surgery with cardiopulmonary bypass are related to the modalities of its administration. Anesthesiology. 2004;101(2): 299–310.
- TerRiet MF, DeSouza GJ, Jacobs JS, et al. Which is most pungent: isoflurane, sevoflurane or desflurane? Br J Anaesth. 2000;85(2): 305–7.
- White PF, Tang J, Wender RH, et al. Desflurane versus sevoflurane for maintenance of outpatient anesthesia: the effect on early versus late recovery and perioperative coughing. Anesth Analg. 2009; 109(2):387–93.
- Agoliati A, Dexter F, Lok J, et al. Meta-analysis of average and variability of time to extubation comparing isoflurane with desflurane or isoflurane with sevoflurane. Anesth Analg. 2010; 110(5):1433–9.
- 32. Juvin P, Vadam C, Malek L, Dupont H, Marmuse JP, Desmonts JM. Postoperative recovery after desflurane, propofol, or isoflurane anesthesia among morbidly obese patients: a prospective, randomized study. Anesth Analg. 2000;91(3):714–9.
- Nordmann GR, Read JA, Sale SM, Stoddart PA, Wolf AR. Emergence and recovery in children after desflurane and isoflurane anaesthesia: effect of anaesthetic duration. Br J Anaesth. 2006;96(6):779–85.
- Fernández-Guisasola J, Gómez-Arnau JI, Cabrera Y, del Valle SG. Association between nitrous oxide and the incidence of postoperative nausea and vomiting in adults: a systematic review and metaanalysis. Anaesthesia. 2010;65(4):379–87.
- 35. Horak F, Zieglmayer P, Zieglmayer R, et al. A placebo-controlled study of the nasal decongestant effect of phenylephrine and pseudoephedrine in the Vienna Challenge Chamber. Ann Allergy Asthma Immunol. 2009;2(102):116–20.
- 36. Kollar C, Schneider H, Waksman J, Krusinska E. Meta-analysis of the efficacy of a single dose of phenylephrine 10 mg compared with placebo in adults with acute nasal congestion due to the common cold. Clin Ther. 2007;29(6):1057–70.
- Hatton RC, Winterstein AG, McKelvey RP, Shuster J, Hendeles L. Efficacy and safety of oral phenylephrine: systematic review and meta-analysis. Ann Pharmacother. 2007;41(3):381–90.
- Caenen M, Hamels K, Deron P, Clement P. Comparison of decongestive capacity of xylometazoline and pseudoephedrine with rhinomanometry and MRI. Rhinology. 2005;43(3):205–9.
- McCluney NA, Eng CY, Lee MS, McClymont LG. A comparison of xylometazoline (Otrivine) and phenylephrine/lignocaine mixture (Cophenylcaine) for the purposes of rigid nasendoscopy: a prospective, double-blind, randomised trial. J Laryngol Otol. 2009;123(6):626–30.
- Groudine SB, Hollinger I, Jones J, DeBouno BA. New York State guidelines on the topical use of phenylephrine in the operating room. The Phenylephrine Advisory Committee. Anesthesiology. 2000;92(3):859–64.
- Wijeysundera DN, Bender JS, Beattie WS. Alpha-2 adrenergic agonists for the prevention of cardiac complications among patients undergoing surgery. Cochrane Database Syst Rev. 2009;7(4): CD004126.
- 42. American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, et al. ACCF/AHA focused update on perioperative beta blockade incorporated into the ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery. J Am Coll Cardiol. 2009;54(22):e13–118.

- 43. De Backer D, Creteur J, Silva E, Vincent JL. Effects of dopamine, norepinephrine, and epinephrine on the splanchnic circulation in septic shock: which is best? Crit Care Med. 2003; 31(6):1659–67.
- 44. Dellinger RP, et al. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock: 2008. Crit Care Med. 2008;36(1):296–327.
- 45. Delmas A, Leone M, Rousseau S, Albanèse J, Martin C. Clinical review: vasopressin and terlipressin in septic shock patients. Crit Care. 2005;9(2):212–22.
- Landry DW, Levin HR, Gallant EM, et al. Vasopressin deficiency contributes to the vasodilation of septic shock. Circulation. 1997; 95:1122–5.
- Bellomo R, Chapman M, Finfer S, Hickling K, Myburgh J. Lowdose dopamine in patients with early renal dysfunction: a placebocontrolled randomised trial. Australian and New Zealand Intensive Care Society (ANZICS) Clinical Trials Group. Lancet. 2000; 356(9248):2139–43.
- Cuffe MS, Califf RM, Adams Jr KF, et al. Short-term intravenous milrinone for acute exacerbation of chronic heart failure: a randomized controlled trial. JAMA. 2002;287(12):1541–7.

- Felker GM, Benza RL, Chandler AB, et al. Heart failure etiology and response to milrinone in decompensated heart failure: results from the OPTIME-CHF study. J Am Coll Cardiol. 2003;41(6): 997–1003.
- Monroe MM, McClelland J, Swide C, Wax MK. Vasopressor use in free tissue transfer surgery. Otolaryngol Head Neck Surg. 2010;142(2):169–73.
- Chen C, Nguyen MD, Bar-Meir E, et al. Effects of vasopressor administration on the outcomes of microsurgical breast reconstruction. Ann Plast Surg. 2010;65(1):28–31.
- Higgins TS, Hwang PH, Kingdom TT, Orlandi RR, Stammberger H, Han JK. Systematic review of topical vasoconstrictors in endoscopic sinus surgery. Laryngoscope. 2011;121(2):422–32.
- 53. Javer AR, Gheriani H, Mechor B, Flamer D, Genoway K, Yunker WK. Effect of intraoperative injection of 0.25% bupivacaine with 1:200,000 epinephrine on intraoperative blood loss in FESS. Am J Rhinol Allergy. 2009;23(4):437–41.
- Demiraran Y, Ozturk O, Guclu E, Iskender A, Ergin MH, Tokmak A. Vasoconstriction and analgesic efficacy of locally infiltrated levobupivacaine for nasal surgery. Anesth Analg. 2008;106(3): 1008–11.

Basic Physiology

Ira Hofer, Samuel DeMaria, Jr., and Adam I. Levine

Introduction

A firm foundation in human physiology is important to anesthesiologists, perhaps more than any other group of physicians. While a review of human physiology in its entirety is beyond the scope of this book, we will review those aspects of human physiology germane to the practice of otolaryngologists and anesthesiologists caring for otolaryngologic patients. Our discussion focuses predominantly on pulmonary and cardiovascular physiology and the autonomic regulation of these systems. A thorough understanding of endocrine physiology is also important for both the otolaryngologist and anesthesiologist. Pertinent endocrine physiology is discussed in Chap. 14.

At the most essential level, the primary function of the pulmonary and cardiovascular systems is to assure the delivery of oxygen to tissues and to remove various waste products created by oxygen consumption. This chapter will trace air, and hence oxygen, as it enters the body and makes its way to the tissues, thus providing an overview of the relevant airway, cardiac, and pulmonary physiology and the autonomic regulation that governs these interrelated systems.

Physiology of the Upper Airway

Air enters the body via the nose (nasal passages) or the mouth (oral cavity) (Fig. 4.1).

The nostrils are the smallest of the apertures and as a result, they account for as much as 50% of all airway resistance during quiet breathing. The nasal cavity provides filtration,

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warmth, and humidification. Air entrained through the nasal cavity is filtered of particulate matter greater than 4 mm and is humidified to a water saturation of 85% as it leaves the nasopharynx. The nasal cavity functions as an immunologic barrier, containing lymphocytes, eosinophils, mast cells, macrophages as well as many immunoglobulins to help manage the inhalation of infectious material. The oral cavity provides humidification as well, but due to its wider diameter and lower resistance is significantly less efficient in providing successful filtration than the nasal cavity.

The oral and nasal pharynxes meet in the posterior pharynx. Unlike the more proximal structures, the posterior pharynx has no rigid support structures. During inspiration, high velocity air moves in a turbulent pattern, creating negative pressure in the pharynx. Airway patency is maintained by the genioglossus, geniohyoid, sternohyoid, sternothyroid, and thyrohyoid muscles; however, if negative pressure overcomes these muscles, airway collapse can occur as predicted by Bernoulli's principle (namely, that as speed of the fluid increases, pressure decreases). The tendency for upper airway collapse is significantly increased by many factors associated with the anesthetized state including: the supine position which posteriorly displaces the mandible, neck flexion, and the loss of the airway muscle tone associated with anesthesia with and without the inclusion of neuromuscular blocking agents. Patients with obesity and an increased neck circumference, obstructive sleep apnea, obligate nasal breathers (e.g., neonates) and those patients with a pathologic narrowing of the pharynx are at increased risk for upper airway collapse. The tendency for airway collapse is mitigated by chemoreceptors activated by both hypercapnia and hypoxemia that preferentially stimulate the airway dilator muscles over the diaphragm resulting in a reduction of negative inspiratory pressure ^[1].

Anatomically the larynx is distal to the pharynx and is innervated by the superior laryngeal and recurrent laryngeal nerves. The larynx is divided into three anatomic regions, the supraglottis, the glottis, and the subglottic regions. The glottis contains the vocal cords that provide the ability to phonate and protect the tracheal inlet from aspirant material.

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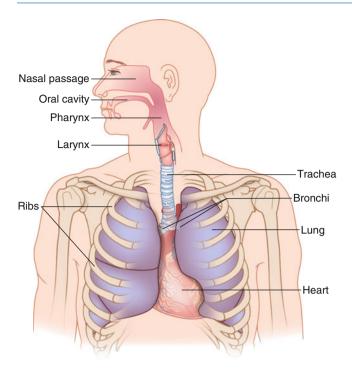


Fig. 4.1 The human respiratory system

Table 4.1	Stages of anesthesia
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Stage 1	Awake
Stage 2	Patients with excited and delirious activity. Notable for airway hyperreactivity
Stage 3	Surgical level. Includes muscle relaxation and a lack of reaction to surgical stimulus
Stage 4	Excessive sedation that may include cardiac or respiratory collapse and necessitates significant hemodynamic support

Upon irritation of the glottis, the vocal cords reflexively close thereby blocking entrance to the trachea and preventing aspiration. Ideally, this powerful reflex should be abolished during intubation to prevent vocal cord trauma. A deep, surgical plane of anesthesia (stage 3 of anesthesia), with or without the use of neuromuscular blocking agents, is used so that the patient can be safely intubated after the induction of anesthesia (Table 4.1); superior and recurrent laryngeal nerve blocks can be used for those patients requiring an awake intubation.

Patients are vulnerable to laryngospasm during light planes of anesthesia (stage 2 of anesthesia) when the laryngeal protective reflexes are exacerbated. In this case, irritants such as secretions, airway manipulation, or surgical stimulation can result in complete glottic occlusion requiring interventions such as positive pressure ventilation (PPV) or the administration of a muscle relaxant. The anesthetized state

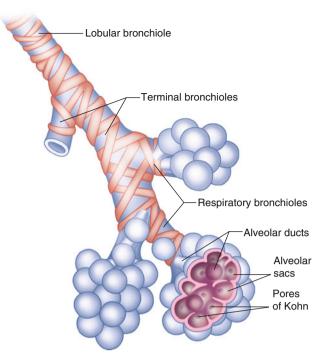


Fig. 4.2 Architecture of the unit of gas exchange in the lungs

makes patients more vulnerable to serious airway collapse and airway obstruction, therefore the placement of oral airways, supraglottic airways (such as the laryngeal mask airways (LMA)) and endotracheal airways (such as endotracheal tubes) are generally required.

Pulmonary Physiology

After leaving the upper airway, air enters the lungs, the principle organs of respiration, via the trachea, bronchi, and bronchioles (the tracheobronchial tree) (Fig. 4.2). The respiratory bronchioles and alveoli of the lung parenchyma provide the unit of gas exchange in the lung that facilitates the movement of oxygen and carbon dioxide into and out of the body (physiologic respiration). To clarify, *oxygenation* is the uptake and distribution of oxygen to cells, tissues, and vital organs, *ventilation* is the elimination of carbon dioxide, and *cellular respiration* is the cellular process whereby oxygen is consumed while carbon dioxide is produced during the ATP production of oxidative phosphorylation.

Lung Volumes

Prior to a discussion of pulmonary physiology it is useful to define some key lung volumes. Figure 4.3 demonstrates a

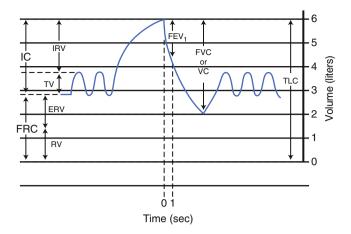


Fig. 4.3 Normal adult spirogram

normal spirogram as well as the volumes that are common for a healthy 70 kg adult. A regular non-forced breath contains the tidal volume (TV) and is roughly 5–7 ml/kg of body weight. A maximum inspiration will include the inspiratory reserve volume (IRV), which is about 40–45 ml/kg. A forced expiration will expire to the residual volume (RV), which is approximately 15 ml/kg and includes the expiratory reserve volume (ERV), which is also approximately 15 ml/kg (see Fig. 4.3).

Capacities are the sum of two or more volumes. There are four relevant capacities, including functional residual capacity (FRC), vital capacity (VC), total lung capacity (TLC), and closing capacity (CC). FRC is the volume in the lung at rest and is approximately 30-40 ml/kg of body weight and represents the oxygen reserve volume during apnea. Many factors can dramatically decrease FRC including positioning (e.g., supine and trendelenburg), patient comorbidities (e.g., obesity, ascites), anesthesia (e.g., the use of neuromuscular blocking agents), and surgical manipulations (e.g., abdominal insufflation and abdominal retractors). VC represents the volume achieved when taking a maximal inspiration after a maximal expiration. It is the sum of the ERV, TV, and IRV. TLC is the total volume that can be accommodated in the lung, VC plus the RV, and is approximately 80 ml/kg. CC is the lung volume at which dynamic compression of the airways begins to occur (i.e., the negative pressures associated with airflow begin to overcome the airway tone of the musculature in some of the smallest airways). This value is usually smaller than FRC in the young healthy upright individual. Although FRC is reduced by many parameters (see above), few parameters increase CC. These include extremes of age, pulmonary disease, and chronic tobacco use. Classically, CC equals FRC in the supine position at age 44 and the upright position at age 66. When CC exceeds FRC distal airways collapse at end-expiration leading to atelectasis.

Pulmonary Mechanics

Pulmonary mechanics, or the mechanisms by which the lungs successfully exchange air with the outside environment, are driven by pressure gradients. In the normal spontaneously breathing patient, the thoracic cavity expands due to contraction of the primary muscles of ventilation that include both the diaphragm and the external intercostal muscles of the chest wall, as well as the natural outward recoil of the thoracic cavity. This expansion causes a decrease in intrathoracic pressure relative to the atmosphere and the entrance of air. The relative changes in this pressure, as well as the resistance and elastance of the lungs, airway, and chest wall will control the size of the breath. The relationship between the changes in thoracic pressure and the resultant lung volume can be described using the equation of motion of the lung^[2]:

 $P_{\rm TP} = {\rm Flow} \times {\rm Resistance} + {\rm Volume} \times {\rm Elastance} + P_{\rm FRC}$

where $P_{\rm TP}$ is the trans-pulmonary pressure (or the pressure gradient between the pleura and the alveoli) and $P_{\rm FRC}$ is the pressure at the resting lung volume.

The normal distribution of air entering the lung can be described using "Zones of West" (Fig. 4.4)^[3].

In West Zone 1, which is traditionally described as the superior most zone, the pressure in the alveolus exceeds arterial and venous pressure. This zone is well ventilated but poorly perfused, resulting in physiologic dead space (DS). In zone 2 arterial pressure exceeds alveolar pressure, which exceeds venous pressure; perfusion and ventilation are well matched and optimized. In zone 3 the arterial and venous pressures exceed alveolar pressure resulting in well-perfused but poorly ventilated alveoli, or shunt.

Physiologically, hypoxic pulmonary vasoconstriction (HPV), whereby vasculature perfusing alveoli with low oxygen content constrict, functions to optimize ^[4] ventilation–perfusion (V/Q) matching by minimizing intrapulmonary shunt. This increase in vascular resistance promotes blood flow away from under-ventilated alveoli to ventilated alveoli, decreasing right to left shunt and improving V/Q matching

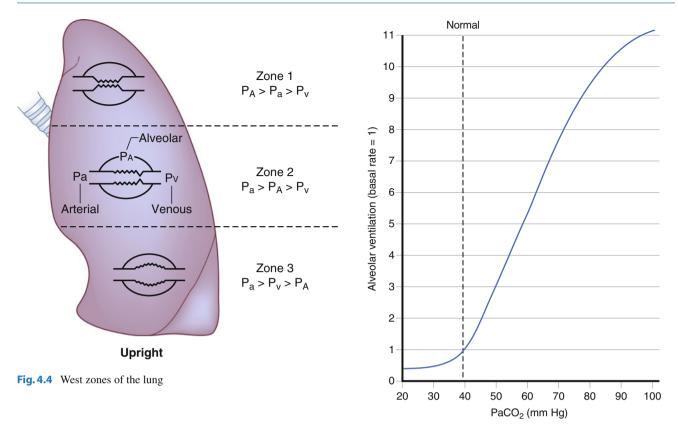


Fig. 4.5 CO₂ response curve

and oxygenation. In the supine position these zones continue to distribute in a superior to inferior direction however, because of the normal geometry of the lung, this distribution tends to decrease the amount of optimally matched zone 2 which increases the fraction of shunt.

Regulation of Ventilation

While pulmonary mechanics are determined by pressure gradients, the drive to ventilate is closely regulated by the body in response to small changes in carbon dioxide. Carbon dioxide is carried in the blood and buffered by carbonic anhydrase based on the equation:

$$H^+ + HCO_3^- \leftrightarrow H_2O + CO_2$$

Carbon dioxide readily disassociates into acid thereby decreasing blood and CSF pH. These changes trigger chemoreceptors in the medulla oblongata that act to increase minute respiration thereby decreasing CO_2 . In addition, receptors in the carotid and aortic bodies directly sense levels of carbon dioxide and act to increase MV. There are several

receptors that detect hypoxia; however, these are only activated at very low values of PaO_2 (i.e., $PaO_2 < 50 \text{ mmHg}$) and rarely come into play in normal physiologic function. The normal response to carbon dioxide levels can be seen in Fig. 4.5. There is a baseline level of CO₂ below which no respirations occurs (the apneic threshold), after which small increases in CO₂ levels lead to a dramatic increase in MV.

This system has important implications for physiologic homeostasis; acute derangements in ventilation, most often due to iatrogenic interventions or disease processes, can rapidly cause significant changes in pH. Intracellular enzymatic processes and protein function are carefully calibrated to a normal pH of 7.40 and significant deviation can result in the breakdown of normal cellular function and rapid clinical deterioration and eventually, death. Individuals with chronic derangements in respiration such as chronic obstructive pulmonary disease (COPD) have a permanent adjustment in the bicarbonate levels as regulated by the kidneys to maintain a relatively normal pH in the face of PaCO, levels significantly greater than 40 mm Hg. Individuals with severe derangements can have a CO₂ set point so high that their respiration is partially driven by hypoxemia. In this situation providing a high fraction inspired oxygen (FiO₂) can result in severe respiratory depression and even apnea.

Alterations in Normal Pulmonary Physiology

The pulmonary physiology described thus far has been representative of a healthy individual who is breathing spontaneously. Unfortunately, individuals undergoing anesthesia are often neither healthy nor breathing spontaneously, and as a result display a variety of pathophysiologic changes. These changes can be broken down into two broad groupings, obstructive and restrictive diseases, and are detailed below.

Obstructive Pulmonary Disease

Obstructive pulmonary diseases (such as asthma and COPD) have different pathological underpinnings but all ultimately result in increased airway resistance. Returning to our equation of motion of the lung,

 $P_{\rm TP} = ({\rm Flow} \times {\rm Resistance}) + ({\rm Volume} \times {\rm Elastance}) + P_{\rm FRC}$

obstructive diseases primarily result in derangements to the flow of air. Patients compensate with slower respiratory rates (minimizing the flow component) and larger TV in order to achieve the same MV. Over time, these changes result in distention of the lungs (or at least those lung units affected by the disease) and a loss of tissue elastance, further helping to minimize the pressure gradients. Initially, the overfilling of these lung units can result in an increase in zone 1, as the alveolar pressures rise in the ventilated portions of the lung. Zone 3 also increases since alveoli that are no longer being ventilated collapse. Over time normal alveolar geometry continues to break down, creating more areas of zone 1 (hyperinflated lung units) and zone 3 (atalectatic units) at the expense of zone 2. In addition, the loss of airway elastance increases airway resistance and diminishes the ability of the airways to remain patent even in the setting of higher flows. This forces obstructive patients to maintain higher flows that can cause airway collapse and exacerbate V/Q mismatch. These individuals can have increased CC above FRC, resulting in atelectatic airways even within normal TV ranges.

Restrictive Pulmonary Disease

Restrictive pulmonary disease results from an increase in tissue elastance and a decrease in compliance, causing ventilation with smaller lung volumes at higher respiratory rates, thus minimizing the volume component of the equation of motion. Because of the low compliance, alveolar pressure will be higher, increasing the percentage of zone 1. Longterm effects include the development of pulmonary hypertension in order to compensate for increased alveolar pressures; while this may serve to decrease the areas of zone 1 it can cause significant derangements in cardiac physiology as well as a decreased ability to provide diffusion between the alveolus and the blood, thereby impairing gas exchange.

Anesthesia and Surgery

Anesthetic Agents

Although opioids and opiates are the most potent, all intravenous and inhaled anesthetic agents are respiratory depressants and desensitize the central chemoreceptors to pH and CO_2 levels. These agents cause an increase in the apneic threshold. The pulmonary CO_2 response curve is thus shifted to the right and its slope is reduced or attenuated. When combined, these agents have a synergistic effect on the ventilatory drive. NMB agents, while not having an effect on the ventilator drive per se, will prevent muscle contraction and hence all spontaneous breathing will cease. These effects are discussed in detail in Chap. 3.

Positioning

Positioning can have a significant effect on pulmonary physiology. Placement of a patient in the supine and trendelenburg positions can dramatically decrease FRC below CC leading to atelectatic airways, elevated pulmonary pressures, R to L shunt and impaired gas exchange, leading to hypoxemia. These changes are exacerbated by old age, obesity, preexisting abdominal processes, and preexisting pulmonary disease, potentially resulting in difficulty with ventilation and oxygenation even prior to the induction of anesthesia.

Upon induction of anesthesia, and especially after muscle paralysis with NMB agents, muscle tone is decreased, abdominal contents move cephalad, mediastinal structures move posteriorly and lung volumes are significantly diminished; FRC may decrease as much as 40% during this time. This reduction of FRC decreases apneic oxygen reserves and accelerates the rate of oxygen desaturation during apnea. The decrease of FRC into CC further increases atelectasis, increases R to L shunt, decreases oxygenation, and makes PPV more difficult ^[5]. Intraoperatively, the trendelenburg position will further decrease lung volumes, specifically FRC, which will further increase atelectasis and R to L shunt, and decrease oxygenation. PPV may become even more difficult, thus increasing intrathoracic pressures further and even leading to barotraumas in rare cases. These changes can be partially overcome by using positive end expiratory pressure (PEEP) that will maintain some pressure in the airway and help prevent atelectasis.

Mechanical Ventilation

Normal respiratory mechanics are certainly altered with the use of PPV. Spontaneously breathing patients expand their thoracic cavity resulting in negative pressure and the influx of air. However, most anesthetic agents suppress the respiratory drive often to the point of apnea. In addition, the use of neuromuscular blocking agents eliminates the ability of the respiratory muscles to function. These changes often, but not always, necessitate mechanical ventilation. In the case of PPV, air is forced into the lungs thereby increasing intrathoracic pressure which decreases venous return and thus preload to the heart, decreasing cardiac output (CO). Because mechanical ventilation overrides the body's normal ability to regulate its own ventilation, careful attention must be paid to ensure that optimal V/Q matching is maintained, barotrauma is minimized and unintentional hyper or hypoventilation does not occur. Significant effects on CO₂ and pH can be caused by over or under zealous mechanical ventilation.

V/Q Matching and the Monitoring of Pulmonary Physiology

In order to successfully mechanically ventilate a patient, the anesthesiologist must regularly attempt to quantify both DS and shunt and adjust the respiratory parameters accordingly. This quantification may be accomplished using the arterial blood gas (ABG). The pH allows for the determination of significant acidemia or alkalemia; the PCO₂ gives a direct measurement of the carbon dioxide content of a given specimen which allows one to quantify deadspace ventilation; and the PO₂ (in conjunction with the hematocrit) gives the measurement of the oxygen content of the specimen and may help quantify shunt. The combined use of these three parameters allows for the direct quantification of DS and shunt and, combined with a thorough understanding of respiratory physiology, allows for optimization of V/Q matching.

In terms of ventilation and perfusion (V/Q), DS represents areas of the lung and tracheobronchial tree that are ventilated, but not perfused and therefore do not contribute to carbon dioxide elimination (ventilation); V/Q approaches infinity in this situation. Clinically there would be a large difference between measured PCO₂ and observed EtCO₂ (PCO₂-EtCO₂ gradient). Total DS is the sum of anatomic (tracheal, bronchus, terminal bronchus) DS and physiologic (areas of the lung that are poorly perfused yet ventilated) DS. Anatomic DS contributes to the majority of DS and typically represents 1/3 of a normal TV. In situations where DS exceeds 1/3 TV, physiologic DS (zone 1) can be assumed to have increased due to low perfusion states or over ventilation.

The formula for calculating DS is:

I

Dead Space
$$(V_d)$$
 = Tidal Volume (V_t)
×[(PaCO₂ – EtCO₂)/PaCO₂]

In terms of V/Q, shunt represents areas of the lung that are perfused, but not ventilated. V/Q approaches zero in this situation. Venous blood leaves the lung without being oxygenated or arterialized and enters the arterial circulation. This results in the dilution of oxygenated arterial blood with oxygen depleted venous blood. This is the classic right (venous) to left (arterial) shunt and is represented by a large difference in the alveoli-arterial oxygen pressures (A–a gradient). Shunt contributes to poor oxygenation but, due to carbon dioxide's high diffusibility, does not usually effect ventilation.

The formula for calculating shunt is:

 $Shunt = \frac{pulmonary}{pulmonary} = \frac{arterial}{oxygen content} = \frac{oxygen content}{pulmonary} = \frac{oxygen}{venous}$ end capillary = oxygen oxygen content = content

Because this equation requires both a mixed venous oxygen saturation (from a pulmonary artery blood sample) and arterial oxygen saturation, shunt is more often evaluated by the A–a gradient. The formula for calculating the alveolar arterial gradient is:

which can usually be simplified as:

$$=$$
 [FiO₂ × (760 - 47) - PaCO₂ / 0.8] - PaO₂

High gradients imply large sections of the pulmonary vasculature that do not encounter ventilated alveoli and are thus shunted from right to left.

Cardiac Physiology

While the lungs serve to take in oxygen from the outside and oxygenate the blood, it is the role of the cardiovascular system to deliver the oxygenated blood to the tissues, and carry away waste products of oxygen consumption. The cardiovascular system consists of the heart which propels blood through the body (CO) and the arterial, capillary, and venous systems which profuse the various tissues. The autonomic nervous system functions to regulate the function and efficiency of the cardiovascular system that is also under the influence of a number of factors including the patient's comorbidities and iatrogenic agents.

The primary metric used to measure the level of perfusion is the mean arterial pressure (MAP), which provides a useful starting point for its study. MAP has two components, systolic and diastolic, with the former providing 1/3 of the total value and the latter 2/3 of the total. The difference between the systolic and diastolic blood pressure (i.e., the pulse pressure) is a result of the stroke volume of the heart. More importantly for understanding the underlying physiology, MAP can be defined as:

 $MAP = cardiac output (CO) \times systemic vascular resistance (SVR).$

By understanding each of these various components, one can understand the drivers of the cardiovascular system and how it can be manipulated. The CO can further be defined as:

 $CO = stroke volume (SV) \times heart rate (HR)$

Thus

$$MAP = SV \times HR \times SVR$$

The SV is the amount of blood ejected per heartbeat and is influenced by preload, afterload, and contractility. Therefore, a complete understanding of the drivers of MAP necessitates an understanding of five factors: preload, afterload, contractility, heart rate, and rhythm.

Preload

Preload is defined as the volume in the chambers of the heart immediately prior to its contraction, (i.e. at the end of diastole). Since physicians generally are concerned with the heart's ability to perfuse the systemic tissues, this is usually

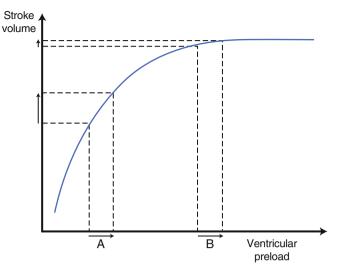


Fig. 4.6 The Frank–Starling curve. "A" shows the effect of a fluid bolus on stroke volume in a normally filled heart and "B" in an overfilled heart

described in terms of the left ventricle. The effect of preload on contractility is based on the Frank–Starling curve (Fig. 4.6) ^[6,7]. Initially, small increases in end diastolic volume produce large changes in stroke volume and contractility (and hence CO); as the amount of preload increases the ventricle becomes "over" stretched and additional preload will no longer increase and may actually decrease the CO (the plateau of the curve). Optimization of CO entails attempting to place the preload near the inflection point of the curve, thereby maximizing the amount of blood that can be delivered with each beat.

Left ventricular preload itself is influenced by a variety of factors including venous return to the heart, patient positioning (specifically head up or down positions), anesthesia with vasodilatation, PPV, PEEP, pulmonary hypertension, valvular heart disease, and ventricular compliance. In the normal individual without cardiac or pulmonary disease, increased venous return will correspond with increased preload and thus an increased CO. However, impediments to forward flow between the right atrium and the left ventricle can have profound effects on the preload.

In the case of pulmonary hypertension, increased pressures of the pulmonary vascular bed will act as an impediment to left heart filling, requiring increasing right ventricular pressure (and possibly right atrial pressures) to promote forward flow. The specific impact of valvular heart disease on preload depends upon the type of underlying disease; but in general most lesions will result in decreased left ventricular filling for a given volume of blood return. The presence of these derangements may cause considerable congestion proximal to the lesion potentially causing significant alterations in pulmonary physiology, as well as systemic vascular congestion.

Afterload

Afterload is the force that the left ventricle must pump against in order to profuse the body. In most clinical situations the afterload is determined by the SVR. High SVRs result in an increased afterload and potentially decreased CO; however, SVR is also a major determinant of venous return and excessively low SVRs can result in decreased preload and thus a lower CO.

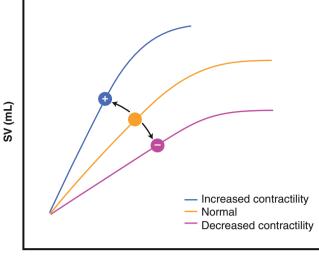
In cases of aortic valve stenosis afterload is not largely equated with SVR. In this situation afterload is predominately determined by the resistance caused by the decreased valve area. In a patient with aortic stenosis maintenance of preload is critical because small decreases in contractility via the frank–starling mechanism can result in the inability of the heart to pump blood past the aortic valve and thereby result in cardiac collapse. Blood viscosity or lack thereof due to the patient's hematocrit can also affect the afterload of the heart.

Contractility

Contractility, or inotropy is the intrinsic ability of the heart to contract independent of preload and afterload. As previously described, contractility is primarily determined by the frank–starling mechanism and the ventricular volume. However, this curve can be shifted as a result of extrinsic factors. In the normal individual catecholamines and inotropic drugs will increase contractility and shift the curve upward. In the presence of scared, stunned (dysfunction which persists for a variable period of time) or ischemic myocardium, contractility will decrease and the curve will be shifted downward (Fig. 4.7).

Heart Rate and Rhythm

The relationship between heart rate and CO is fairly straight forward; increases in HR will increase CO. This is true for most of the normal range of heart rates; however at the extremes, there are relevant deviations. The majority of the cardiac cycle is spent in diastole (approximately 2/3) when the ventricle fills passively; tachycardia decreases time in diastole more than systole. In extreme situations a pathologic tachycardia can result in a dramatic decrease in preload due to an inadequate time for ventricular filling. In this case, increases in heart rate may result in a decreased CO. Slowing the heart rate may prove to be beneficial, but care must be taken to determine the cause of the tachycardia



LVEDP (mm Hg)

Fig. 4.7 Effect of changes in contractility on the Frank-Starling curve

since arbitrarily slowing the rate could have devastating effects on the cardiovascular system (e.g., profound hypotension).

Arrhythmias when atrial and ventricular function are not matched may also result in an under filled ventricle. For example during atrial fibrillation, or premature ventricular contractions the ventricular contraction may be coupled with a relatively empty ventricle resulting in a lower CO despite a relatively normal or high heart rate. In this situation, the arrhythmia must be corrected in order to return CO to a normal state.

Although cardiac wall tension increases oxygen demand greater than any other parameter, increased heart rate places the myocardium at the highest risk for ischemia due to an imbalance of oxygen demand and oxygen delivery. Coronary filing and myocardial perfusion occurs during diastole. As heart rate increases, time in diastole decreases, thus reducing coronary perfusion time. As discussed below, instances of even mild tachycardia can lead to myocardial ischemia, especially in the event of coronary artery disease, that may need correction.

Cardiac Blood Supply

The heart is supplied by the coronary arteries which leave the aorta just distal to the aortic valve. Because of their position near the valve leaflets, the coronary arteries fill during diastole and have a pressure equal to the diastolic blood pressure. Opposing this pressure is the pressure in the left ventricle that peaks at the end of diastole when the volume is greatest. Thus, we can define coronary profusion pressure as:

Coronary profusion pressure = diastolic blood pressure - LV end diastolic pressure

Factors that decrease the former (hypovolemia, hypotension) or increase the latter (excessive blood volume, a noncompliant ventricle) can contribute to myocardial ischemia. Cardiac oxygen demand is significantly affected by heart rate, thus maximization of perfusion and minimization of cardiac demand is best accomplished with a relatively slow heart rate, and filling pressures which end near the inflection point of the Frank Starling curve.

Anesthetic and Surgical Effects on the Cardiovascular System

Most anesthetic drugs have direct and indirect effects on the cardiovascular system. In general, anesthetic agents have depressant effects on both vascular tone and contractility and therefore affect CO. Additionally, anesthetic agents will indirectly affect the cardiovascular system by their effect on the autonomic nervous system by augmenting or suppressing the sympathetic or parasympathetic tone. Like the effects on the pulmonary system and ventilatory drive, the deleterious effects of anesthetic agents on the cardiovascular system tend to be synergistic. A variety of anesthetic effects on the cardiovascular physiology will be discussed in subsequent chapters throughout the book. Surgical interventions will also directly and indirectly affect the cardiovascular system including the activation of both the sympathetic and parasympathetic nervous systems.

Oxygen Delivery and Consumption

While the cardiovascular system is responsible for delivering blood to tissues and vital organs, it is the blood that is responsible for transport of the oxygen and waste products. In the lungs, oxygen diffuses through the thin alveolar wall and is absorbed by blood in the capillaries where it is then carried to the tissues. The regulation of this transport is tightly controlled and centers upon hemoglobin.

Oxygen Carrying Capacity

Oxygen in the blood is carried in two forms; oxygen bound to hemoglobin and oxygen dissolved in plasma. The total

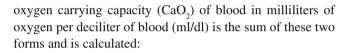


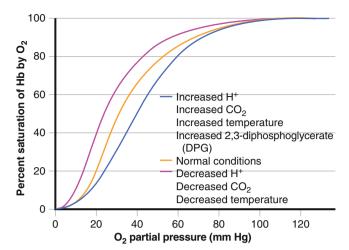
Fig. 4.8 The oxygen-hemoglobin dissociation curve

$$CaO_{2} = (SaO_{2} \times Hb \times 1.34) + 0.003(PaO_{2})$$

where the SaO₂ is the oxygen saturation of hemoglobin, Hb is the amount of hemoglobin (g/dl), and PaO₂ (mmHg) is the partial pressure of oxygen in the blood. The factor 1.34 is the amount of oxygen in milliliters that binds to a gram of hemoglobin (ml/g) and 0.003 is the amount of oxygen that dissolves in a deciliter of blood per mmHg of oxygen. The first part of the equation represents the oxygen bound to hemoglobin and the latter represents the amount of oxygen dissolved in the plasma. It should be apparent that once the hemoglobin is fully saturated (SaO₂ of 100%, usually a PO₂ of approximately 100) further increases in PaO₂ with higher FiO₂ only increases the dissolved portion of oxygen that contributes little to overall oxygen delivery under normal situations. At virtually all hemoglobin levels the bound portion of oxygen is the more clinically significant component.

Understanding oxygen delivery requires understanding the relationship between oxygen tension and hemoglobin saturation as defined by the oxygen-hemoglobin dissociation curve (Fig. 4.8).

The curve, classically described as a sigmoidal "S" shape, demonstrates hemoglobin's affinity for oxygen and oxygen's influence on this affinity. At low PO_2 levels most of the hemoglobin is unbound and binding of oxygen is slow. As oxygen binds, the hemoglobin molecule's affinity for oxygen dramatically increases, thus facilitating further oxygen binding represented by the steep portion of the dissociation curve. Eventually, as virtually all of the hemoglobin is bound with oxygen or saturated, further increases in blood's carrying capacity occurs only because of an increase in dissolved oxygen and the second plateau is reached.



Delivery of oxygen to the tissues is controlled by changes in hemoglobin's affinity for oxygen (left and right shifts in the curve) as a result of various factors. A "normal" curve is based on a point known as the p50; that PaO₂ that results in a saturation of 50%. Different hemoglobins have different P50s. Adult hemoglobin has a P50 of 26.8 mmHg whereas the P50 of fetal hemoglobin, which has a higher affinity for oxygen compared to adult hemoglobin, is 19 mmHg. A rightward shift of the curve results in a decrease in affinity. This facilitates oxygen unloading to tissues. Factors that decrease hemoglobin's affinity for oxygen include, an increase in PCO₂ and a decrease in pH, an increase in temperature and an increase in 2,3-diphosphoglycerate (DPG) levels. During increased tissue metabolism CO₂ is produced, temperature is generated and 2,3 DPG is created as a breakdown product of glucose metabolism. In this situation the hemoglobin dissociation curve is shifted to the right, hemoglobin's affinity for oxygen is decreased and oxygen supply to tissues is increased. The opposite conditions increase hemoglobin's affinity for oxygen thus producing a leftward shift in the curve and preferentially promoting oxygen-hemoglobin binding. Such a situation occurs in the lungs and promotes oxygen binding to hemoglobin.

The total oxygen delivery to tissues and vital organs can be determined by multiplying oxygen carrying capacity of arterial blood by blood flow per minute or CO.

Total oxygen delivery = $CaO_2 \times CO$

 $[(SaO_2 \times Hb \times 1.34) + 0.003(PaO_2)] \times (SV \times HR)$

Therefore, in situations when oxygen delivery is inadequate to meet demand, treatment may include increasing oxygen carrying capacity [improving oxygen saturation (the addition of PEEP or increasing FiO_2)] or red blood cell (RBC) transfusion) or improving CO (optimizing heart rate, rhythm, preload, contractility, and or afterload).

Oxygen Consumption

As already discussed, oxygen delivery is dependent on the CO, hemoglobin concentration and oxygen saturation; in contrast, oxygen consumption is a more complicated process that depends on a variety of factors. Oxygen consumption is based on the total cellular needs for oxidative metabolism, which is dependent on factors including body temperature, demands for tissue work, local and systemic inflammation, and genetic factors. A normal 70 kg individual at rest will consume roughly 250 ml of oxygen per minute in order to meet their metabolic requirements (i.e., 3-4 ml O₂ per kg per minute). Hyperthermia, surgical stress, infection, postoperative shivering and other catabolic states (hyperthyroidism)

Table 4.2 Distribution of blood flow

Organ	Percent of cardiac output		
Liver	25%		
Brain	15%		
Kidneys	20%		

can raise oxygen consumption dramatically; which will in turn increase CO₂ production and ventilatory requirements.

In the normal state, blood flow (and similarly oxygen consumption) is distributed as demonstrated in Table 4.2 with the vast majority of blood perfusing the vessel rich organs consisting of the heart, lungs, kidneys, liver, and brain. In situations where oxygen delivery is insufficient to meet demand, these percentages change such that vasoconstriction shunts blood away from the peripheral tissues and preferentially towards the vessel rich group and vital organs (brain and heart). The Fick Equation is used to quantify oxygen consumption and metabolic needs.

Oxygen consumption = $CO \times (C_a - C_v)$

where C_a is the oxygen content of the arterial blood and C_v is the oxygen content of the venous blood. Intraoperatively, this data can be used to help assess perfusion of vital organs. The use of a mixed venous saturation is crucial in this assessment and is beyond the scope of this chapter.

The Autonomic Nervous System and its Physiology

The autonomic nervous system provides the mechanism by which the body controls various organ system functions. The autonomic nervous system can be divided into two distinct parts, the sympathetic and parasympathetic branches. The sympathetic system is often named the "fight or flight" branch and is activated in response to stress inducing responses such as tachycardia, hypertension, hyperthermia, pupil dilatation, diaphoresis, and tachypnea. The parasympathetic system will invoke the opposite bodily responses.

The structure of both systems has many similarities. Autonomic control of temperature and blood pressure begins in the hypothalamic nuclei of the brain while the medulla and pons are the source of hemodynamic and ventilatory control. The spinal cord gives rise to preganglionic mylenated fibers that exit at various levels and synapse with postganglionic fibers that innervate the various organs and effect a response. The location of both the origin and synapse of these fibers differs between the systems, as does the neurotransmitter involved.

The Sympathetic System

The preganglionic fibers of the sympathetic system arise in the thoracolumbar region of the spinal cord from T1 to L3. These myelinated nerves exit the cord and enter the sympathetic chain that consists of 22 paired ganglia. Most of the fibers then synapse with the postganglionic neurons using acetylcholine as a neurotransmitter. Some fibers exit the chain without synapsing in the sympathetic chain, but rather at other more distant ganglia such as the celiac or mesenteric ganglia that are closer to the end organs. The postganglionic fibers of the sympathetic nervous system then synapse with the end organs and release the neurotransmitters norepinephrine or dopamine. Norepinephrine stimulates alpha and beta receptors while dopamine stimulated the various classes of the dopamine receptor (D1–D5).

Alpha receptors are responsible for the constriction of blood vessels and promote hypertension while beta receptors serve to increase heart rate and cardiac ionotropy as well as vasculature dilation. Dopamine has a more variable physiologic effect, but in general results in decreased vascular tone at low doses, increasing renal blood flow and renal filtration, and increased ionotropy. Vasoconstriction occurs at increased levels.

In general, the sympathetic activation results in tachycardia, increased cardiac ionotropy, increased venous return, increased CO, and increased afterload. These effects, as well as effects on the other end organs, will increase metabolic demands and oxygen consumption and, via the effects on carbon dioxide concentration, increase ventilatory requirements.

The Parasympathetic System

The myelinated preganglionic neurons of the parasympathetic nervous system exit the spinal cord from the cranial and sacral regions and then travel much closer to the end organs where they synapse with the postganglionic neurons releasing acetylcholine as the neurotransmitter. The postganglionic neurons themselves also release acetylcholine as the neurotransmitter and synapse with two types of receptors; muscarinic and nicotinic receptors. The nicotinic receptors are found at the neuromuscular junction as well as in the ganglia of the sympathetic nervous system, while the muscarinic receptors are found at the other sites of parasympathetic action including the heart, GI sphincters and the eyes where they cause decreased ionotropy, bradycardia, sphincter relaxation, and pupil constriction.

Physiologic Monitoring and Cardiovascular and Pulmonary Physiology

A detailed discussion concerning physiologic monitoring principles follows in Chap. 5. Suffice it to say that in order to use physiologic monitoring affectively to optimize homeostasis, physicians caring for the otolaryngologic patient need an in-depth understanding of the principles that govern pulmonary and cardiovascular physiology.

Conclusion

The cornerstone of the practice of anesthesiology is to monitor and manipulate physiologic parameters to promote patient safety during significant anesthetic and surgical derangements. Otolaryngologists can serve their patients better by having a working knowledge of cardiovascular and pulmonary physiology, understand how surgical interventions impact normal function, and understand how anesthesiologists monitor and manage these derangements to improve patient outcomes.

As demonstrated, a thorough understanding of physiology is imperative to the successful practice of anesthesia. It is through mastery of these concepts as well as the effects of various interventions, be they pharmacologic or otherwise, that the anesthesiologist can successfully bring the patient safely through a physiologically stressful surgical experience.

References

- 1. Rajagopal MR, Paul J. Applied anatomy and physiology of the airway and breathing. Indian J Anaesth. 2005;49(4):251–6.
- West J. In: West J, editor. Respiratory physiology: the essentials. Philadelphia: Lippincott Williams and Wilkins; 2008.
- West JB, Dollery CT, Naimark A. Distribution of blood flow in isolated lung; relation to vascular and alveolar pressures. J Appl Physiol. 1964;19:713–24.
- Evans AM, Hardie DG, Peers C, Mahmoud A. Hypoxic pulmonary vasoconstriction: mechanisms of oxygen-sensing. Curr Opin Anaesthesiol. 2011;24(1):13–20 [Review].
- Gunnarsson L, Tokics L, Gustavsson H, et al. Influence of age on atelectasis formation and gas exchange impairment during general anesthesia. Br J Anaesth. 1991;66:423–32.
- 6. Frank O. Zur Dynamik des Herzmuskels. Z Biol. 1895;32:370.
- 7. Starling EH. Linacre lecture on the law of the heart. London: Longmans, Green, & Company; 1918.

Physiologic Monitoring

Jaime B. Hyman and David L. Reich

Introduction

Perioperative patient monitoring has changed drastically since the specialty of anesthesiology emerged in the nineteenth century. At its most basic level, monitoring involved palpating pulses, observing chest expansion, and inspecting skin coloring for cyanosis. Technological advances now provide the anesthesiologist and surgeon with a wealth of information not available by direct observation alone. In addition, alarms for alerting clinicians to parameters outside of normal ranges provide an added safety feature. Documenting intraoperative events and recording the patient's vital signs are also a major role of the anesthesiologist. The emergence of automated electronic medical records has freed the anesthesiologist to devote attention to other critical aspects of patient care intraoperatively. Technically complicated monitors are not infallible and are prone to failure and interference; the importance of a vigilant clinician who is constantly assessing the overall clinical status cannot be overemphasized. Information from direct patient observation, physiologic monitors, and the surgical field must be integrated to ensure hemodynamic and respiratory stability, adequate anesthesia and analgesia, and optimal operating conditions in the face of rapidly changing situations that are common during surgery. Effectively monitoring patients undergoing otolaryngologic procedures is also complicated, due to the proximity of the surgical site to the head, neck, and thorax; visual inspection is generally difficult, auscultation impractical, and noninvasive monitoring interference and failure likely. In light of this, otolaryngology procedures are associated with frequent airway compromise, wide variability in the level of noxious stimulation, and sudden reflex-mediated hemodynamic derangements, making close patient monitoring especially important.

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Guidelines for basic anesthetic monitoring were adopted by the American Society of Anesthesiologists (ASA) that mandated the minimum standard of care necessary for all anesthetics. These include continuous pulse oximetry, capnography, inspiratory airway oxygen analysis, ventilator disconnect alarms, body temperature, continuous electrocardiogram (ECG), and measurement of systemic blood pressure at least every 5 min ^[1]. Depending on the patient's medical history and the extent of surgery, this basic monitoring may be expanded to include more intensive (and often more invasive) monitors. This chapter will first discuss the standard ASA monitors, followed by an explanation of the benefits and indications for additional advanced monitoring consideration for various categories of otolaryngology procedures.

Basic Monitors

Electrocardiogram

Continuous ECG is used perioperatively to evaluate heart rate and rhythm and to detect myocardial ischemia, conduction abnormalities, pacemaker malfunctions, and occasionally electrolyte abnormalities. Intraoperatively, lead II and lead V5 are monitored via the placement of five surface contacts; four limb leads and the V5 precordial lead (5th intercostal space anterior axillary line). The electrical axis of lead II (from the right arm to left leg) parallels the electrical axis of the atria, resulting in the largest P wave voltages and enhancing the identification of atrial arrhythmias. It will also permit the detection of right ventricular (inferior wall) ischemia. Lead V5 is sensitive for detecting left ventricular (anterior and lateral wall) ischemia. The combination of leads II and V5 may detect as much as 95% of intraoperative myocardial ischemic events.

ECG evidence of ischemia includes ST-segment depression or elevation. ST segment depression is significant if it exceeds 0.1 mV from baseline (generally calibrated as 1 mm on printouts), is horizontal or downsloping in configuration, or is in

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ECG Patterns Indicative of Myocardial Ischemia



Fig. 5.1 ECG criteria for myocardial ischemia consist of ST depression of 0.1 mV or elevation of 0.2 mV, measured at 60–80 ms following the J point. Downsloping or horizontal ST-segments are more diagnostic of ischemia, as upsloping patterns may be a normal variant, especially

with tachycardia. Some authorities set a criterion of 0.2 mV depression to define ischemia when the ST-segment is upsloping. From Goldschlager N. Use of the treadmill test in the diagnosis of coronary artery disease in patients with chest pain. Ann Intern Med 1982;97:383–8

conjunction with T wave inversion. Significant ST-segment elevation associated with ischemia is that exceeding 0.2 mV (2 mm on printouts) (see Fig. 5.1)^[2]. In order to accurately interpret ECG monitoring for ischemia the filtering settings of the monitor must be considered. Filtering that suppresses electrocautery interference may adversely affect the detection of myocardial ischemia. Many monitors permit ECG filtering to be set according to the clinical circumstances. It is also important to confirm that the ST-segment monitoring algorithm has correctly identified both the isoelectric point (usually the P-R interval or the T-P interval) and the j-point (point of inflection marking the transition from the S wave to the ST-segment). The ST-segment's elevation or depression is generally measured either 60 or 80 ms after the j-point. Because of the high prevalence of coronary artery disease in the otolaryngology patient (especially those undergoing oropharyngeal cancer surgeries), it is important for the clinician to understand the factors that influence the ability to accurately use the ECG and be vigilant to detect ST segment abnormalities consistent with intraoperative ischemia.

Electrocautery and motion artifacts can mimic arrhythmias or obscure the detection of the true heart rate or rhythm. Procedures involving the head and neck make the ability to recognize rhythms associated with overriding parasympathetic tone from vagal stimulation (e.g., oral, ocular, and carotid artery manipulations) including sinus bradycardia, junctional rhythms, and asystole particularly important. The additional pulse data derived from the pulse oximetry plethysmograph or arterial pressure waveform (discussed in following sections) may be used to confirm the true heart rate in these instances.

Arterial Blood Pressure

Blood pressure is one of the most important and fortunately easiest cardiovascular variables to measure, though it gives only indirect information about the patient's cardiovascular status. A change in blood pressure may indicate a change in cardiac output, systemic vascular resistance, or both. The time-weighted average of arterial pressure is the mean arterial pressure (MAP) and can be calculated using the following formula:

MAP =
$$\frac{\text{SBP} + 2(\text{DBP})}{3}$$
 or DBP + 1/3 (SBP - DBP)

SBP=systolic blood pressure and DBP=diastolic blood pressure.

With the exception of the left ventricle, where diastolic blood pressure is the most important determinant of tissue perfusion, the MAP is the most useful parameter for assessing organ perfusion in the rest of the body. Blood pressure detection is greatly affected by the anatomic site of measurement. The more distal the site of measurement the more distorted the arterial waveform becomes. Even though the systolic peak increases while the diastolic trough decreases, the MAP measured in peripheral arteries should be consistent and similar to the central aortic pressure unless arterial disease is present.

Noninvasive Blood Pressure Monitoring

Before automated noninvasive blood pressure monitoring was possible, anesthesiologists used a sphygmomanometer and auscultation (for Korotkoff sounds) or palpation techniques. This was technically challenging and in many cases impractical. In the mid-1980s automated, noninvasive techniques were introduced into the practice of clinical anesthesia. Whether manual or automatic, a noninvasive technique for measuring blood pressure involves the placement of an inflatable cuff around a limb, usually the upper arm. An appropriately sized cuff has a width that is approximately 40% the circumference of the arm. A cuff that is too small results in a spuriously high blood pressure reading, and conversely a cuff that is too large will result in a falsely low reading ^[3]. Perioperatively, the oscillometric method is used for automated blood pressure

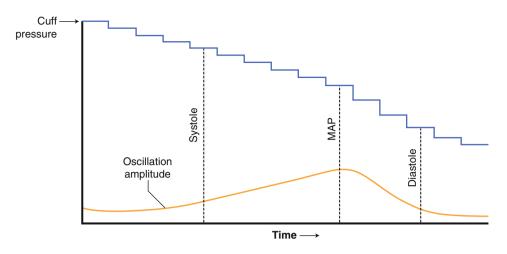


Fig. 5.2 Oscillometric determination of blood pressure. Note that maximal oscillation occurs at the point of the mean arterial pressure

measurements most commonly. This method is based on the oscillations in cuff pressure cause by arterial pulsations. These oscillations are minimal when the cuff is inflated above systolic pressure. When the cuff pressure is decreased to systolic pressure the oscillations markedly increase. Maximal oscillations correlate with MAP, after which the oscillations decrease as the cuff pressure further diminishes (see Fig. 5.2). Systolic and diastolic blood pressures are both derived by an algorithm; therefore, MAP is the most accurate value when the oscillometric method is used. Generally, automated noninvasive devices initially inflate to a predetermined pressure; therefore, the initiation of this device may be perceived by patients as uncomfortable or even painful. If inflated above the patient's systolic blood pressure, oscillations will not be detected and the cuff will systematically deflate at a rate consistent with the patient's heart rate and a systolic, mean and diastolic blood pressure will be recorded. If oscillations are detected after the initial inflation, the device will further inflate until oscillations are no longer sensed. Once the patient's blood pressure is determined, subsequent inflations will be based on the determined systolic blood pressure and therefore less uncomfortable for the patient. Limitations to this method include the inability or difficulty to detect or determine timely information during abrupt extreme changes in blood pressure that may be common during some surgical procedures, sensitivity to motion artifact, compression from surgical personnel, difficulties in detection and delays in data collection during bradycardia and dysrhythmias, difficulties in the morbidly obese, and rare cuff-related trauma or impaired distal limb perfusion from excessively frequent cycling.

Intraarterial Blood Pressure Measurement

Indications for invasive arterial blood pressure monitoring are listed in Table 5.1. Briefly, insertion of an arterial line should be considered for patients with significant cardiovascular or

Table 5.1

Indications for invasive arterial blood pressure monitoring

Major surgical procedures involving large fluid shifts or blood loss

Patients with recent myocardial infarctions, unstable angina, or severe coronary artery disease

Patients with decreased left ventricular function or significant valvular heart disease

Patients with right heart failure, severe COPD, pulmonary hypertension, or pulmonary embolism

Planned pharmacologic or surgical cardiovascular manipulation, including deliberate hypotension

Repeated blood sampling

Failure of noninvasive arterial blood pressure measurement (e.g., morbid obesity)

Supplemental diagnostic information from the arterial waveform (e.g., respiratory variation in amplitude suggesting hypovolemia)

respiratory disease or for surgical procedures that are associated with large blood loss, fluid shifts, or hemodynamic variation. The advantage of continuous, beat-to-beat blood pressure monitoring is that it facilitates rapid interventions to correct hemodynamic derangements. Components of an invasive pressure measurement system include an indwelling arterial catheter (usually 20 gauge or smaller), fluid-filled pressure tubing, stopcocks, a continuous flushing device, a transducer, and analysis and display systems. The transducer contains a diaphragm that is distorted by the arterial pressure wave that is transmitted via fluid-filled tubing. The mechanical energy applied to the diaphragm varies the resistance in one limb of a Wheatstone bridge circuit such that the voltage output across the circuit is proportional to the pressure applied.

Key requirements for accurate transducer reflection of arterial pressure are to zero the transducer to atmospheric pressure and to place the level of the transducer at an appropriate height relative to the patient. In a supine patient, the transducer is generally placed level with the patient's

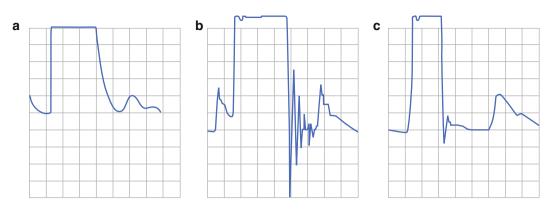


Fig. 5.3 Fast-flush test. (a) Overdamped system. (b) Underdamped system. (c) Optimal damping. From Irwin RS, Rippe JM: Manual of intensive care medicine, 4th edition. Philadelphia: Lippincott Williams & Wilkins, 2005

midaxillary line so as to measure pressure at the level of the heart and brain. For every 13 cm in height that the transducer is moved up or down there is a corresponding (and inverse) error of 10 mmHg on the monitored pressure reading. A discussion regarding the issues of pressure monitoring in "beach-chair" or sitting position cases is included below.

Inaccurate pressure measurement can also occur due to resonance (natural frequency characteristics) and/or damping of the transducer–tubing system^[4]. The peaks and troughs of an arterial pressure waveform will be amplified by resonance if the transducer–tubing–catheter system has a natural frequency that is too low. This problem occurs with longer pressure tubing (which may be required in otolaryngology cases due to the need for distance from the surgical field) as the natural frequency of the tubing approaches the frequencies that comprise those of the arterial pressure waveform (typically less than 20 Hz)^[5]. Damping has the opposite effect. It is the tendency of air bubbles, compliant tubing, and friction to absorb energy and decrease the amplitude of peaks and troughs in the waveform.

The optimal degree of damping counterbalances resonance. The relative balance between damping and resonance can be assessed by observing the response to a rapid high-pressure flush of the system—the fast-flush test. In a system with high resonance and a low damping coefficient, the fast-flush test results in more than 1-3 oscillations above and below the baseline before the pressure waveform stabilizes. In an adequately damped system, the baseline is reached after one oscillation and in an overdamped system the baseline is reached after a delay without oscillations ^[6] (Fig. 5.3).

The radial artery is most commonly used for invasive blood pressure monitoring because of its relative ease of cannulation and extensive collateral circulation that minimizes complications. The dorsalis pedis artery is a convenient site for otolaryngologic procedures performed 180° away from the anesthesia machine. Other potential sites include the axillary and femoral arteries. The brachial artery is rarely used because of the lack of collateral circulation and the potential for forearm and hand ischemia. Factors that influence the site of cannulation include prior and current surgical location including anticipated free flap harvesting sites, history of ischemia on the limb to be cannulated, and the possible compromise of arterial flow due to patient positioning or surgical manipulation. Complications include hematoma, bleeding (which can be significant if the transducer tubing is not tightly affixed and any part of the system becomes disconnected), vasospasm, thrombosis, emboli, and rarely, infection.

Pulse Oximetry

Pulse oximetry provides invaluable information about the function of both the respiratory and circulatory systems. Oximetry depends on the observation that oxygenated and deoxygenated (reduced) hemoglobin differ in their absorption of red (660 nm) and infrared (940 nm) spectra of light. Oxyhemoglobin absorbs most infrared light and transmits most red light. Reduced hemoglobin absorbs more red light and transmits infrared light. The pulse oximeter uses this principle to determine the relative concentration of oxyhemoglobin in the blood. Red and infrared light is also absorbed by capillary blood, venous blood, soft tissue, skin, and bone. Therefore, it is the pulsatile component of light absorbance that the oximeter uses to calculate arterial oxygen concentration.

The accuracy of pulse oximetry is adversely affected when there is diminished tissue perfusion (e.g., with arterial inflow disease, hypothermia, or vasoconstriction). As the signal strength of the pulse decreases in relation to surrounding tissue absorbance, the ratio of a very weak signal of red to infrared light absorbance approaches unity, which correlates with a saturation of 85%. Therefore a poor plethysmography tracing with a saturation of 85% may represent artifact that should be checked against an independent technique. Similarly, methemoglobin (oxidized, ferric hemoglobin) has the same absorption coefficient at both red and infrared wavelengths and will register a saturation of 85%. Carboxyhemoglobin absorbs light at 660 nm identically to oxygenated hemoglobin and a pulse oximeter will register a falsely high reading in patients with carbon monoxide poisoning ^[7].

Another important clinical consideration is that the hemoglobin-oxygen dissociation curve is sigmoid in shape, making pulse oximetry insensitive to wide ranges of PaO_2 variability. Only the lower range of PaO_2 values are effectively monitored with pulse oximetry because the PaO_2 must fall below 100 mmHg before a value less than 100% will be detected, and below 60 mmHg before rapid changes will occur to alert the clinician.

Capnography

A capnogram is a plot of CO₂ concentration versus time during the ventilatory cycle. Modern capnography relies on the absorption of infrared light by CO₂. Gas concentration may be analyzed by continuous suction from the breathing circuit into a sample cell within the monitor or by using an adapter that is placed in the breathing circuit. Infrared light transmission through the gas is measured, and CO₂ concentration is then calculated. Monitoring capnography is useful to ensure that the patient is in fact being ventilated, to estimate PaCO₂, to estimate dead space (portion of the respiratory anatomy that is ventilated but not perfused), and for detecting certain other problems listed below. The gradient between PaCO₂ and ETCO₂ (normally 2–5 mmHg) reflects total deadspace, which is the sum of anatomic (the tracheobronchial tree that does not participate in gas exchange) and physiologic (the proportion of alveoli that are ventilated without being perfused) deadspace. The gradient increases with an increase of physiologic deadspace which occurs with low cardiac output, hypotension, or air embolism. Proper interpretation of the capnogram can alert a clinician to important problems and includes the examination of three key features: an inspiratory baseline of zero, a sharp increase in CO₂ concentration corresponding to the expiratory upstroke, and a steady alveolar plateau ^[8] (see Fig. 5.4). Rebreathing of carbon dioxide, incomplete paralysis, and expiratory airway disease may all be assessed using capnography.

Temperature

Thermoregulatory function and homeostasis are altered by anesthesia and surgery. Hypothermia commonly occurs from a combination of anesthetic-induced impairment of thermoregulatory function and exposure of the patient to the cool operating room environment. Because anesthetics have vasodilating properties, thermal energy redistributes from the core of the body to the periphery resulting in heat loss through radiation, conduction, convection, and evaporation ^[9]. As a general rule, core temperatures decrease by approximately 1°C during the first hour after anesthetic induction. Hypothermia has several negative consequences including delayed recovery from anesthesia, coagulopathy, and impaired wound healing. Hypothermia prevention probably decreases cardiovascular complications and infections [10, 11]. Shivering increases oxygen utilization by as much as 200-300% which can result in myocardial ischemia. Oncologic resection surgeries with peripheral tissue harvesting will require large surgical area preparation and result in significant heat loss with the inability to apply active surface warming devices. Underbody hot air warming devices are affective to prevent hypothermia and can rewarm the hypothermic patient in these cases. Because of the limited surgical exposure, hypothermia is generally not a problem for many otolaryngologic procedures and the use of active warm air circulating devices may result in an overheated patient.

Core temperature is considered the most important temperature monitoring variable because it determines thermoregulatory response and core temperature decreases have been associated with complications of hypothermia. All temperature monitoring devices, therefore are judged based on their ability to estimate core temperature. The gold standard is central blood temperature as measured from a pulmonary artery (PA) catheter. Esophageal temperature probes, if properly placed in the lower esophagus, also provide an accurate measurement of core body temperature because of its proximity to the aorta. More proximal positioning can lead to falsely lowered values as a result of the proximity to the trachea, which contains cooler inspired gas. Nasopharyngeal temperature can be measured with a probe positioned above the palate and is reasonably close to brain and core temperatures. A urinary catheter with a temperature thermistor can be used to measure bladder temperature, which is close to core temperature, although the accuracy of this measurement decreases with low urine output. Finally, skin and axillary temperatures are the least reflective of core body temperature because of the significant gradient between the core and periphery ^[12]. Though less common intraoperatively, temperature should also be measured to detect hyperthermia, which could indicate sepsis, malignant hyperthermia, or other hypermetabolic syndromes.

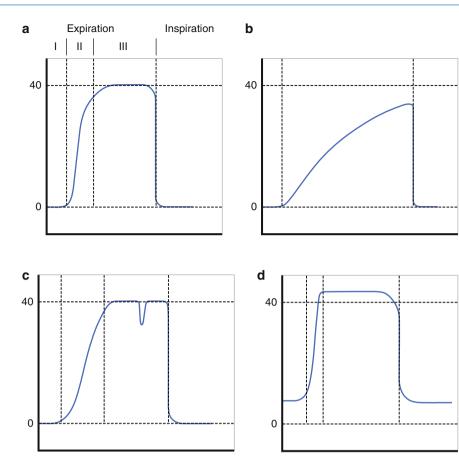


Fig. 5.4 (a) A normal capnograph demonstrating the three phases of expiration: phase I—dead space; phase II—mixture of dead space and alveolar gas; phase III—alveolar gas plateau. (b) Capnograph of a patient with severe chronic obstructive pulmonary disease with a

Additional Monitors

Beyond the basic monitors of the cardiovascular, pulmonary and thermoregulatory system that are ASA standards for all anesthetics, additional modalities are used, depending upon patient characteristics, surgical factors, and anesthetic plans. These include advanced monitors of the cardiovascular system [e.g., central venous pressure (CVP) monitoring, PA catheterization monitoring, and transesophageal echocardiography (TEE)], brain function monitoring [e.g., bispectral index (BIS)], brain perfusion monitors [(e.g., cerebral oximetry, transcranial Doppler (TCD)], and noninvasive hemoglobin measurement.

Central Venous Pressure

The CVP can be monitored by the placement of a catheter in one of several central veins including the internal jugular, subclavian, or femoral vein. The CVP reflects a patient's

gradual upslope and loss of the plateau. (c) Depression of the plateau indicates spontaneous respiratory effort and hence lack of muscle paralysis. (d) Failure of the inspired CO₂ to return to zero may represent an incompetent expiratory valve or exhausted CO₂ absorbent

blood volume, venous tone, and right ventricular performance and is a useful monitor if the factors affecting it are recognized and its limitations are understood. The CVP may reflect left heart filling pressures, but only in patients with good left ventricular function ^[13]. Following trends in serial measurements is more useful than individual numbers. Thromboses of the vena cavae and increased intrathoracic pressure, as occurs with PEEP, affect measurement of the CVP. In addition to the numerical value, the CVP waveform morphology can provide useful clinical information.

The normal CVP waveform (see Fig. 5.5) includes the following components:

- 1. The A wave occurs with right atrial contraction.
- The C wave occurs during isovolumetric ventricular contraction as the tricuspid valve bulges into the right atrium.
- 3. The X descent occurs as the tricuspid valve is pulled away from the right atrium during right ventricular ejection.
- 4. The V wave then occurs during rapid atrial filling in late diastole.

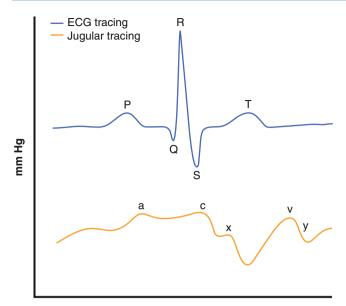


Fig. 5.5 The upward deflections (a, c, v waves) and downward deflections (x, y descents) of a central venous pressure tracing correlated with the ECG

5. The Y descent occurs when the tricuspid valve opens and blood from the right atrium empties rapidly into the right ventricle.

Abnormal CVP waveforms can provide insight regarding certain types of cardiovascular conditions. With the onset of atrial flutter or fibrillation, there will be a loss of the A wave. In junctional rhythm, cannon A waves occur as the right atrium contracts against a closed tricuspid valve. These dys-synchronous atrial contractions that produce A waves may also occur in complete heart block and ventricular arrhythmias. They also occur with increased resistance to right atrial emptying, such as occurs in tricuspid stenosis and with right ventricular hypertrophy due to pulmonic valve stenosis or pulmonary hypertension. Large "C–V" waves can indicate that there is a noncompliant ventricle ^[14]. Table 5.2 lists some indications for central venous catheter placement.

Pulmonary Artery Catheter

When the CVP cannot be used to estimate left-sided pressures, a PAC may be employed. A PA catheter is long and contains multiple lumens. The tip of the adult-sized catheter has a balloon with a volume capacity of 1.5 mL. The PAC is placed through a "cordis" or PAC introducer placed in a central vein. A PA catheter can measure cardiac output, mixed venous oxygen partial pressure/saturation, PA and right atrial pressures. The pulmonary capillary wedge pressure (PCWP) is an indirect indication of left ventricular end-diastolic pressure (LVEDP). Complications from insertion are rare but serious,

Table 5.2		
Indications for central venous catheter placement		
Major operative procedures involving large fluid shifts or blood loss in patients with good heart function		
Intravascular volume assessment when urine output is not reliable (e.g., renal failure)		
Surgical procedures with high risk of air embolism. The central venous pressure catheter may be used to aspirate intracardiac air		
Venous access for vasoactive drugs		
Inadequate peripheral venous access		

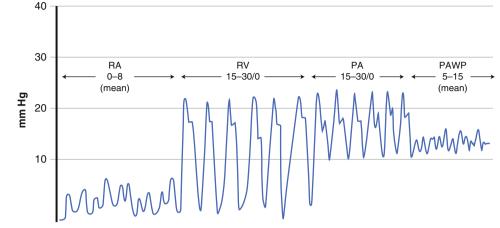
and include arterial injuries and hematoma development from the placement of the introducer, as well as arrhythmias, catheter knotting, valve injury, and PA rupture^[15].

PACs are placed in the central circulation and positioned with the use of fluoroscopy or more commonly by the use of insertion distance and wave form recognition. The PAC is inserted through the introducer and by inflating the balloon the catheter is advanced a few centimeters with each heartbeat. The "flow-directed" catheter is advanced from the right atrium into the right ventricle, where a sudden increase in systolic pressure with little-to-no change in diastolic pressure is detected. The catheter is then advanced past the pulmonic valve into the PA where there is a sudden increase in diastolic pressure. The PCWP tracing, which usually reflects left atrial pressure and LVEDP, is obtained by advancing the catheter several centimeters further until there is a change in the waveform associated with a drop in the measured mean pressure (Fig. 5.6). Deflation of the balloon results in reappearance of the PA waveform. Figure 5.7 shows a PA catheter removed from the package.

The PCWP waveform is analogous to the CVP waveform described previously. The PCWP will not accurately reflect LVEDP in the presence of pulmonary vascular disease, high levels of PEEP, or mitral valvular disease. Normal values for pressures observed during PA catheterization are listed in Table 5.3, and some indications for PA catheterization are listed in Table 5.4. The use of PA catheterization is less common in recent years due to clinical investigations demonstrating no outcome benefit or actual harm ^[16]. Nevertheless, it may provide valuable information in subsets of patients, especially in those with pulmonary hypertension prior to surgery. Head and neck surgery may require subclavian or femoral venous insertion of a PA catheter so as not to interfere with the surgical field.

Transesophageal Echocardiography

The ability to visualize cardiac and mediastinal morphology, chamber fullness, cardiac wall motion, blood flow, and valvular function has proven to be invaluable in **Fig. 5.6** Waveform of a pulmonary artery catheter as it is advanced from the right atrium to a "wedged" position in the pulmonary artery



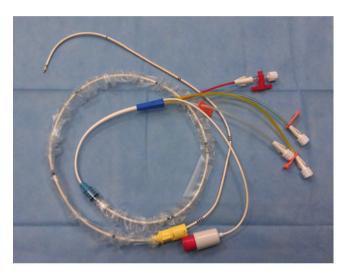


Fig. 5.7 PA catheter with protective sheath attached

Table 5.3

Normal intravasular pressures				
Location	Mean	Range		
Right atrium	5	1–10		
Right ventricle	25/5	15-30/0-8		
Pulmonary arterial systolic/diastolic	23/9	15-30/5-15		
Mean pulmonary arterial	15	10–20		
Pulmonary capillary wedge pressure	10	5-15		
Left atrial pressure	8	4–12		
Left ventricular end-diastolic pressure	8	4–12		
Left ventricular systolic pressure	130	90-140		

the management of patients undergoing cardiac surgery by cardiac anesthesiologists. Intraoperative TEE has become an accepted monitor for the management of cardiac patients undergoing noncardiac surgery. The ASA has endorsed an educational program and certification for limited TEE use by noncardiac anesthesiologists. Although many head and neck

Table 5.4

Indications for pulmonary artery catheterization include major procedures involving large fluid shifts or blood loss in patients with: Right heart failure, pulmonary hypertension Severe left heart failure not responsive to therapy Cardiogenic or septic shock with multiple-organ failure Hemodynamic instability requiring ionotropes or intra-aortic balloon counterpulsation

surgical patients have significant cardiovascular comorbidities and would therefore benefit from the use of intraoperative TEE monitoring, its use is impractical and often impossible because of the proximity of the surgical site and is therefore likely reserved for preoperative patient assessment.

Brain Monitoring

Cerebral perfusion pressure is determined by the formula:

CPP=MAP – CVP or ICP (whichever is higher)

Therefore, a decrease in MAP or significant increase in CVP could compromise cerebral blood flow. When the head is elevated relative to the rest of the body, adjusting the arterial line pressure transducer to the level of the ear, rather than the typical location at the midaxillary line will approximate the MAP at the Circle of Willis. The anesthesia team may use various forms of monitoring to evaluate the adequacy of cerebral perfusion.

Processed Electroencephalographic Monitoring

Brain function monitors have been introduced mainly as tools to monitor depth of anesthesia/level of consciousness. They generally process 1–4 frontal lobe electroencephalographic (EEG) channels using proprietary algorithms. For example, BIS monitors process two EEG channels to display a number, which correlates with likelihood of patient awareness ^[17]. In general, a number of 90–100 represents the awake state, 80–90 represents mild sedation, and 45–60 is consistent with general anesthesia. Intermediate numbers (e.g., 70) in a patient undergoing general anesthesia is associated with more awareness than lower numbers. Figure 5.8 shows a properly applied BIS monitor.

Brain function monitoring may reduce patient awareness during general anesthesia, although the studies yield conflicting data on this subject ^[18, 19]. Other than awareness monitoring, brain function monitors may aid in resource utilization, if less anesthetic drug is administered and cost and recovery times are optimized ^[20, 21]. Additionally, sudden changes in the EEG waveform, such as isoelectricity, may alert the clinician to the potential that cerebral ischemia is present. ENT procedures may be especially prone to cerebral ischemia, due to manipulation of carotid arteries in patients with atherosclerotic disease and use of the "beach-chair" position and hypotensive techniques in patients undergoing sinus surgery and similar operations and these devices may prove beneficial.

In 2006 an ASA Task Force examined the available literature and generated a practice advisory for brain function monitoring ^[22]. They did not find sufficient evidence at the time to recommend routine use of such monitors solely for the reduction of intraoperative awareness. They did recommend, however, that in patients with specific risk factors, the decision to use brain function monitors should be made on a case-by-case basis. Some of the risk factors cited that are relevant to otolaryngology patients include: a history of substance use or abuse, a previous episode of intraoperative awareness, a history of or anticipated difficult intubation, patients on high doses of opioids, ASA physical status 4 or 5, patients with limited hemodynamic reserve, airway surgery, trauma surgery, emergency surgery, use of neuromuscular blockers, and planned use of nitrous-opioid anesthesia.

Cerebral Oximetry (Near-Infrared Spectroscopy)

Cerebral oximetry is a noninvasive technique that involves placing near-infrared emitting diodes and sensors that use reflectance oximetry to measure the oxygen saturation of hemoglobin in the tissues below the sensor. These are often applied bilaterally on the forehead over the frontal lobe. Unlike pulse oximetry, brain oximetry measures arterial, venous, and capillary blood oxyhemoglobin saturation as well as cerebral tissue oxygenation. The displayed saturation is therefore an amalgam of oxygenation status of the compartments noted above ^[23]. At the onset of cerebral ischemia, oximetry values decrease as a result of increased oxygen extraction and loss of oxygenated blood in the arterial and

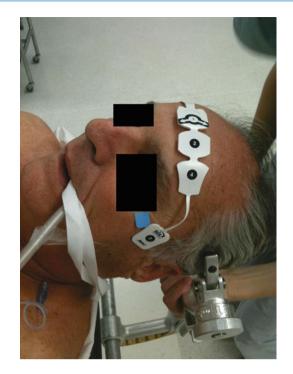


Fig. 5.8 BIS monitor

venous components. The utility and predictive value of cerebral oximetry have been shown in thoracic aortic repairs requiring hypothermic circulatory arrest, other cardiac surgical patients, and carotid endarterectomies—procedures where cerebral blood is impaired during periods of no-flow or hemodynamic instability. Both absolute nadirs and integrals of cerebral desaturation over time have been associated with postoperative cognitive dysfunction and longer recovery times ^[24–26].

In ENT cases that are prone to cerebral ischemia due to elevation of the head of bed ("beach-chair" position) or intentional ("controlled") hypotension, there may be a particular benefit to cerebral oximetry monitoring. There are investigations ongoing in this surgical subgroup.

Transcranial Doppler

TCD monitoring uses pulsed wave Doppler ultrasound for measurement of the blood flow velocity characteristics and for embolic detection in one or more major intracranial arteries. Most commonly, the middle cerebral artery is examined (insonated) through the acoustic window provided by the relatively thin temporal bone. The technique is not a direct measurement of cerebral blood flow, good signals can be difficult to obtain and maintain, and probe displacement during surgery can result in loss of the signal or inaccurate measurement due to angle of insonance-induced changes in the observed velocity profile. Despite these limitations, studies of patients undergoing carotid endarterectomy with TCD monitoring suggest a critical blood flow velocity reduction of around 50% as indicative of inadequate CBF that warrants intervention ^[27–29]. In complex ENT and skull base surgery where there is risk of elevated intracranial pressure, the pulsatility index of the TCD waveform is a means of monitoring for that complication.

Noninvasive Hemoglobin Measurement

Otolaryngology procedures, which often involve highly vascular areas, can be associated with significant blood loss. Furthermore, blood loss can be concealed in drapes due to a lack of pooling during head and neck cases, so blood loss is often difficult to estimate intraoperatively. A rapid, accurate noninvasive tool for dynamic hemoglobin or hematocrit monitoring would be ideal for this setting and would be less time consuming than intermittent blood sampling. There is now a noninvasive hemoglobin measurement device that is available in the USA, based upon near-infrared spectroscopy with transillumination of the fingertip. The advantage of this technique is the potential for integration into existing pulse oximeters. Limitations of this method include interference from chromophores other than hemoglobin in the skin and large variations in photon interaction lengths due to varying tissue thickness, blood vessel location and density, and changes in vessel diameter and subsequent optical pathlength during pulsation. Volumetric restriction of blood flow through the finger and monitoring of changes in transmission can help differentiate the absorption contributions of hemoglobin from those of surrounding tissues and therefore improve accuracy over purely optical methods. Several small clinical trials evaluating commercially available devices that utilize these techniques have demonstrated reasonable correlation with invasive measurements of hemoglobin^[30].

Monitoring Considerations Specific To Otolaryngology Procedures

Airway Compromise

There are several aspects of otolaryngology surgery that guide the anesthesiologist in deciding whether to employ more advanced monitoring. First and foremost is the fact that airway compromise is frequently of concern. Patients may present with distorted upper airway anatomy or airway Endotracheal tubes can become dislodged or kinked despite being carefully secured or the breathing circuit can become disconnected. Lateral rotation of the head during ear procedures, or head extension during neck procedures can cause a change in position of the tip of the endotracheal tube. During intraoral surgery, instrumentation to expose the mouth may obstruct the airway. Loss of the capnogram and low pressure alarms can signal dislodgement of the endotracheal tube or a circuit disconnect. High-pressure alarms often occur in the presence of a kinked tube or a tube that has been inadvertently advanced into a mainstem bronchus.

assessment and correction of problems more difficult.

Concealed Blood Loss

Another consideration in otolaryngology surgery is that blood loss may be concealed in the sterile drapes due to the lack of pooling, as occurs in the abdominal cavity. Alternative signs of hypovolemia, such as oliguria, systolic respiratory variation of the arterial waveform amplitude, or decrease in CVP may be helpful in identifying and monitoring the adequacy of volume resuscitation.

Common Comorbidities

Because patients with head and neck pathology often have a history of heavy tobacco use, there is a high incidence of COPD and cardiovascular disease that may require more invasive monitoring. In the case of COPD, an arterial line allows for serial blood gas measurements. For patients with uncontrolled hypertension, coronary artery disease, and renal insufficiency, an arterial line and/or a central venous catheter can be useful for close blood pressure monitoring and assessment of volume status.

Intense Surgical Stimulation

Laryngoscopy, bronchoscopy, and esophagoscopy are brief procedures with minimal blood loss. They are, however, intensely stimulating for short durations, often resulting in profound hemodynamic alterations. Invasive blood pressure monitoring may be considered for these relatively minor procedures if the patient is at high cardiovascular risk.

Challenging Ventilation Circumstances: Jet Ventilation

Jet ventilation poses monitoring challenges for the anesthesiologist. The adequacy of ventilation should be assessed continuously by observation of chest movement, oxygen saturation readings, and by listening for changes to the quality of sounds generated during air injection and exhalation. The patency of the airway can also be assessed by visually monitoring the endoscopic image. Capnography is unreliable during jet ventilation.

Calcium Homeostasis

Another otolaryngology procedure of special note is parathyroid surgery, where there is a requirement for frequent blood sampling to assess parathyroid hormone (PTH) levels. It is possible to obtain blood from a large peripheral IV that has been dedicated to this purpose. For the patient with poor peripheral access, however, an arterial line or central venous catheter may be more practical.

Neurophysiological Monitoring

Facial nerve monitoring is used during middle ear, mastoid, inner ear, and parotid and submandibular gland surgeries in order to identify the facial nerve and reduce the incidence of iatrogenic injury. Facial nerve injuries may occur secondary to nerve division, stretch, compression, thermal or electrical injury, and ischemia. The technique involves placing needle electrodes in locations that are optimal for recording activity from muscles innervated by branches of the facial nerve, including the frontal, zygomatic, buccal, and marginal mandibular, along with ground and stimulator anode electrodes. A stimulation probe is placed in the operative field.

Facial muscle activity is continually tracked during surgery, using a pulse-generator to evoke EMG responses ^[31].

The goals of facial nerve stimulation include:

- 1. Facial nerve identification by mapping the course of the nerve and differentiating the facial nerve from trigeminal branches.
- 2. Warnings of impending injury by unexpected facial nerve stimulation.
- 3. Reduction of mechanical trauma to the nerve.
- 4. Evaluation of the intactness of nerve function at the conclusion of the procedure.

Audible alarms may be activated when the facial nerve is stimulated by surgical movement or from evoked electrical stimulation. Neuromuscular blockade abolishes EMG signals, and must be avoided whenever the facial nerve is monitored.

Permanent facial nerve paralysis is an uncommon event following otolaryngology surgery, and studies thus far have not been sufficiently powered to address the issue of whether facial nerve monitoring reduces the likelihood of permanent injury. The choice as to whether to employ facial nerve monitoring is based on individual surgeon preference and experience ^[31]. EMG monitoring of the recurrent laryngeal nerve during thyroidectomy surgery is discussed in detail in Chap. 14.

Conclusion

ENT surgery provides unique monitoring challenges for the anesthesia team. Knowledge of the unique aspects of the surgical procedures is essential towards formulating a monitoring plan that anticipates the problems that may occur. Using the information provided to work with the surgical team to assure optimal patient outcomes is undoubtedly aided by appropriate patient monitoring.

References

- American Society of Anesthesiologists: standards for basic anesthetic monitoring. 2005. Accessed at www.asahq.org/publications-AndServices/standards/02.pdf. Accessed 1 May 2011.
- Goldschlager N. Use of the treadmill test in the diagnosis of coronary artery disease in patients with chest pain. Ann Intern Med. 1982;97:383–8.
- Pickering TG. Principles and techniques of blood pressure measurement. Cardiol Clin. 2002;20:207–23.
- Kleinman B. Understanding natural frequency and damping and how they relate to the measurement of blood pressure. J Clin Monit. 1989;5:137–47.
- Todorovic M, Jensen EW, Thogersen C. Evaluation of dynamic performance in liquid-filled catheter systems for measuring invasive blood pressure. Int J Clin Monit Comput. 1996;13:173–8.
- Kleinman B, Powell S, Kumar P, Gardner RM. The fast flush test measures the dynamic response of the entire blood pressure monitoring system. Anesthesiology. 1992;77:1215–20.
- 7. Tremper KK, Barker SJ. Pulse oximetry. Anesthesiology. 1989;70:98–108.
- Thompson JE, Jaffe MB. Capnographic waveforms in the mechanically ventilated patient. Respir Care. 2005;50:100–8 [discussion 8–9].
- 9. Sessler DI. Perioperative heat balance. Anesthesiology. 2000;92: 578–96.
- Frank SM, Fleisher LA, Breslow MJ, et al. Perioperative maintenance of normothermia reduces the incidence of morbid cardiac events. A randomized clinical trial. JAMA. 1997;277:1127–34.
- Kurz A, Sessler DI, Lenhardt R. Perioperative normothermia to reduce the incidence of surgical-wound infection and shorten hospitalization. Study of Wound Infection and Temperature Group. N Engl J Med. 1996;334:1209–15.
- Cereda M, Maccioli GA. Intraoperative temperature monitoring. Int Anesthesiol Clin. 2004;42:41–54.

- Mangano DT. Monitoring pulmonary arterial pressure in coronaryartery disease. Anesthesiology. 1980;53:364–70.
- Mark JB. Central venous pressure monitoring: clinical insights beyond the numbers. J Cardiothorac Vasc Anesth. 1991;5:163–73.
- 15. American Society of Anesthesiologists Task Force on Pulmonary Artery Catheterization. Practice guidelines for pulmonary artery catheterization: an updated report by the American Society of Anesthesiologists Task Force on Pulmonary Artery Catheterization. Anesthesiology. 2003;99:988–1014.
- Connors Jr AF, Speroff T, Dawson NV, et al. The effectiveness of right heart catheterization in the initial care of critically ill patients. SUPPORT Investigators. JAMA. 1996;276:889–97.
- Glass PS, Bloom M, Kearse L, Rosow C, Sebel P, Manberg P. Bispectral analysis measures sedation and memory effects of propofol, midazolam, isoflurane, and alfentanil in healthy volunteers. Anesthesiology. 1997;86:836–47.
- Myles PS, Leslie K, McNeil J, Forbes A, Chan MT. Bispectral index monitoring to prevent awareness during anaesthesia: the B-Aware randomised controlled trial. Lancet. 2004;363:1757–63.
- 19. Avidan MS, Zhang L, Burnside BA, et al. Anesthesia awareness and the bispectral index. N Engl J Med. 2008;358:1097–108.
- Warrington RJ, Buehler SK, Roberts KB. Inflammation-inducing factors from human lymphocytes. Correlation with polymorphonuclear leucocyte migration enhancement of inhibition. Int Arch Allergy Appl Immunol. 1976;51:186–97.
- Yli-Hankala A, Vakkuri A, Annila P, Korttila K. EEG bispectral index monitoring in sevoflurane or propofol anaesthesia: analysis of direct costs and immediate recovery. Acta Anaesthesiol Scand. 1999;43:545–9.
- 22. American Society of Anesthesiologists Task Force on Intraoperative Awareness. Practice advisory for intraoperative awareness and

brain function monitoring: a report by the american society of anesthesiologists task force on intraoperative awareness. Anesthesiology. 2006;104:847–64.

- Madsen PL, Secher NH. Near-infrared oximetry of the brain. Prog Neurobiol. 1999;58:541–60.
- Rigamonti A, Scandroglio M, Minicucci F, Magrin S, Carozzo A, Casati A. A clinical evaluation of near-infrared cerebral oximetry in the awake patient to monitor cerebral perfusion during carotid endarterectomy. J Clin Anesth. 2005;17:426–30.
- Murkin JM, Adams SJ, Novick RJ, et al. Monitoring brain oxygen saturation during coronary bypass surgery: a randomized, prospective study. Anesth Analg. 2007;104:51–8.
- Slater JP, Guarino T, Stack J, et al. Cerebral oxygen desaturation predicts cognitive decline and longer hospital stay after cardiac surgery. Ann Thorac Surg. 2009;87:36–44 [discussion 44–5].
- Ackerstaff RG, Moons KG, van de Vlasakker CJ, et al. Association of intraoperative transcranial doppler monitoring variables with stroke from carotid endarterectomy. Stroke. 2000; 31:1817–23.
- Ogasawara K, Suga Y, Sasaki M, et al. Intraoperative microemboli and low middle cerebral artery blood flow velocity are additive in predicting development of cerebral ischemic events after carotid endarterectomy. Stroke. 2008;39:3088–91.
- Dunne VG, Besser M, Ma WJ. Transcranial Doppler in carotid endarterectomy. J Clin Neurosci. 2001;8:140–5.
- McMurdy JW, Jay GD, Suner S, Crawford G. Noninvasive optical, electrical, and acoustic methods of total hemoglobin determination. Clin Chem. 2008;54:264–72.
- Eisele DW, Wang SJ, Orloff LA. Electrophysiologic facial nerve monitoring during parotidectomy. Head Neck. 2010;32: 399–405.

Oxygen Delivery Systems and the Anesthesia Workstation

James B. Eisenkraft and Robert Williams

Introduction

The term anesthesia workstation is defined as a system for the administration of anesthesia to patients. It consists of the anesthesia gas supply device (i.e., the anesthesia machine), anesthesia ventilator, monitoring devices, and protection device(s)^[1]. Because otolaryngologists operate in close proximity to the head, neck, and airway, anesthesiologists are usually required to manage otolaryngologic patients who may be at a distance from the anesthesia machine. Emergent situations such as airway loss, electrical power outages, fires and equipment failures in the operating room must be immediately addressed to assure patient safety and prevent adverse outcomes^[2]. Given these unique situations, an otolaryngologist should be familiar with the anesthesia machine and workstation should he/she be called upon to assist his/her anesthesia colleagues during routine care and intraoperative emergencies. Additionally many otolaryngology patients have significant pulmonary, cardiovascular, and airway pathology requiring the need for supplemental oxygen in the perioperative period. A working knowledge of various oxygen delivery systems is critical for the proper care and wellness of the perioperative patient. Therefore, it is important that every otolaryngologist gains a familiarity with the devices and technologies that deliver oxygen to patients. The purpose of this chapter is to introduce the otolaryngologist to the basic principles of the anesthesia workstation, anesthesia gas delivery system, and oxygen supply equipment while serving as a basic overview of these devices for the practicing anesthesiologist.

In 1986, the American Society of Anesthesiologists published standards for basic anesthetic monitoring, and these standards are periodically updated as practice and technologies change. Standard I requires that: "Qualified anesthesia personnel shall be present in the room throughout the

conduct of all general anesthetics, regional anesthetics and monitored anesthesia care." The stated objective of this standard is: "Because of the rapid changes in patient status during anesthesia, qualified anesthesia personnel shall be continuously present to monitor the patient and provide anesthesia care. In the event there is a direct known hazard, e.g., radiation, to the anesthesia personnel which might require intermittent remote observation of the patient, some provision for monitoring the patient must be made. In the event that an emergency requires the temporary absence of the person primarily responsible for the anesthetic, the best judgment of the anesthesiologist will be exercised in comparing the emergency with the anesthetized patient's condition and in the selection of the person left responsible for the anesthetic during the temporary absence" [3]. It is possible that the anesthesia care provider may be indisposed when an emergency occurs or he/she may request assistance from other physicians in the operating room. It is at such times that some knowledge of the basic functions and controls of anesthesia workstation may be indispensable to correct hypoxemia or hypercapnia. The anesthetized patient should be at minimal risk of injury when all care providers acquire this expertise and are able to act to maintain oxygenation and ventilation.

Oxygen Supply Systems

Oxygen is the most important gas delivered to the OR and to the anesthesia workstation. An understanding of how oxygen is supplied to the OR is important because failures of pipedin oxygen systems continue to be reported ^[4]. Recent studies have found that education of anesthesia personnel in how to respond to such situations appears to be inadequate ^[5, 6].

Oxygen can be piped into the operating rooms from bulk liquid oxygen reservoirs or manifold systems of large high pressure oxygen cylinders connected together^[7]. In both cases the oxygen is stored at high pressures so that as the oxygen is drawn from these sources, before entering the pipeline it passes through a pressure regulator that reduces the oxygen pressure to approximately 50 pounds/inch² gauge (psig; this is

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Fig. 6.1 Bulk liquid oxygen storage vessels



Fig. 6.2 Cylinder manifold arrangement for pipeline supply

the pressure measured above atmospheric pressure). Modern facilities that consume large amounts of oxygen use liquid oxygen bulk systems that provide enough oxygen to supply an entire hospital for extended periods (Fig. 6.1).

Older facilities may use tank manifolds. These consist of two sets of cylinder banks connected together into a central controller mechanism (Fig. 6.2). One bank acts as the primary oxygen source. The secondary system is used when the primary system requires refilling or installation of full cylinders. All oxygen supply systems are required to have a secondary or backup system in the event that the primary system is drained or experiences mechanical failure. The controller box measures the pressures in both banks of cylinders and automatically switches from the primary to the secondary bank when pressure falls below safe levels. Safety systems with alarm mechanisms monitor system pressure. Alarms are activated when pressure falls below a set minimum level (normally 40 psig). Under this circumstance, bulk oxygen would be supplied from the secondary system.

Cylinder Systems

Oxygen cylinders are available in various sizes depending on the intended application. Sizes are classified by a lettering system ^[8]. Generally two sizes are used in hospitals: size "H" is a large cylinder not intended to be portable and is generally used in locations where piped-in wall oxygen is unavailable. The smaller size "E" cylinders are used for patient transport. E cylinders are also installed in the oxygen hanger yokes on the anesthesia workstation as backup in the event that the pipeline supply system fails (Fig. 6.3). Oxygen cylinders are filled to very high pressures (e.g., ~2,200 psig for a full



Fig. 6.3 Oxygen (*green*) and air (*yellow*) E cylinders in respective hanger yokes in rear of workstation

oxygen tank). As tank oxygen enters the workstation it first passes through a pressure reducing regulator so that oxygen from the tank enters the anesthesia workstation at 45 psig. Similarly, a regulator (and flow meter) must be attached to a freestanding E cylinder in order to deliver oxygen at low flows to respiratory therapy devices such as nasal cannulae (Fig. 6.4). A tank system is now available that "fuses" the regulator permanently to the cylinder (Fig. 6.5). This is a great advantage as the clinician is never burdened with changing the regulator.

The pressure of oxygen in a standard E cylinder is displayed on a mechanical pressure gauge. Because oxygen exists as a gas at room temperature, it obeys the gas laws ^[9]. Thus the pressure displayed on the gauge can be used to estimate the volume of oxygen available from that cylinder. A full E cylinder of oxygen with a pressure of 2,000 psig can



Fig. 6.4 Oxygen tank with detachable regulator and flow control knob (red)



Fig. 6.5 Oxygen cylinder with integrated reducing valve and flow control for flows of 0.25–25 L/min

evolve approximately 670 L of gaseous oxygen at room temperature and pressure. The oxygen flow rate (L/min) determines the time until the cylinder is empty.

The following is the calculation for standard "E" cylinders:

minutes of oxygen = $0.28 \times \text{cylinder pressure (psig) / flow (L / min)}$

Example: a cylinder with a pressure of 1,800 psig and an oxygen flow of 10 L/min will empty in approximately 50 min.

It is important to be familiar with the locations in which full tanks of oxygen are stored so that they can be readily obtained whenever necessary. Empty tanks should be stored separately to avoid confusion (Fig. 6.6).

Medical Gas Safety

The Compressed Gas Association (CGA) publishes the national standards for specifications of fittings for medical gases including oxygen, air, nitrous oxide, and vacuum ^[10]. These standards are intended to assure that there is no possibility of errors in gas connection. Accidental connection of a gas other than oxygen for delivery to a patient could prove catastrophic. The two standard systems used are the Diameter Index Safety System (DISS) and Pin Index Safety System (PISS). DISS is used mainly for high pressure gas delivery at 50 psig from piped-in wall gas sources and also larger cylinders. The fittings for each gas are unique (different diameters) and will not allow for cross-connections. DISS connectors are found at the gas inlets of the anesthesia

workstation. (Fig. 6.7) At the wall end of the gas supply hose connecting the machine to the wall gas outlet there may be a DISS connector (Fig. 6.8), but more often there is a manufacturer gas-specific "quick connect" that is designed for ease of connection/disconnection of the gas hose from the wall outlet. These "quick connects" are not standardized among manufacturers. Thus while a Chemetron oxygen connector will not fit a Chemetron N₂O wall outlet, it will not fit an Ohmeda oxygen wall outlet (Fig. 6.9). Thus the quick connects are gas specific but only within a manufacturer. This can create problems when a facility has quick connects from several different manufacturers. Ideally there should be uniformity within a facility, using only DISS connectors (Fig. 6.8).

The PISS is used mainly for small (e.g., "E" sized) portable medical gas cylinders. Pin configurations on the cylinder and on the hanger yoke are unique for each medical gas. It is never acceptable to attempt to defeat these gas-specific connections (Fig. 6.10). The standard E cylinder for oxygen can be hung in the oxygen hanger yoke of an anesthesia workstation because of the pin-indexed system in the tank valve (Fig. 6.11). A full E cylinder provides the machine a backup supply of 670 L of gaseous oxygen.

The newer E cylinder shown in Fig. 6.12a, which has the regulator "fused" to the tank, does not have a PISS connection and therefore cannot hang in a yoke on the anesthesia



Fig. 6.6 Storage rack of full oxygen tanks. Empty tanks are stored elsewhere to avoid confusion



Fig. 6.7 Diameter-indexed safety system (DISS) gas connectors in back of anesthesia workstation. *Green*=oxygen; *yellow*=air; *blue*=nitrous oxide; *white*=vacuum

machine. It does, however, offer two important features. First, the tank is filled with oxygen to a pressure of 3,000 psig which can evolve 1,000 L of oxygen at room temperature and pressure (Fig. 6.12b). Second, the tank has a pressure regulator and DISS outlet connector that can deliver oxygen at 50 psig. It can therefore be used as a backup supply for the machine by disconnecting the machine hose from a DISS wall outlet and connecting it to the 50 psig outlet on the tank. (Fig. 6.12c). This only applies, of course, if the wall end of the machine's oxygen supply hose is fitted with a DISS connector (and not a manufacturer-specific quick connect).

The Anesthesia Workstation

The concept of the anesthesia workstation has evolved over time based on experience and advances in technology. The goal of its design is to maximize utility and ergonomics of the safe administration of anesthesia and patient monitoring. The workstation comprises:

- The anesthesia machine (gas delivery device)
- Anesthesia vaporizers
- Common gas outlet
- Ventilator
- Ventilatory monitoring with gas/agent analysis
- Auxiliary gas outlet (oxygen)
- Physiologic monitors and alarms

The anesthesia gas delivery system includes the above plus an anesthesia breathing system and waste gas scavenging system

Individual institutions will determine the fine-tuning of the anesthesia workstation's components by various factors including federal and state regulations, accrediting body standards, best practice, manufacturer's reliability and support, and available resources. Key components of the anesthesia workstation are reviewed in the following section.

Anesthesia Machine

Anesthesia machines have evolved from simple pneumatic (requiring only gas under pressure) devices to complex, integrated electronically controlled computerized systems with numerous controls, displays and alarm systems. In the USA, the two largest manufacturers are G.E. Healthcare (Madison, WI) and Drager Medical (Telford, PA). Other manufacturers include Mindray (formerly Datascope), Penlon, Blease and Maquet. Figures 6.13 and 6.14 show examples of recent models of anesthesia workstations with the latest technology. Despite differences in appearance, all workstations are basically the same in function.

The anesthesia machine (gas delivery device) receives compressed gases (O_2 , N_2O and air) from wall (pipeline) sources, or backup tank (cylinder) sources ^[11]. Gas cylinders and wall hoses are color coded as follows: green=oxygen; blue=nitrous oxide; yellow=air; white=hospital vacuum; purple=separate vacuum system for removing waste anesthesia gases (Fig. 6.7). The pressures of the gases supplied to the machine are displayed on gauges, or, on some recent electronic workstations, digitally on a computer display screen (Fig. 6.15). These gauges are used to monitor the adequacy of the gas supplies. The anesthesia machine is used to create a gas mixture of known composition at a known gas

Fig. 6.8 DISS wall outlet connectors

OXYGEN OXYGEN AIR SUCTION USE NO OIL

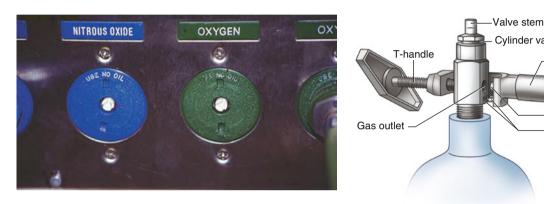


Fig. 6.9 Ohmeda "quick connect" gas outlets. These are gas specific within each manufacturer but not between manufacturers

Fig. 6.11 Oxygen tank and hanger yoke. It is important to be able to exchange tanks on the machine, and verify that a tank key/wrench is present for opening or closing the tank valve

Cvlinder valve

Hanger voke

check valve (inside)

То Gasket machine

"Pin-indexed" configuration

familiar glass rotameter tubes to measure the flow by observing the vertical height at which a ball or bobbin rises in the rotameter tube and reading the flow on an adjacent calibrated scale (Fig. 6.16). On machines with mechanical flow control valves, turning the knob counterclockwise opens the valve and increases flow. Conversely, clockwise rotation decreases and eventually stops gas flow. The recent electronic workstations use flow sensors to measure gas flows, and display the flows as virtual rotameters on a computer screen, as well as digitally (Fig. 6.17). Some brands of workstation use flow control knobs and mechanical valves but use gas flow sensors and screen displays of flow. In the most recent model from GE (Aisys), gas flows are selected, controlled, and displayed all electronically (Fig. 6.18). When approaching a workstation the operator must determine: (1) how do I control the flows (knob, electronic control)? and (2) how/where is the flow displayed?

Anesthesia machines incorporate flow sensitive proportioning systems that are designed to prevent delivery of <25% oxygen when it is administered with nitrous oxide. If air is used with oxygen, then an oxygen concentration of 21% could be delivered.



Fig. 6.10 Pin-indexed safety system for medical gas cylinders. Nitrous (blue) and oxygen (green) tanks showing different positions of the recepticles where the two pins in the hanger vokes would engage for each of these two gases. This system is a national standard

flow rate (e.g., $O_2 1 L/min + N_2O 2 L/min = 33\% O_2 in N_2O$ at a fresh flow rate of 3 L/min).

Traditional anesthesia machines use flow control knobs that operate mechanical valves to adjust gas flow and the

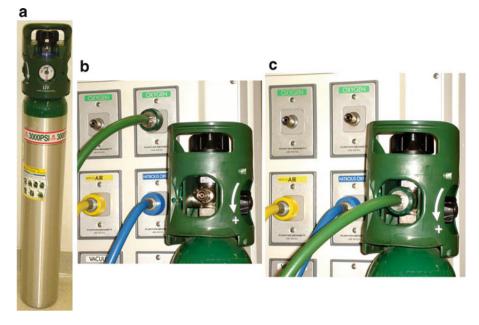


Fig. 6.12 Oxygen tank with integrated valves filled to 3,000 psig: (a) E cylinder full view. The low pressure outlet nipple can provide 0.25-25 L/min of oxygen; (b) showing DISS 50 psig outlet connector on tank. Note that the workstation's green oxygen hose is connected to

the wall oxygen DISS outlet; (c) if pipeline oxygen fails, the oxygen hose from the workstation can be connected to the 50 psig DISS connector on the tank. A full tank provides up to 3,000 L of gaseous oxygen

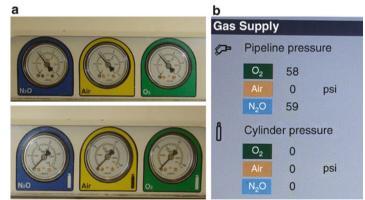
Fig. 6.13 GE Aisys Carestation (GE Healthcare, Madison, WI). All workstation functions are controlled electronically. In the event of power failure, oxygen may be delivered into the breathing system from the Alternate Oxygen system which has a manual flow control and a mechanical rotameter to measure oxygen flow



Fig. 6.14 Drager Apollo Anesthesia Workstation (Drager Medical, Telford, PA). This workstation uses mechanical gas flow controls and vaporizers



Fig. 6.15 Gas supply pressure displays on anesthesia workstations, showing pipeline and tank pressures. (a) Mechanical gauges; (b) in this workstation, gas pressures are measured by pressure transducers and displayed digitally on the workstation's computer screen



The gas mixture created at the flowmeters enters the anesthesia vaporizer where a potent inhaled volatile anesthetic (e.g., sevoflurane, desflurane, isoflurane) is added to produce a precisely controlled concentration of that agent in the outflowing gas mixture. Inhalational anesthetics are liquids at atmospheric pressure and room temperature and must be converted to a vapor for clinical use. The vaporizer converts a liquid anesthetic agent to its vapor phase and adds a controlled amount to the fresh gas flow ^[12]. Isoflurane, desflurane, and sevoflurane are the three anesthetic agents that are **Fig. 6.16** Workstation with traditional gas flow control knobs that operate mechanical needle valves, traditional rotameter gas flow meters, and mechanical vaporizers





Fig. 6.17 Screen display of a computerized workstation. Gas flows are measured electronically and displayed as virtual flow meters. Agent concentration is also controlled electronically and displayed on the screen



Fig. 6.18 Screen of GE Aisys Carestation in which all functions are controlled and displayed electronically. The green com wheel (*right lower corner*) is used to adjust settings and then pressed to confirm them. Settings are *increased by clockwise* rotation of the com wheel

currently available in the USA. Vaporizers are specific for each anesthetic agent. Up to three vaporizers can be mounted on the anesthesia machine and are removable by the user (Fig. 6.16). Safety systems are incorporated into the design so that only one vaporizer/agent can be in use at a given time. Anesthetic agent analysis and monitoring are necessary for safe delivery ^[13]. This is a function of the gas analyzer that also analyzes oxygen, nitrous oxide, and end-tidal CO₂. Anesthetic agents have a specific odor which may alert personnel to possible leaks in the system and minimize accidental exposure.

The two major manufacturers of anesthesia machines and vaporizers in current use are GE Healthcare's Datex Ohmeda division and Drager Medical. There are several types of vaporizers: mechanical and electronic. Mechanical vaporizers are simple gas flow splitting devices that are operated by adjusting a dial to the desired concentration (Fig. 6.19). GE Datex Ohmeda's Tec 7 series and the Drager Vapor 2,000



Fig.6.19 Mechanical vaporizers for isoflurane (*purple*) and sevoflurane (*yellow*). The concentration dial is turned *clockwise to decrease* settings



Fig. 6.20 Aladin electronic vaporizing system on GE Datex ADU workstation. The yellow cartridge is agent-specific for sevoflurane. In the ADU the large silver colored wheel controls agent concentration and is *rotated clockwise to decrease* concentration. Clearly (from Figs. 6.18, 6.19 and this figure) it is important to know in which direction to turn the wheel/dial to change agent concentration so as to avoid an unintended overdose or underdose

series are the most commonly used vaporizers. With these vaporizers, the agent concentration is decreased or turned off by turning the dial in a clockwise direction.

The GE Tec 7 and Drager D-Vapor are electronic vaporizers that resemble the Tec 7 and Vapor 2000 series but are specially designed to deliver desflurane, a very volatile anesthetic agent (i.e., it boils at 22.8 °C, which is just above room temperature) ^[14]. These vaporizers require electrical power because they are heated and pressurized to 2 atm.

Electronic vaporizing systems are manufactured by GE Datex Ohmeda and used in the ADU workstation and Aisys Carestation ^[15]. The Aladin and Aladin 2 cassettes contain the liquid anesthetic agent and are inserted into a bay in the front of the anesthesia machine.

Aladin Electronic Vaporizers

The electronic control mechanism is incorporated into the anesthesia machine and also identifies the agent-specific cassette (Fig. 6.20). The desired agent concentration is set on the main control panel. Cassettes are color coded by agent. There is only one bay on these anesthesia machines and cassettes are easily inserted and removed. Cassettes must be removed from the machine for refilling. An advantage is that they are lightweight and portable. In the ADU, the agent control dial is similar to that of the conventional mechanical

vaporizers in that clockwise rotation decreases or turns off the agent. In the GE Aisys Carestation, the electronic agent control is such that turning the knob clockwise increases concentration of agent. This is important to remember when, in an emergency, one wishes to discontinue administration of agent and deliver only oxygen.

Oxygen Flush Valve

All anesthesia workstations have a prominently displayed oxygen flush control (Fig. 6.21), usually located at the front of the machine. Activating the oxygen flush control by pressing the button allows oxygen supplied to the machine (whether from pipeline or tank supply) to be delivered directly to the common gas outlet (CGO) (i.e., bypassing the flowmeters and vaporizers) at a flow of 35-75 L/min, and (depending upon the model of machine and any pressurelimiting system) at pressures of up to 55 psig. Indications for its use include emergent need for 100% oxygen and to refill the ventilator bellows or breathing circuit reservoir bag. The valve will deliver oxygen as long as it is depressed. The valve must be single purpose, self-closing, and designed to minimize accidental activation. Hazards associated with the flush valve include the possibility of barotrauma as unknown levels of pressure are introduced into the breathing circuit.

Fig. 6.21 Oxygen flush controls. Pressing this control button results in an oxygen flow of 35–75 L/min being delivered from the machine's common gas outlet into the breathing system. Care must be taken to avoid barotrauma when using this control



Auxiliary Oxygen Flowmeter

Anesthesia workstations are equipped with an auxiliary oxygen flow meter that has the same oxygen supply source as the machine itself (i.e., pipeline or tank) and which permits flows of up to 12 L/min. The auxiliary flow meter is of the same design as a rotameter and supplies oxygen via a nippleor diameter-indexed safety system (DISS) oxygen threaded connection. An auxiliary oxygen flowmeter is incorporated into the anesthesia machine's design allowing for oxygen delivery to the patient without turning on the anesthesia machine (see Fig. 6.22). It may be used to deliver oxygen directly to the patient via nasal cannula or face mask. Most importantly, this flowmeter should be used to connect to the self-inflating resuscitation bag in the event of a problem with the anesthesia machine and will function independently of the anesthesia machine and in the absence of electrical power.



Fig. 6.22 Auxiliary oxygen flow meter (0–10 L/min) and outlet nipple

Oxygen Proportioning System

Anesthesia machines incorporate systems to prevent delivery of a hypoxic gas mixture to the breathing system. If oxygen supply is compromised, N_2O is automatically decreased or discontinued. Proportioning systems are designed to deliver a minimum of 25% oxygen in the fresh gas mixture when N_2O is being used. When air/O₂ is used, the minimum oxygen concentration is of course 21%. An oxygen analyzer to continuously monitor the patient's inspired oxygen concentration is a requirement.

Fresh Gas Mixture

The gas mixture of O_2/N_2O or air/ O_2 with added agent that emerges from the vaporizer is the so-called "fresh gas" mixture. This leaves the anesthesia machine via the common gas outlet (CGO) and is conducted to the anesthesia breathing system or patient circuit. While the CGO may be obvious on some machines (Fig. 6.23), it may not be accessible in some newer designs.

Fig. 6.23 Examples of accessible common gas outlets



Anesthesia Breathing System

The most commonly used breathing system is the circle system (Fig. 6.24) ^[16]. It is designed so that gases flow in a circular path through separate inspiratory and expiratory pathways. The system prevents the rebreathing of exhaled carbon dioxide by incorporating a CO_2 absorber but allows the rebreathing of other exhaled gases. The components of the circle system include (Fig. 6.25, see letters below):

- Fresh gas inlet (A)
- Inspiratory unidirectional valve (B)
- Inspiratory limb (C)
- Y-piece (D)
- Oxygen, CO₂ and agent analyzer
- Connector to patient's airway (E)
- Expiratory limb (F)
- Spirometry and pressure monitoring
- Expiratory unidirectional valve (G)
- Reservoir bag (H)
- Adjustable pressure-limiting (APL or "pop-off") valve (I)
- Bag/ventilator switch (J)
- Carbon dioxide absorber (K)
- Waste gas scavenging system

The CGO delivers the fresh gas flow to the circuit inlet connection. During inspiration, the inspiratory valve opens and gas flows down the inspiratory limb to the Y-piece. Gas for analysis is continuously sampled from this site. The inspired gas mixture enters the patient's airway and lungs. During exhalation the inspiratory unidirectional valve closes and gas flows up the expiratory limb through the now open expiratory unidirectional valve. The exhaled gas, which contains the patient's excreted CO_2 , takes the path of least resistance and begins to fill the reservoir bag. When the bag is filled to a pressure as set by the APL valve, the APL valve opens to allow excess gas to leave the circuit and enter the waste gas scavenging system for removal from the operating room (Fig. 6.24).

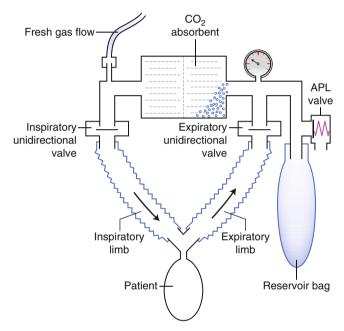


Fig. 6.24 Schematic of circle breathing system. For details, see text

Adjustable Pressure Limit Valve

The APL valve is a user adjustable valve also known as a "pop-off" or pressure relief valve (Fig. 6.26). Its purpose is to dump gas to the scavenging system when the valve's opening pressure threshold is exceeded. APL valves are commonly designed as a rotary control knob. The American Society for Testing and Materials (ASTM) standard requires that valves with rotary controls be designed so that turning clockwise will increase the pressure limit and will ultimately close the valve. It is recommended that less than one full turn of the knob can adjust the valve's entire range. Also, the valve should be marked with arrows or pressure values. During manual ventilation, the valve should be partially open and set to the desired pressure limit.

Fig. 6.25 Circle breathing system on GE Datex ADU workstation. For details, see text

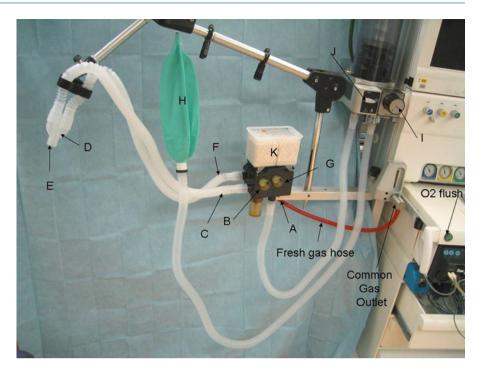


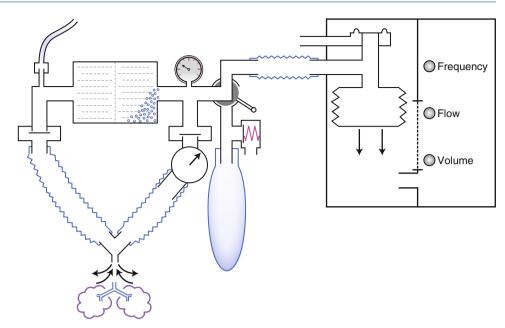


Fig. 6.26 Adjustable pressure-limiting (APL or "pop-off") valve and bag/ventilator selector switch



Fig. 6.27 Carbon dioxide absorber cartridge containing soda lime. Fresh soda lime is white in color but changes to purple as carbon dioxide is absorbed. Below the cartridge are the inspiratory and expiratory unidirectional valves of the circle breathing system

When the patient begins his/her next inspiration, fresh gas entering the circuit (Fig. 6.25a) begins to flow towards the Y-piece (Fig. 6.25d) but the fresh gas flow rate from the machine is not sufficient to satisfy the patient's spontaneous inspiratory flow rate. The difference is therefore drawn from the gas stored in the reservoir bag (Fig. 6.25h). Since this gas contains CO_2 , it must flow through the CO_2 absorber (Fig. 6.25k) where an absorbent (usually soda lime) scrubs the gas of CO_2 . The general principle of carbon dioxide absorption is that of a base neutralizing an acid. Carbonic acid formed by the reaction of carbon dioxide and water reacts with the base hydroxide. The end products are water and carbonate. Color indicators are incorporated into the absorbent so that soda lime changes color (from colorless to purple) as CO_2 is absorbed (Fig. 6.27). It is possible that after downtime, soda lime may revert to its original (white) color but have a **Fig. 6.28** Circle system connected to anesthesia ventilator. The reservoir bag has been switched out and functionally replaced by the hanging ventilator bellows in a bellows housing



much lower absorption capacity. It is recommended that absorbent is changed with regular frequency. The gas leaving the absorber joins the fresh gas flowing to the patient.

The bag/ventilator switch determines the type of ventilation to be used (Fig. 6.26). In the bag position, the patient may breathe spontaneously, inhaling from the reservoir bag as described above. If one wishes to provide positive pressure ventilation, one must squeeze the reservoir bag and increase the pressure limit setting on the APL valve so that gas flows to the patient rather than to the scavenging system. Ideally, for a positive pressure inspiration one wants to squeeze the bag while the APL valve is completely closed, and for exhalation (which is spontaneous) let go of the bag so it can refill, and at the same time, open the APL valve so excess gas can leave the breathing circuit. This function is provided automatically by the anesthesia ventilator. The ventilator circuit connects to the circle system via a selector switch that selects bag mode in which the reservoir bag and APL valve are in use, or the ventilator circuit. In principle the ventilator functions as follows: The reservoir bag is replaced by a bellows in a bellows chamber or by a piston in a cylinder, and the APL valve is replaced by the ventilator pressure relief valve (PRV) (Fig. 6.28). During inspiration, the bellows (which has replaced the reservoir bag) forces its contained gas into the breathing circuit and at the same time closes the PRV so no gas escapes from the circuit. During passive exhalation, the bellows/piston is no longer forcing gas into the circuit so it refills and at the same time the PRV opens to allow excess gas to leave to scavenging. Mechanical ventilation will begin using selected settings after switching

the valve to ventilation mode. Unidirectional valves control the directional flow of inspiratory and expiratory gases.

Spirometry and Pressure Monitoring

Flow sensors or an externally attached monitor measure parameters including inspiratory and expiratory tidal volume and may graphically display flow-volume or pressure-volume loops. The flow sensors act to assure accuracy of tidal volume delivered by the ventilator and to measure spontaneous and manually delivered breaths. Periodic calibration may be necessary to assure accuracy.

Pressure monitoring is accomplished by manometry or use of pressure transducers. Values are shown on a gauge or on the screen display of the anesthesia machine. An important parameter to monitor is peak inspiratory pressure to avoid risk of barotrauma. Positive end-expiratory pressure (PEEP) is also monitored. Unusual variations in pressure may be due to breathing system resistance caused by obstructions, leaks or other mechanical factors and should be immediately addressed.

Scavenging Systems

Waste gas scavenging is essentially the collecting of excess gases used in administration of anesthesia or exhaled by the patient. Scavenging removes exhaled anesthetics from the operating room environment and vents them safely to an appropriate place with minimal risk of exposure to personnel. Hoses are attached to an interface between the outflow of the APL valve and the ventilator PRV, and to a central vacuum or evacuation system. Unique connections and fittings determined by the CGA are used to connect the scavenging system. Loss of active scavenging may cause unintended PEEP.

The anesthesia machine and monitors that make up the anesthesia workstation are not always intuitive and require expertise to manipulate. To accomplish the goals of this chapter, it is recommended that the nonanesthesia care provider become familiar with the manual (bag) ventilation system, the oxygen controls on the anesthesia machine and the auxiliary oxygen flowmeter. Mastering the use of these three components of the anesthesia workstation will be of great importance in managing environmental and equipment emergencies.

Nonanesthesia Workstation Oxygen Therapy Modalities

In addition to supplying oxygen from an anesthesia machine into the patient breathing circuit, it important to review some of the more commonly used oxygen delivery devices ^[17]. Expertise at setting them up and connecting them to the patient greatly decreases the opportunity for hypoxic events. Selection of an oxygen therapy modality should be based on the severity of hypoxemia ^[18]. The following modalities will be discussed: nasal cannula, air entrainment mask, rebreather and non re-breather masks as well as T-pieces and tracheostomy masks for the spontaneously breathing patient with an artificial airway. The design, function, and use of the manual resuscitation bag will also be reviewed as it is the most important tool in emergent situations.

Nasal Cannula

Also known as nasal prongs, this modality is very commonly used (Figs. 6.29 and 6.30). Note that the prongs are curved and should be placed in the direction of the oropharynx to avoid blockage by contact with the nasal mucosa. Oxygen is delivered in flows of liters per minute to the patient. The maximum effective oxygen delivery flow rate is 6 L/min. Each liter incrementally adds approximately 4% to the inspired oxygen concentration For example, a nasal cannula with an oxygen flow of 2 L/min delivers approximately 28% oxygen to the patient. Nasal cannulae are used to treat mildto-moderate hypoxemia in a spontaneously breathing patient. Nasal cannulae can reliably deliver 40% FiO₂.



Fig. 6.29 Nasal cannula for oxygen administration

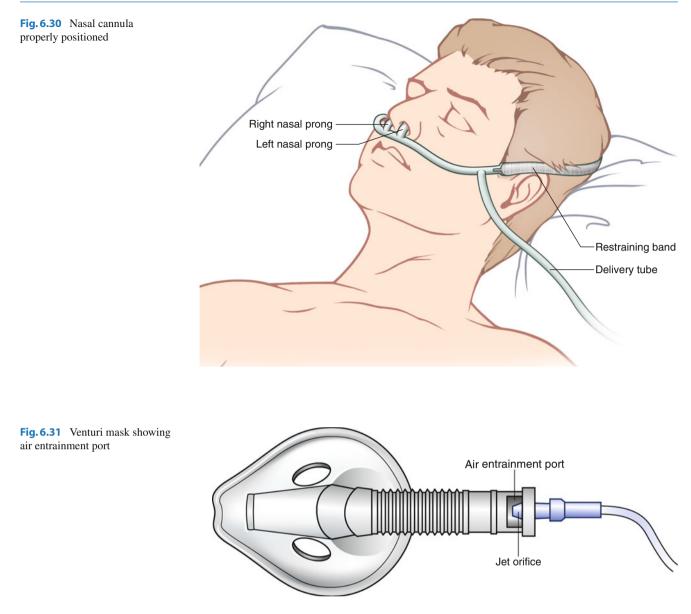
Air Entrainment Masks

Air entrainment masks, also known as venti or venturi masks, use the venturi principle to entrain air into an oxygen stream. In principle, a jet of oxygen of a specific diameter at a specific L/min flow entrains air through a fixed sized orifice to achieve delivery of a specific concentration of oxygen (Fig. 6.31).

Color coded adapters provide delivery of a specific oxygen concentration (FIO₂) when connected to oxygen flowing at a specific flow rate (Fig. 6.32). Venturi masks can deliver a maximum of 50% oxygen (see Table 6.1).

Rebreather and Non-rebreather Masks

These masks are easily identified by the attached reservoir bag and can deliver high concentrations of oxygen. They are indicated for use when severe hypoxemia occurs (Fig. 6.33). The re-breather bag is aptly named because the patient may re-breathe exhaled gas into the reservoir. It can deliver an oxygen concentration of greater than 80%. The non rebreather bag does not allow the re-breathing of exhaled gas because there is a one-way valve between the mask and the reservoir bag. It is the only mask capable of delivering 100% oxygen. To achieve delivery of high concentrations of oxygen, these masks should be used with oxygen flows of 15 L/ min. It should be noted that the reservoir bag must remain inflated to ensure high concentrations of oxygen.



Oxygen Modalities for Patients with Artificial Airways

Although infrequent, it may be necessary to administer oxygen to a spontaneously breathing patient who is tracheally intubated or who has a tracheostomy. This may be accomplished by using a T-piece (Fig. 6.34). This is an adapter that connects to the tracheal tube, to an oxygen source, and to the atmosphere via large bore corrugated tubing. It is an open system that allows the patient to exhale spontaneously into the atmosphere. During the expiratory pause, the corrugated tubing provides a reservoir of oxygen that helps to prevent the entrainment of room air during the subsequent inspiration which might dilute the concentration of inspired oxygen.

Figure 6.35 shows the Trach Tee Adapter (Hudson RCI Products, Research Triangle Park, NC). This is designed with a reservoir and an adapter for oxygen tubing specifically for the patient with an artificial airway. These T-pieces can be used both by a tracheally intubated patient and a patient who has a tracheostomy.

Alternatively, a modality known as the tracheostomy mask may be used for a patient who has a tracheostomy tube (Fig. 6.36). It is attached around the neck and fits loosely over the tracheostomy tube. It is more comfortable because it is designed to minimize torque on the tube. For long-term



Fig.6.32 Venturi mask with color coded adapters for different inspired oxygen fractions (FIO,s)

Table 6.1	Color coded adapters for	Venturi masks (see Figs. 6.31 a	and
6.32)			

O ₂ (%)	Color	L/min
24	Blue	2
28	Yellow	4
31	White	8
35	Green	12
40	Pink	15
50	Orange	15

use, it should be used in conjunction with continuous aerosol humidification to avoid drying of the mucosa.

Manual Self-Inflating Resuscitator Bags

The manual self-inflating resuscitator bag (SIRB), commonly known as the "AMBU" bag from one of its manufacturers and "bag-valve-mask" in ACLS terminology, is indispensable in an emergency situation (Fig. 6.37). Resuscitators are used to ventilate and oxygenate a patient's lungs prior to tracheal intubation and when a mechanical ventilator fails or is not available. They are also used to provide ventilation during transport of tracheally intubated or apneic patients throughout the hospital. It is the mode of ventilation of choice during cardiopulmonary resuscitation and for any emergent situation such as equipment failure. Manual resuscitators have standard 15 mm internal/22 mm external diameter connections that fit a standard tracheal tube connector, or a cushioned facemask that may be used if the airway has not been secured.

Construction of the Manual Resuscitator

The manual SIRB may be a reusable device that requires cleaning and sterilization between patients but more commonly is a single patient use disposable item. It consists of the

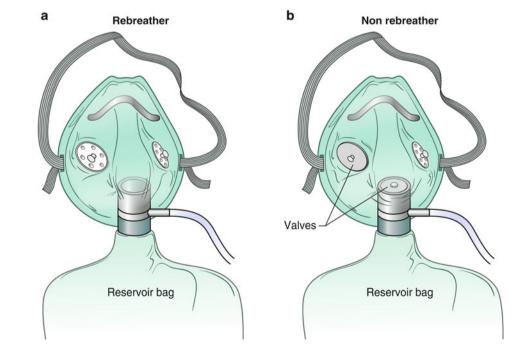


Fig. 6.33 Rebreather (**a**) and non-rebreather (**b**) oxygen facemasks



Colorimetric CO2 detector

Fig. 6.37 Self-inflating resuscitation bag (SIRB) also showing colorimetric carbon dioxide detector. Color changes from *purple* (no CO_2) on inspiration to *yellow* (CO₂ detected) on exhalation

Fig. 6.34 T-piece adapter for connecting to airway device, oxygen source, and expiratory limb to atmosphere



Fig. 6.35 Hudson Trach-Tee shown connecting to tracheostomy tube



Fig. 6.36 Tracheostomy mask

following parts: a self-inflating bag, an extendable oxygen reservoir, a non re-breathing valve, one-way valves to guarantee unidirectional flow through the bag (Fig. 6.38a), and optional items such as a pressure-limiting valve or positive end-expiratory pressure (PEEP) valve ^[19]. During re-expansion of the bag after it has been squeezed to deliver a breath, unidirectional flow draws fresh gas into the bag from the atmosphere (21% O₂) or from an oxygen supply source or reservoir (Fig. 6.38b). Oxygen-enriched air is then stored in the bag for delivery on the next breath (Fig. 6.38c). When the bag is squeezed, the one-way inlet valve from the reservoir closes, the fishmouth valve opens to the patient and at the same time occludes the exhalation pathway, thereby inflating the lungs. When the bag is released, the fishmouth valve closes to the patient and opens the exhalation pathway (Fig. 6.38b).

The volume of the resuscitator bag is manufacturer dependent and ranges from 1.1 to 2.2 L for adults and 0.2 to 0.9 L for infants and children. The estimated tidal volume is dependent on how forcefully the bag is compressed by the operator. For example, if the patient is known to be hypercapneic or hypoxic, more vigorous bag compressions are indicated to increase delivered tidal volume. The American Society for Testing and Materials (ASTM) recommends a minimum tidal volume capability of 600 mL for adult resuscitators, 70–300 mL for child resuscitators, and 20–70 mL for infant resuscitators.

Multiple factors may affect the tidal volume delivered by a resuscitator. Leaks are common when ventilating via a facemask. This may be rectified by having a second person to hold the mask in place and perform the head tilt/chin lift to maintain a patent airway. Technique and hand size are also variables that affect tidal volume. A recommendation is to use both hands when ventilating the lungs of an adult. Other variables include the patient's total thoracic compliance and airways resistance. Airway obstruction will limit the ability

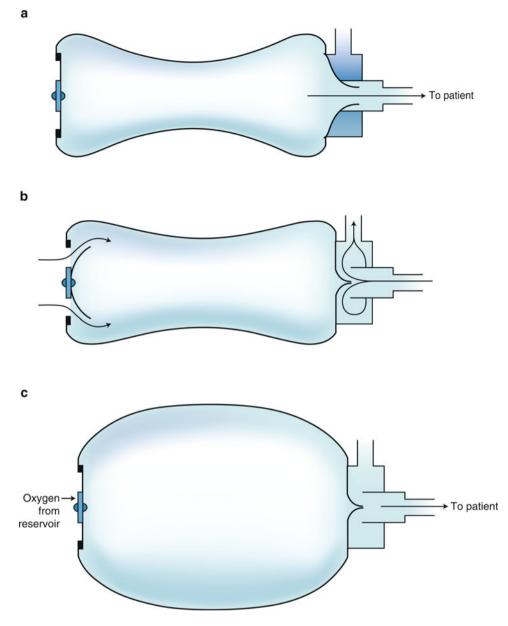


Fig. 6.38 Schematics of SIRB. (a) Bag squeezed=Inflation of lungs as the fishmouth valve opens to patient and closes exhalation path. (b) Bag released=exhalation as the fishmouth valve closes to patient

and opens exhalation pathway. Bag re-expands and fills with oxygen from reservoir via one-way valve. (c) Between breaths

to ventilate by bag. Bags with preset pressure-limiting valves (usually set to 40 cm H_2O) may provide insufficient ventilation in situations of increased airway resistance. Some manufacturers make bags without this valve or with pressure-limiting valve overrides. Pressure-limiting valves are not recommended for adult resuscitation bags because adequate ventilation may require high inflation pressures, especially in emergent situations such as CPR. Pressure-limiting valves are, however, recommended for routine use with infant and pediatric resuscitators to reduce the risk of barotrauma. An optional feature that is dependent on the bag's design is a port for connection to a pressure manometer to measure inflation pressures. During ventilation with a resuscitator, particularly in an emergent situation, a conventional capnograph instrument may not be available to confirm breath-by-breath CO_2 elimination. A resuscitator is now available that incorporates a colorimetric CO_2 detector in its design (Capno-FloTM Single-Patient-Use Resuscitation Bag, Covidien, Cardinal Health, Dublin, Ohio) (Fig. 6.37). Placed between the resuscitator and the patient's airway device/facemask, the CO_2 detector changes color from purple to yellow when CO_2 passes through it during exhalation, and back to purple during inspiration when CO_2 -free gas is being delivered ^[20]. The CO_2 detector is specified by the manufacturer to deliver continuous monitoring for up to 2 h.

Assuring Adequate Oxygen Delivery

SIRBs do not require oxygen to fill the volume of the bag. The bag must be connected to an oxygen source such as a flow meter to deliver supplemental oxygen. Ideally, high concentrations of oxygen (80-100%) are required during emergent situations such as CPR or desaturation. The bag needs to be connected to an oxygen flow meter set at 10-15 L/ min for adults and 5-10 L/min for children to achieve increased FIO2. To achieve delivery of 100% oxygen, the bag's design should include an expandable reservoir (Fig. 6.39). The reservoir's design varies by manufacturer and is normally attached to the rear of the bag. Two common designs include corrugated tubing or a reservoir bag. In order to deliver 100% oxygen, the reservoir must be least the same volume as the bag. It is common to find corrugated tubing reservoirs that are expandable. Pulling on the corrugated tubing to expand the length may double the size of the reservoir. Squeezing the bag delivers the breath to the patient. When re-expanding, gas is drawn into the bag through the reservoir. The system will only deliver high concentrations of oxygen when the reservoir is fully expanded. It is very important to be familiar with the design of the bag's reservoir to maximize oxygen delivery. It is also important to test the SIRB for correct functionality before using it on a patient. If the inspiratory valve is incompetent, the SIRB will not be able to develop pressure to ventilate the patient's lungs. If the exhalation port is obstructed, then oxygen will enter the patient's lungs but be unable to leave and may cause barotrauma.

Jet Ventilation

Jet ventilation, either by a manual jet apparatus or by mechanical ventilator, is an important adjunct to the anesthesia workstation. There are two indications for jet ventilation in the perioperative arena. In an emergency when an airway cannot be established and the patient's lungs cannot be ventilated, trans-tracheal jet ventilation should be considered as a life-saving measure ^[21]. Alternatively, jet ventilation may be used electively as the primary ventilation technique during rigid bronchoscopy, thoracic, laryngeal, and ENT surgeries. Each breath is delivered as a pulsed jet of high pressure gas through a cannula in or at the airway or attached to a device such as the bronchoscope. The size of each breath is determined by the frequency, pressure delivered and duration of inspiration. The term high-frequency jet ventilation is used for frequencies > 120 breaths or cycles per minute. The focus of this section will be to familiarize the reader with the equipment used for manual and mechanical jet ventilation.

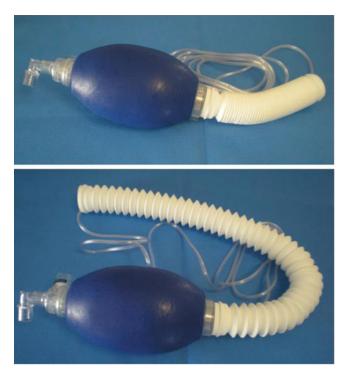


Fig. 6.39 SIRB with expandable reservoir (corrugated tubing)

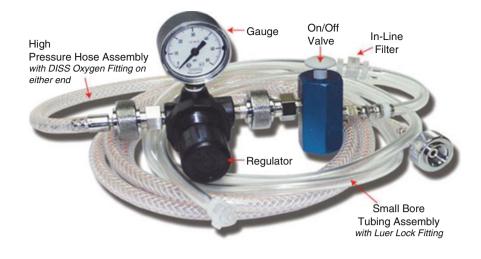
Manual Jet Ventilator

The apparatus consists of a high pressure gas hose, a variable pressure regulator, a gauge to monitor peak inspiratory pressures, an on/off trigger and tubing or a connector to attach to patient. The delivery tubing is then connected to the cannula or other device (Fig. 6.40).

It is important to be familiar with the configuration of the manual jet as ventilation using this device is completely dependent on the expertise of the user. The patient must be constantly observed. Chest movement should indicate successful ventilation. It is necessary to set desired peak inspiratory pressure on the regulator by triggering a breath and observing the manometer prior to attaching to the patient. The greatest safety concerns are barotrauma caused by excessively high pressures and impedance to exhalation due to airway obstruction.

Gas Source for Manual Jet Apparatus

The manual jet apparatus is a solely pneumatic device and requires a high pressure (50 psig) gas source to be able to deliver desired peak inspiratory pressures. A 50 psig oxygen outlet may be incorporated into the design of the anesthesia machine or located on the medical gas panel or boom where **Fig. 6.40** Manual jet ventilation apparatus



the anesthesia machine pipeline hoses are connected. The high pressure oxygen hose must be connected to this gas source. As discussed earlier, the connections for gas hoses are set by standards defined by the CGA. Unique connections (DISS or quick connects) allow this hose to be connected to an oxygen outlet only. Delivery of high oxygen concentrations is desired in emergent situations but may be contraindicated during routine use of the manual jet. For example, when using the manual jet apparatus electively during procedures in which a laser is used, it may be necessary to jet ventilate using much lower oxygen concentrations to avoid the risk of airway fires. To accomplish this, an oxygen/air mixer or blender is required so that specific oxygen concentrations may be set. Mixers require two high pressure gas sources, both medical air and oxygen, that feed a central controller with an outlet to deliver the specified oxygen concentration.

If a particular site is designated for elective laser cases that will regularly use manual jet ventilation, it would be prudent to permanently install a blender set-up or consider a mechanical jet ventilator.

Mechanical Jet Ventilators

Mechanical jet ventilators offer many advantages over manual jets for elective applications especially during long cases (Fig. 6.41). They include the ability to adjust FIO_2 , monitor ventilatory pressures accurately and improve safety by incorporating integrated alarm systems. Precisely controlled regular breaths will be delivered at regular intervals ^[22]. Manual jet ventilators require constant hands-on attention and monitoring by the anesthesiologist. A huge advantage of the



Fig. 6.41 Mistral II automatic jet ventilator (Acutronic Medical Systems Susquehannah Micro Inc., Windsor, PA)

mechanical jet is that it frees the anesthesiologist to be able to independently perform other necessary tasks to maintain the anesthetized patient. Users of this ventilator need specialized education and training to operate the device safely. Parameters to be set include the following:

- FIO₂
- Drive pressure (cm H₂O)
- Inspiratory time (%)
- Frequency
- · Alarms such as peak inspiratory pressure limit.

Parameters and alarm settings vary by design of different manufacturers. Only skilled clinicians who have been trained on the particular device and have demonstrated competency will be able to ventilate accurately and safely.

Managing Emergent Situations

The possible scenarios that create emergencies and unsafe conditions are many. Intraoperative fires ^[23], gas system problems, and electrical failures all have been reported. Even with proper preventative maintenance, equipment failure may occur. One of the most important responsibilities of the anesthesiologist is dealing with unplanned events. More commonly, these include managing acute physiologic abnormalities due to various causes but also emergencies related to equipment and the operating room environment. In the unlikely event that the anesthesia care provider is unavailable or absent, all members of the clinical team need to be able to assess and manage these situations to ensure patient safety and to minimize the risk of adverse outcomes. In order to accomplish this goal, critical thinking along with expertise in manipulating equipment controls is necessary. For the anesthetized patient, prolonged hypoxemia due to any cause must be avoided or corrected. The skills needed to accomplish this are few-understand how to increase oxygen concentration and how to manually ventilate the patient's lungs using either the anesthesia breathing system on the workstation or the self-inflating resuscitation bag.

Conclusion

Familiarity with equipment and calm quick thinking are keys in managing emergent situations such as electrical and equipment failures. It is also important to be able to quickly increase oxygen delivery if the patient becomes hypoxemic under normal conditions when equipment is functioning normally. Systems for medical gas delivery to the operating rooms have been reviewed along with basic oxygen therapy modalities as well as design and use of the versatile manual resuscitator. The various components of the anesthesia carestation have been discussed to familiarize the reader with methods to deliver oxygen and maintain ventilation. The focus of this chapter is to assure knowledge to ventilate and oxygenate anesthetized patients when systems fail, especially in the absence of an anesthesiologist. There are two options for doing this-using "bag" mode on the anesthesia machine or a manual resuscitator connected to a functional oxygen source. Ability to use and manipulate these systems will maximize patient safety, and assure adequate oxygenation and ventilation.

General Reading

1. Cairo J, Pilbean S. Mosby's respiratory care equipment. 8th ed. St. Louis: Mosby; 2009.

- Wilkins R, Kacmarek R, Stoller, J. Eagan's fundamentals of respiratory care, 9th ed. St. Louis: Mosby; 2009.
- Dorsch JA, Dorsch SE. Understanding anesthesia equipment, 5th ed. Philadelphia: Lippincott Williams & Wilkins; 2008.
- Al-Shaikh B, Stacey J. Essentials of anesthesia equipment, 3rd ed. St. Louis: Mosby; 2007.

References

- American Society for Testing and Materials. Standard specification for particular requirements for anesthesia workstations and their components. ASTM F–1850–00. West Conshohocken: ASTM; 2000.
- Eisenkraft JB. Hazards of the anesthesia delivery system. In: Rosenblatt MA, Butterworth JB, Gross JB, editors. ASA refresher courses in anesthesiology, vol. 37. Philadelphia: Lippincott-Raven, Williams & Wilkins; 2009. p. 37–55.
- 3. The American Society of Anesthesiologists (ASA) Standards For Basic Anesthetic Monitoring. ASA, Park Ridge, IL. Approved by the ASA House of Delegates on October 21, 1986, and last amended on October 20, 2010 with an effective date of July 1, 2011. Available at: http://www.asahq.org/For-Members/Clinical-Information/~/media/ For%2520Members/documents/Standards%2520Guidelines%252 0Stmts/Basic%2520Anesthetic%2520Monitoring%25202011. ashx. Last accessed 20 Nov 2011.
- Schumacher SD, Brockwell RC, Andrews JJ. Bulk liquid O₂ supply failure. Anesthesiology. 2004;100:186–9.
- Weller J, Merry A, Warman G, Robinson B. Anaesthetists' management of oxygen pipeline failure: room for improvement. Anaesthesia. 2007;62:122–6.
- Mudumbai SC, Fanning R, Howard SK, Davies MF, Gaba DM. Use of medical simulation to explore equipment failures and human-machine interactions in anesthesia machine pipeline supply crossover. Anesth Analg. 2010;110:1292–6.
- Branson R, Hess D, Chatburn R. Respiratory care equipment. 2nd ed. Philadelphia: Lippincott; 1999. p. 28–34.
- Branson R, Hess D, Chatburn R. Respiratory care equipment. 2nd ed. Philadelphia: Lippincott; 1999. p. 39–47.
- 9. Branson R, Hess D, Chatburn R, editors. Respiratory care equipment. 2nd ed. Philadelphia: Lippincott; 1999. p. 7–14.
- Compressed Gas Association. Standard for compressed gas cylinder valve outlet and inlet connections (CGS V-1). Chantilly, VA: 2005.
- Dorsch JA, Dorsch SE. Understanding anesthesia equipment. 5th ed. Philadelphia: Lippincott Williams & Wilkins; 2008. p. 83–118.
- Eisenkraft JB. Anesthesia vaporizers. In: Ehrenwerth J, Eisenkraft JB, editors. Anesthesia equipment: principles and applications. St. Louis: Mosby Year Book; 1993.
- Eisenkraft JB. Respiratory gas monitoring. In: Reich DL, Kahn RA, Mittnacht AJC, Leibowitz AB, Stone ME, Eisenkraft JB, editors. Monitoring in anesthesia and perioperative care. New York: Cambridge University Press; 2011. p. 150–170.
- Dorsch JA, Dorsch SE. Understanding anesthesia equipment. 5th ed. Philadelphia: Lippincott Williams & Wilkins: 2008. p. 121–89.
- Riutort K, Eisenkraft JB. Anesthesia workstations and delivery systems. In: Barash PG, Cullen BF, Stoelting RK, Cahalan N, Stock MC, editors. Clinical anesthesia, 7th ed. Elsevier: Philadelphia; 2012.
- Dorch JA, Dorsch SE. Understanding anesthesia equipment. 5th ed. Philadelphia: Lippincott Williams & Wilkins: 2008. p. 223–75.

- Branson R, Hess D, Chatburn R. Respiratory care equipment. 2nd ed. Philadelphia: Lippincott; 1999. p. 63–77.
- AARC: Clinical Practice Guideline. Oxygen therapy for adults in the acute care facility—2002 revision and update. Respir Care. 2002;47:717–20.
- Branson R, Hess D, Chatburn R. Respiratory care equipment. 2nd ed. Philadelphia: Lippincott; 1999. p. 193–200.
- Bhende MS, Allen Jr WD. Evaluation of a Capno-Flo resuscitator during transport of critically ill children. Pediatr Emerg Care. 2002;18:414–6.
- 21. Patel R. Percutaneous transtracheal jet ventilation: a safe, quick, and temporary way to provide oxygenation and ventilation when conventional methods are unsuccessful. Chest. 1999;116: 1689–94.
- 22. Evan E, Biro P, Bedforth N. Jet ventilation. Contin Educ Anaesth Crit Care Pain. 2007;7:2–5.
- American Society of Anesthesiologists Task Force on Operating Room Fires. Practice advisory for the prevention and management of operating room fires. Anesthesiology. 2008;108: 786–801.

Preoperative Assessment and Optimization

Jaime B. Hyman and Andrew B. Leibowitz

Introduction

Intraoperative anesthetic care is dictated by a number of factors. An understanding of the patient's medical status and reserve is critical in the planning and administration of a successful anesthetic that idealizes surgical conditions while optimizing patient safety. The preoperative evaluation and medical optimization of the patient presenting for otolaryngology procedures is central to the role of the anesthesiologist, and it is essential that all otolaryngologists have a working knowledge of the concepts and importance of the preoperative patient assessment. While the focus of this chapter is medical in nature, the psychological and potential medical-legal importance of a thorough preoperative visit cannot be overstated. Most patients admit to being more frightened of the anesthetic than the surgery and the ability of the anesthesiologist to gain the patient's trust and alleviate their anxiety while simultaneously obtaining a pertinent anesthetic history and physical, and informing them of the likely risks and benefits of the proposed anesthetic and monitoring, is equally important as administering sound intraoperative care.

Every written preoperative evaluation should conclude: (1) the patient's American Society of Anesthesiologists (ASA) classification; (2) the planned anesthetic (e.g., general, MAC, regional); (3) the requirements for special monitoring (e.g., arterial catheter, central line); (4) the likely need for blood products; and (5) the possibility of needing postoperative critical care monitoring and mechanical ventilation.

Preoperative Medical History

A focused medical history is the cornerstone of preoperative evaluation and should establish the patient's general state of health, present illness, exercise tolerance, current

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medications, drug allergies, surgical and anesthetic history, history of tobacco, alcohol, or other drug use and a physical exam including an airway assessment. While not specific to patients undergoing otolaryngologic procedures, a facile working knowledge of the ASA classification system is an important place to start regarding preoperative assessment. In essence, it rapidly distils the most important aspects of the preoperative evaluation into a numerical short hand that codifies the patients overall condition, which in turn is often used to stratify overall perioperative risk independent of the procedure planned (see Table 7.1). Although this classification system does not quantify risk, it is widely used in virtually all anesthesiology databases and investigations and is also frequently employed to decide on postoperative triage.

Cardiovascular Disease

Ischemic Heart Disease

Perioperative cardiac complications are among the most common and concerning potential risks of surgery and anesthesia. The standard of care for preoperative cardiac evaluation is established by the 2007 update of the American College of Cardiology/American Heart Association (ACC/ AHA) guidelines on Perioperative Cardiovascular Care and Evaluation for Noncardiac Surgery ^[1]. The guidelines assist in deciding on appropriate preoperative workup and testing, but local practice and adherence to these guidelines varies tremendously. They take into consideration three variables: the patient's history, exercise capacity, and relative risk of the planned surgery (see Fig. 7.1).

Emergency surgery by necessity must proceed with the best risk factor management possible given the time constraints. Patients presenting for nonemergency surgery with active cardiac conditions (e.g., unstable coronary syndromes, decompensated heart failure, significant arrhythmias, or severe valvular disease) should have their procedure postponed until the condition is optimized. If these active cardiac conditions are excluded, the next consideration is the risk of

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Physical status classification	Description
ASA-PS1	A healthy patient
ASA-PS2	A patient with mild systemic disease that results in no functional limitations
ASA-PS3	A patient with severe systemic disease that results in functional limitations
ASA-PS4	A patient with severe systemic disease that is a constant threat to life
ASA-PS5	A moribund patient who is not expected to survive without surgery
ASA-PS6	A declared brain dead patient whose organs are being procured for donor purposes
Emergency (E)	Any patient in whom an emergency surgery is required. This is in addition to one of the above six classifications.

 Table 7.1 American Society of Anesthesiologists physical status classification

Available from www.asahq.org

surgery. For low-risk procedures (e.g., superficial lymph node removal), it is unnecessary to perform further workup. For moderate and high-risk surgeries, the patient's functional capacity should be assessed, and if adequate (i.e., ability to perform four METS or greater, equivalent to climbing two flights of stairs without symptoms), no further workup or treatment should be necessary.

If functional capacity is limited or cannot be assessed, then the revised cardiac risk index (RCRI) (Table 7.2)^[2] may help to determine whether further testing is indicated.

If none, or only one (unless that one is known coronary artery disease) of these clinical predictors is present, the patient is at very low risk of perioperative morbidity or mortality and no cardiac workup or testing is indicated. Given that the ACC/AHA guidelines consider most otolaryngology procedures as "intermediate" risk, if two or more of these clinical risk factors are present, heart rate control with a beta blocker, and, or, further testing should be considered, but are not uniformly recommended in complete absence of symptomatic cardiac disease. Testing will usually first entail either a stress echocardiogram or nuclear stress test potentially followed by cardiac catheterization if a significant abnormality is found.

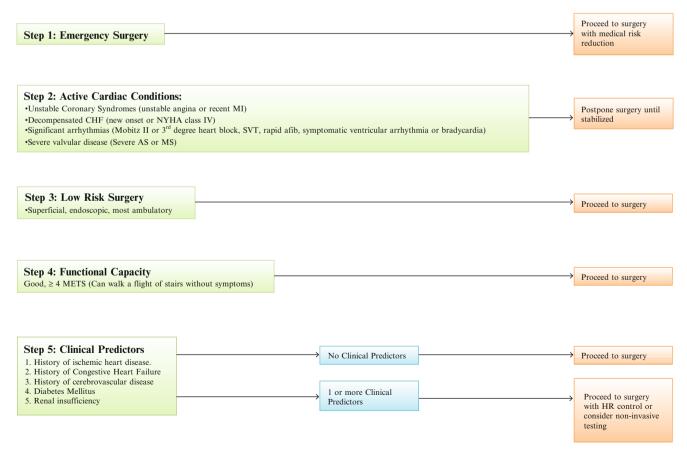


Fig. 7.1 Cardiac evaluation for noncardiac surgery. Adapted from J Am CollCardio, 150, Fleisher LA, Beckman JA, Brown KA, et al., ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation

and care for noncardiac surgery, pp. e159-e241, copyright 2007, with permission of Elsevier

Table 7.2 Revised Cardiac Risk Index		
High-risk surgery (intraperitoneal, intrathoracic, or suprainguinal vascular procedures)		
Ischemic heart disease		
History of congestive heart failure		
History of cerebrovascular disease		
Diabetes Mellitus requiring insulin		
Creatinine>2 mg/dl		

From Lee TH, Marcantonio ER, Mangione CM, et al.: Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. Circulation 100:1043-1049, 1999

The use of the RCRI can help determine the need for further cardiac testing. In the original study of the RCRI, there was a direct correlation with RCRI factors and cardiac events. The percentage incidence of major cardiac events including cardiac death, nonfatal MI, and nonfatal cardiac arrest was 0.4%, 0.9%, 7%, and 11% with zero, one, two, or three risk factors, respectively. More recently a Medicare database review reported a mortality rate (not restricted to only cardiac related death) of 1.4%, 2.2%, 3.9%, 5.8%, and 7.4% with zero, one, two, three, or four, or more factors, respectively ^[3]. While more germane to the following section, the reader should be aware that it was this study that concluded that the presence of two or more factors should be an indication to start preoperative beta blockade. Patients with less than two factors appeared to have an increased mortality rate with the administration of beta blockers.

Perioperative Beta Blockade

Inadvertent discontinuation of beta blockers should be avoided. Patients who are already taking beta blockers should continue them perioperatively to avoid rebound hypertension and tachycardia that will lead to increased myocardial oxygen demand and potentially myocardial ischemia, myocardial infarction or sudden cardiac death. Beta blockers may also lead to thrombus stabilization and their discontinuation may promote plaque rupture and hemorrhage.

Initiation of beta blockers preoperatively in patients at risk for cardiac complications is a much debated topic with recent clinical trials yielding conflicting results, making it difficult to develop a consensus recommendation. The perioperative setting offers a unique chance to intervene on chronic medical conditions in a patient who may not otherwise seek medical care. For this reason, if a chronic condition, such as a history of MI, congestive heart failure, stable angina, or a known positive stress test exists, beta blockade therapy is indicated independent of the perioperative setting based on ACC/AHA guidelines ^[1,4,5].

In the absence of a purely medical indication, but with known cardiac risk factors, the largest prospective randomized clinical trial to date examining the benefits and risks of initiating perioperative beta blockade was the PeriOperative Ischemic Evaluation (POISE) trial [6]. Inclusion criteria consisted of age >45 and a history of CAD, PVD, stroke, or hospitalization for CHF within the past 3 years, or three of the following risk factors: intrathoracic, intraperitoneal, or major vascular surgery, history of CHF, history of DM, history of TIA, serum creatinine > 1.75, age > 70, or emergency or urgent surgery. Patients were randomized to orally receive 100 mg of controlled-release metoprolol 2-4 h before surgery and 200 mg for 3 days postoperatively, or placebo. The primary outcome was a composite of cardiovascular events including primary cardiac death, nonfatal MI, and nonfatal cardiac arrest. The metoprolol group had lower rates of myocardial infarction and need for cardiac revascularization postoperatively, but there were more deaths and strokes than in the placebo group. Based on this trial, the popularity of widespread use of beta blockade to reduce perioperative cardiac risk was called into question, although critics of this trial cite the relatively large dose of beta blocker used as responsible for these negative findings.

A large retrospective study briefly discussed above reviewed 782,969 patients who had undergone noncardiac surgery and found that for patients with a RCRI score of 0 or 1, beta block-ade treatment was associated with no benefit and possible harm, whereas among the patients with a score of 2, 3, or 4 or more the odds ratios for death in the hospital were 0.88, 0.71, and 0.58, respectively for those treated with beta blockers ^[3]. The most recently published DECREASE-IV trial looked to further clarify the relative benefits of beta blockade in the population at intermediate risk (defined as a risk of 1–6% for perioperative cardiac events) and found a significant reduction in 30-day cardiac death and nonfatal MI in a group treated with bisoprolol ^[7].

Given the mixed results of these recent studies, it seems reasonable to initiate beta blockade in patients with three or more nonoperative RCRI factors for typical otolaryngologic surgery with a target heart rate less than 60 beats per minute, but exert caution to avoid hypotension secondary to overzealous beta blocker administration. If the patient has two RCRI factors the data supporting routine beta blockade is less convincing and the administration of beta blockers may be reasonably reserved for patients with tachycardia and or hypertension as judged by clinicians caring for the patient.

Hypertension

Despite the fact that 25% of adults in the USA have hypertension and it is the most common preexisting condition in patients presenting for surgery, there is little data available regarding its perioperative management. There has never been definitive evidence that hypertension without evidence of end-organ damage poses an increased risk for perioperative cardiac complications. A 2004 meta-analysis of 30 observational studies yielded a pooled odds ratio for an association between hypertension and perioperative cardiac risk of 1.35^[8]. The authors of the meta-analysis conceded that this odds ratio is small and must be interpreted with caution given the heterogeneity of the observational studies with no correction for confounding variables. In the context of a low event rate, this odds ratio probably represents a clinically insignificant association between a diagnosis of hypertension and perioperative cardiac risk despite statistical significance.

When patients with severe hypertension are examined independently in limited perioperative observational studies there is an association between markedly elevated blood pressures (i.e., >180 mm Hg systolic or >110 mm Hg diastolic) and ischemia, arrhythmia, and cardiovascular lability. It is therefore frequently recommended that elective surgery be delayed for severe hypertension until the blood pressure is below 180/110 mm Hg; however, there is no clear evidence that deferring surgery to correct elevated pressure actually reduces risk. Overzealous blood pressure correction may in fact lead to coronary or cerebral ischemia and perioperative treatment should be limited to a reduction of only 20% from baseline.

There are a few general recommendations regarding the management of antihypertensive medications in the perioperative period:

- Diuretics should usually be held on the morning of surgery because of the potential for hypovolemia and electrolyte disturbances ^[9].
- 2. Angiotensin converting enzyme (ACE) inhibitors and angiotensin receptor blockers (ARBs) are known to intensify the hypotensive effects of anesthesia induction ^[10], though this can be treated with fluids and alpha-agonists and/or vasopressin, and should be expected. Although there is no consensus to withdraw ACE inhibitors and ARBs routinely in the perioperative period, several authors recommend that they should not be taken on the day of surgery and if a prolonged high blood loss procedure is planned (e.g. extensive cancer case with microvascular free flap) they should be avoided the night before surgery too. It is important to note that many patients are on these medications for renal or myocardial protection in the face of diabetes mellitus or congestive heart failure, respectively, and not hypertension.
- 3. Calcium channel blockers, beta blockers, and central acting alpha-agonists such as clonidine should be continued without interruption in the perioperative period. Beta

blockers (see above) and clonidine in particular can be associated with rebound hypertensive withdrawal symptoms if discontinued and it should be reinforced with the patient to continue these medications up to and including the day of surgery as per usual routine.

Pulmonary Disease

Asthma

Asthma is a reversible and inducible chronic inflammatory condition characterized by airway hyperreactivity and bronchoconstriction. Several perioperative factors can lead to its exacerbation ^[11]. General anesthesia causes alteration in diaphragmatic function, impaired cough, and decreased mucociliary function ^[12]. Tracheal intubation and airway manipulation during anesthetic management and surgery may lead to bronchoconstriction ^[13].

It is important to ascertain whether prior tracheal intubation, hospital admission, or emergency room visitation have been required. This information along with the history of pulmonary complications with previous surgeries will immediately categorize the general severity of the patient's asthma and likelihood of a perioperative exacerbation. A history regarding usual triggers, steroid use, and other medication use must also be obtained. In order to ascertain the patient's current status, a focus on the accelerated need for bronchodilators or recent steroid regimens, and evidence of recent upper respiratory tract infections or pneumonia must also be determined.

Every asthmatic should have their lungs carefully auscultated to determine the presence or absence of wheezing at baseline. Chest radiographs or pulmonary function tests (PFTs), in the absence of signs or symptoms of an asthmatic exacerbation or a suspicion of pneumonia, are not generally indicated or useful. If a patient's asthma symptoms are not at their baseline, then a course of inhaled or oral steroids may be indicated and the optimization of inhaled beta agonists and perhaps anticholinergics is recommended (see Table 7.3) [11]. Elective surgery should be deferred until the asthma exacerbation is treated and medically optimized.

Chronic Obstructive Pulmonary Disease

The term "Chronic Obstructive Pulmonary Disease" (COPD) is used to include chronic bronchitis and emphysema, is usually due to cigarette smoking or more rarely other airborne pollutants, and is characterized by excessive sputum

 Table 7.3
 Guidelines for preoperative medication administration in asthmatics

Asthma symptoms	Recommended action
No flairs in asthma over past year and not taking medications	• None
On bronchodilators only	 If spirometry/symptoms are at baseline initiate therapy with inhaled bronchodilator prior to surgery If spirometry is below baseline, or patient is symptomatic consider adding prednisone 0.5 mg/kg for 5 days prior to surgery
On inhaled corticosteroid chronically	 Continue inhaled corticosteroid Consider treating with prednisone 0.5 mg/kg for 5 days preoperatively and hydrocortisone 100 mg IV on the morning of surgery and every 8 h postoperatively until stable
On oral steroids chronically	 Increase dose of oral steroids for 5 days preoperatively. Treat with hydrocortisone 100 mg IV on the morning of surgery and every 8 h postoperatively until stable

Adapted with permission from Tirumalasetty J, Grammer LC. Asthma, surgery, and general anesthesia: a review. J Asthma 2006;43:251–4

production and obstruction to airflow that is not fully reversible with bronchodilators. The perioperative management of COPD is similar to that of asthma.

An assessment of the patient's current respiratory status relative to their baseline, questioning symptoms such as increased dyspnea, increased amount or change in the color of sputum, coughing, or wheezing is essential. Patients with advanced COPD should have their room air oxygen saturation noted. The perioperative treatment of COPD is virtually the same as for those not undergoing surgery, and is based on disease stage, as outlined in Table 7.4 ^[14]. However, they should be aggressively treated preoperatively to achieve their best possible baseline functional status. Counseling patients regarding the possible need for prolonged mechanical ventilation after surgery is important for surgeons and anesthesiologists alike.

Obstructive Sleep Apnea

Obstructive sleep apnea (OSA) is characterized by repetitive collapse of the pharyngeal airway during sleep, causing apnea or hypopnea and resulting in intermittent episodes of hypoxia and hypercarbia ^[15]. Factors that predispose to upper airway collapse and increase the likelihood of OSA include obesity (BMI>35 kg/m²), large neck circumference (>40 cm), male gender, and age >50. Uvulopalatopharyngoplasty is a

 Table 7.4 Recommended therapy for COPD based on disease severity

severity	
COPD disease stage	Recommended treatment
I: Mild • FEV₁/FVC <0.70 • FEV₁≥80% predicted	Short-acting bronchodilators as needed
 II: Moderate FEV₁/FVC<0.70 FEV₁ 50–80% predicted 	 Short-acting bronchodilators as needed Regular treatment with long acting bronchodilators Pulmonary rehabilitation as needed
 III: Severe FEV₁/FVC<0.70 FEV₁ 30–50% predicted 	 Short-acting bronchodilators as needed Pulmonary rehabilitation as needed Add inhaled glucocorticos- teroids if repeated exacerbations
 IV: Very severe FEV₁/FVC<0.70 FEV₁<30% predicted or FEV₁<50% pre- dicted + chronic respiratory failure 	 Short-acting bronchodilators as needed Pulmonary rehabilitation as needed Add inhaled glucocorticos- teroids if repeated exacerbations Add long-term oxygen therapy if chronic respiratory failure

Adapted from Klaus F. Rabe, Suzanne Hurd, Antonio Anzueto, et al., Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. Am J Respir Crit Care Med 2007;176:532–55

commonly performed otolaryngologic procedure used to treat OSA.

Patients and their families should be questioned about the presence of loud snoring, daytime somnolence, whether a partner has observed them stop breathing during sleep, and presence of hypertension. The presence of three or more of these features predicts a high risk of OSA [16]. Certain craniofacial abnormalities (retrognathia, micrognathia, brachycephaly, large uvula, large tonsils, macroglossia) can also predispose to pharyngeal obstruction. If a patient has had testing, then the results should be in the preoperative medical record and if the patient wears a device, the device, or at least the face mask, should be brought by the patient to the hospital. Also, the mode (Bilevel Positive Airway Pressure (BIPAP), or Continuous Positive Airway Pressure CPAP), and exact settings should be confirmed and noted for postoperative use. It should be appreciated that logistically the use of these devices may not be possible postoperatively due to nasal packing or other surgical issues (i.e. wounds or dressings). The presence of OSA has been shown to increase postoperative respiratory complication rates, thus requiring hospital admission and pulse oximetery monitoring for most patients requiring general anesthesia.

Although effective preoperative interventions to reduce risk for OSA patients are grounded in common sense, these authors suggest the following basic principles: (1) meticulous preoxygenation given that these patients may be more difficult to ventilate and intubate; (2) judicious use of intraoperative and postoperative opioids and other sedatives ^[17]; (3) considering using regional anesthesia for the principle anesthetic technique and for postoperative pain management; (4) extubate only when the patient is wide awake, easily follows commands and appears to have airway reflexes completely intact; (5) close monitoring of oxygen saturation in the PACU; (6) postoperative admission to a step-down unit where oxygen saturation can be monitored; (7) continuation of preoperative CPAP/BiPAP use; (8) plan to admit most patients for overnight observation. OSA is covered in more detail in Chap. 9.

Endocrine Disease

Diabetes Mellitus

The Overview

In 2007 nearly 8% of the population of the USA had diabetes. The incidence of diabetes, specifically type 2 diabetes that reflects 90–95% of all cases, is on the rise and is generally associated with age, obesity, genetics, and inactivity. The preoperative evaluation of patients with diabetes mellitus (DM) should focus on related organ damage, specifically delayed gastric emptying, autonomic dysfunction, syncope, peripheral neuropathy, and nephropathy. It is also particularly important to employ the principles of cardiac risk assessment discussed previously because coronary artery disease is more prevalent in diabetics and is frequently silent.

To assess glucose control, the range of blood glucose levels and a hemoglobin A1C level can be obtained. Care to avoid hypoglycemia and hyperglycemia should be exercised in the face of perioperative metabolic derangements. Hypoglycemia can cause neurocognitive defects, including coma, and more rarely arrhythmia or seizures. Hyperglycemia can lead to volume depletion from osmotic diuresis, increase the risk of wound infection, and in extreme instances lead to ketoacidosis in type 1 diabetics and nonketotic hyperosmolar state in type 2 diabetics.

The Evidence

To date, studies performed to examine the role of tight glucose control on outcome provide conflicting results.

In a trial published in 2001, very tight glucose control (80-110 mg/dl) in a cardiac surgical ICU patient population usually given intravenous parenteral nutrition resulted in a significant decrease in morbidity and mortality [18]. However, subsequent randomized controlled trials, including the largest published to date, the Normoglycemia in Intensive Care Evaluation-Survival Using Glucose Algorithm Regulation (NICE-SUGAR), a multicenter, multinational randomized controlled trial that evaluated glycemic control in 6,104 critically ill patients, concluded that mortality was significantly higher in the intensively controlled group. Severe hypoglycemia was also more common in the very tightly controlled group ^[19] and many experts feel that the danger of hypoglycemia has been historically underestimated. Furthermore, there is a lack of studies examining aggressive glucose control in noncritically ill surgical patients.

The Recommendations

Based on the conflicting data it seems prudent to avoid tight glucose control in the perioperative patient who is expected to be NPO for prolonged duration. Tight glucose control places the patient at increased risk of incurring severe hypoglycemia that clearly outweighs the benefit of preventing hyperglycemia. The American Association of Clinical Endocrinologists and American Diabetes Association's most recent consensus statement, which has been updated since the NICE-SUGAR trial, recommends insulin therapy to target a blood glucose level goal of 140–180 mg/dl for inpatients ^[20]. Other professional societies have also increased the treatment threshold from 110 (based on the first study of intensive insulin therapy in 2001) to 180 mg/dl, although some guidelines have yet to reflect the more recent data ^[21]. The evolution of these guidelines suggests that further change is likely as more trials are conducted.

The American Association of Clinical Endocrinologists and American Diabetes Association's consensus statement also concludes that oral, long acting noninsulin diabetic medications are for the most part inappropriate in hospitalized patients who are not expected to consume meals at regular intervals. In particular, caution must be exercised with the use of metformin, which should be stopped the day prior to surgery, because of the potential for development of lactic acidosis if there is concomitant renal insufficiency, hemodynamic instability, or need for imaging studies with contrast radiographic. Therefore, diabetic patients who will be admitted postoperatively should usually have their oral hypoglycemic agents held and be covered with insulin as needed to maintain glucose levels below 180 mg/dl.

Adrenal Disorders

The Overview

Surgery is one of the most potent activators of the hypothalamic-pituitary-adrenal (HPA) axis, with the greatest cortisol secretion occurring during emergence from anesthesia, extubation, and in the immediate postoperative period presumably in response to pain ^[22]. Aside from the response to stress, excess adrenal hormone can result from pituitary tumors (Cushing's disease), adrenal tumors, adrenal hyperplasia, ectopic ACTH from neoplasms, and more commonly from exogenous steroids administered to treat asthma, COPD, rheumatologic disorders, and especially in otolaryngologic patients to reduce preoperative inflammation and postoperative swelling. Significant preoperative findings in patients with an excess of adrenal corticosteroids may include obesity, moon facies, and a buffalo hump which could affect airway management; easy bruising and friability of the skin such that mild pressure may cause hematoma, removing adhesive tape may tear the skin, and wound healing may be impaired. Hypertension, hyperglycemia, and electrolyte abnormalities may also be of concern and should be evaluated preoperatively.

Adrenal insufficiency, on the other hand, results from destruction of the pituitary or adrenal glands or from withdrawal of long-term exogenous administration of steroids. Symptoms with perioperative implications include weakness, hypotension, hypovolemia, orthostasis, and electrolyte abnormalities including hyponatremia and hyperkalemia.

Patients on chronic exogenous steroids may need replacement glucocorticoid therapy perioperatively to avoid these symptoms. In general, functional suppression of the HPA axis does not occur if a patient has received (1) glucocorticoids, regardless of the dose, for less than 3 weeks, (2) less than 5 mg/day of prednisone or its equivalent, (3) in patients treated with alternate-day therapy. These patients should continue their usual dose of glucocorticoids perioperatively. However, in patients taking prednisone at a dose greater than 20 mg/day or its equivalent for greater than 3 weeks, or those patients who have a Cushingoid appearance, then "stress dose" steroids are probably indicated and are unlikely to cause harm ^[23].

The Recommendations

Current consensus opinion recommends guidelines for glucocorticoid administration based on the known glucocorticoid response associated with the magnitude of stress seen with various surgeries. For minor surgical stress (e.g., tonsillectomy) patients can take their usual morning steroid dose

Corticosteroid	Approximate equivalent dose (mg)
Dexamethasone	0.75
Methylprednisolone	4
Prednisone	5
Hydrocortisone	20

and no extra supplementation is necessary. For moderate surgical stress (e.g., total thyroidectomy) patients should take their usual morning steroid dose and receive 50 mg of hydrocortisone intravenously with induction and 25 mg every 8 h for 24 h, resuming their usual dose on postoperative day two. For major surgical stress (e.g., radical neck dissection with blood loss) patients should take their usual morning steroid dose, receive 100 mg of hydrocortisone with induction, and 50 mg every 8 h for 24 h, followed by a taper of this dose by half per day until reaching their maintenance level ^[23]. Table 7.5 provides potency-dosage equivalents for various corticosteroids ^[24].

The preoperative considerations for patients with thyroid and parathyroid disease are covered in Chap. 14.

Kidney Disease

Patients with chronic kidney disease (CKD) are prone to anemia, fluid imbalances, electrolyte disturbances, coagulopathies, and drug excretion abnormalities. CKD places the patient at a significant risk for perioperative cardiac complications and is included in the ACC/AHA risk assessment algorithm previously discussed. The evaluation and history should focus on the patient's dialysis scheduling. Ideally it is recommended that dialysis be conducted on the day preceding surgery. If the patient receives hemodialysis, the access used for dialysis should be noted—tunneled percutaneous catheters and arm fistulas and grafts may both pose intraoperative challenges and limit the ability to accomplish intravenous access and arterial catheter placement. Arm fistulas and grafts are also prone to clotting when the arm is tucked too tightly or hypotension ensues.

Hematologic considerations for surgical patients with CKD include anemia from a lack of erythropoietin production by the kidneys and a qualitative platelet dysfunction especially seen in underdialyzed patients. Once on dialysis, however, patients may be more prone to a hypercoagulable state. Hyperkalemia is the most serious potential electrolyte disturbance and hypocalcemia is also common, with the eventual development of secondary hyperparathyroidism. Nonsteroidal Anti-Inflammatory Drugs (NSAIDS) interfere with autoregulation of renal perfusion and should be avoided in patients at risk for renal insufficiency. ACE inhibitors and ARBs are renal-protective in the long term but may worsen renal function acutely during episodes of hypoperfusion. Since many drugs are cleared by the kidneys, their duration of action can be prolonged with renal dysfunction. Interestingly, some commonly administered drugs (e.g., rocuronium, midazalom) have a longer duration of action in patients with kidney disease even though their renal excretion in healthy patients is minimally important. Of note, lowmolecular-weight heparins are cleared by the kidneys and are generally contraindicated because of the inability to titrate them correctly.

Hepatic Disorders

Since the liver has several major functions including synthetic, metabolic, and endocrine, hepatic disease and hepatic dysfunction places the patient at significant risk of metabolic, hematologic, pharmacologic, pulmonary, and neurologic derangements. Important issues to determine in patients with liver disease include the etiology; acute vs. chronic (e.g., viral hepatitides, alcoholic, primary sclerosing cholangitis, primary biliary cirrhosis) and severity. Severity is frequently quantified using the serum bilirubin and albumin, Prothrombin Time/International Normalized Ratio (PT/INR), and presence of ascites and encephalopathy. Particularly concerning to perioperative care is the presence of ascites and pleural effusions with resultant hypoxemia, encephalopathy, coagulopathy, and peripheral edema.

Reduction of ascites by sodium restriction, diuretics, or paracentesis preoperatively may improve pulmonary function in the unusual circumstance that this is a primary limiting factor. Cirrhotic patients undergoing surgery are at high risk of becoming encephalopathic. Constipation, infection, upper GI bleeding, uremia, alkalosis, and overuse of sedatives are known precipitating factors of encephalopathy. Patients with liver disease are particularly susceptible to anesthetic effects intraoperative and hemodynamic fluctuations and decreased hepatic blood flow can lead to worsening of liver function postoperatively ^[25]. Additionally, a number of factors can affect the metabolism of the drugs commonly used in the perioperative period. Hepatocellular dysfunction, cholestasis, altered drug binding, and decreased blood flow can all delay drug metabolism. It is prudent to decrease the dose of opioid analgesics, particularly morphine which is largely cleared hepatically, and monitor for side effects ^[25]. Fentanyl, however, is generally safe in routine doses. A decrease in pseudocholinesterase which is produced by hepatocytes can lead to prolonged activity of depolarizing neuromuscular blockade but is rarely clinically significant. Intubating doses of non-depolarizing neuromuscular blocking

Coagulopathy in patients with liver disease can be multifactorial and etiologies include thrombocytopenia due to hepatosplenomegaly, deficiency of clotting factors due to loss of synthetic function, or less importantly vitamin K deficiency as a result of inability to secrete bile. Vitamin K, fresh frozen plasma (FFP), or platelets may be used as necessary to correct deficiencies. Vitamin K takes 12-24 h to work even after administered intravenously and is rarely effective, the volume of FFP required is quite large and the effect fleeting, and the increase in platelet count after their administration is also relatively short lived. Although recommended for emergency situations and the acute reversal of anticoagulant agents, newer agents such as recombinant factor VIIa (rFVIIa) or prothrombin complex concentrate (PCC) may be useful and decrease the patients' exposure to large transfusion volumes. During long operations, point-of-care testing and thromboelastography may be helpful to guide replacement therapies.

Perioperative Management of Anticoagulation

Perioperative management of patients who require temporary discontinuation of anticoagulants must weigh the risk of a thrombotic event during interruption of the medication against the risk of bleeding related to the procedure.

Warfarin

The American College of Chest Physicians has published consensus guidelines, most recently updated in 2008, for the perioperative management of patients receiving antithrombotic therapy with warfarin ^[26]. These recommendations are based on stratifying patients on a risk scale from high to low risk according to their underlying indication for anticoagulation (e.g., patients with mechanical valve, atrial fibrillation, or venous thromboembolism) (see Table 7.6). For patients with mechanical valves, high-risk patients include those with any mechanical mitral valve, an older (caged-ball or tilting disc) aortic valve prosthesis, or a recent stroke or TIA. Patients with atrial fibrillation are stratified by the CHADS2 system, which assigns one point each for a history of CHF, Hypertension, Age>75, or Diabetes, and two points for history of Stroke or TIA^[27]. According to the American College of Chest Physicians consensus guidelines, patients are

Risk category	Mechanical heart valve	Atrial fibrillation	Venous thromboembolism
High	 Any mechanical mitral valve Older aortic valve (caged-ball or tilting disc) Stroke or TIA within past 6 months 	 CHADS2 score of 5 or 6 Stroke or TIA within past 3 months Rheumatic valvular heart disease 	VTE within past 3 monthsSevere thrombophilia
Moderate	• Bileaflet aortic valve plus one of the following: Atrial fibrillation, prior stroke/TIA, hyperten- sion, diabetes, heart failure, age >75	CHADS2 score of 3 or 4	 VTE within past 3–12 months Nonsevere thrombotic conditions Active cancer
Low	• Bileaflet aortic valve without other risk factors	 CHADS2 score of 0–2 (and no prior stroke or TIA) 	• Single VTE within past 12 months and no other risk factors

 Table 7.6
 Patient risk stratification for perioperative arterial or venous thromboembolism

Adapted with permission from Douketis JD, Berger PB, Dunn AS, et al. The perioperative management of antithrombotic therapy: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). Chest 2008;133:299 S–339 S

considered high risk for perioperative thromboembolism if they have a CHADS2 score of 5 or 6. Additionally, patients with a history of venous thromboembolism fall into the highrisk group if the event occurred within the previous 3 months.

According to these guidelines, all high-risk patients should receive bridging anticoagulation therapy with subcutaneous therapeutic-dose low-molecular-weight heparin (LMWH) or intravenous unfractionated heparin (IV UFH). Patients identified as moderate risk for thromboembolism should receive bridging anticoagulation with either therapeutic dose LMWH, IV UFH, or low-dose LMWH, and finally those at low risk should receive bridging anticoagulation with low-dose LMWH, or no bridging.

Warfarin is typically discontinued 5–7 days prior to surgery, with bridging therapy beginning 36 h after the last dose of warfarin. The last dose of LMWH should be administered approximately 24 h before surgery.

Antiplatelet Agents

There are a number of antiplatelet agents on the market and patients may present taking one or a combination of these agents simultaneously. Aspirin irreversibly inhibits platelet cyclooxygenase and if necessary should be stopped 7–10 days prior to surgery because the circulating platelet pool is replaced in this amount of time. Clopidogrel inhibits adenosine diphosphate receptor-mediated platelet activation and aggregation and if necessary should be stopped 7 days prior to surgery. Bleeding complications from clopidogrel are much more common than aspirin.

Patients on antiplatelet therapy because of coronary stents deserve special mention secondary to the high risk of thrombosis in the perioperative period if therapy is interrupted. Surgery results in a prothrombotic and inflammatory state and abrupt discontinuation of antiplatelet therapy can lead to a rebound effect marked by increased platelet adhesion and aggregation. When combined with an incompletely endothelialized drug-eluting stent, these factors can lead to stent thrombosis and acute myocardial infarction ^[28].

If interruption of antiplatelet therapy is desired from a surgical perspective, conservative recommendations are currently as follows. Following placement of a bare metal stent, non-urgent procedures should be delayed for at least 4–6 weeks, and following placement of a drug-eluting stent, non-urgent procedures should be delayed for at least 1 year. For patients with a recent drug-eluding stent placement in whom surgery cannot be delayed, aspirin and clopidogrel treatment should be continued without interruption if the stent was placed within the previous 6-12 months. If stenting occurred greater than 6 months prior, consideration can be given to continuing aspirin without interruption, continuing clopidogrel until 5 days before surgery and resuming therapy as soon as possible after surgery ^[1].

These situations are extremely high risk, guidelines have been rapidly changing, and the decision to withhold antiplatelet therapy preoperatively and resume antiplatelet therapy postoperatively is extremely challenging; therefore, it is the authors' strong recommendation to enlist the active involvement of a cardiologist to guide antiplatelet management in these patients.

Exposures and Substance Abuse

Tobacco Use

In 2000, 25.7% of males and 21.0% of females in the USA were smokers ^[29]. Tobacco use is of special concern for the otolaryngology patient, particularly for those undergoing head and neck cancer surgery. There are few studies examining the effects of smoking cessation on this patient

population in particular; however, data from general surgical patients can be extrapolated. Carbon monoxide and nicotine elimination occurs within 12-24 h of discontinuing smoking, and the major benefit in the immediate preoperative period is a decrease in carboxyhemoglobin content and better tissue oxygenation. There is also evidence that abstinence reduces the increased upper airway irritability of smokers and that 1-2 weeks of abstinence results in a significant reduction in sputum volume ^[30]. In general, the correlation between tobacco use and increased risk of postoperative wound infection [31-33] and cancer recurrence [34] is well established. Furthermore, the findings from several studies suggest that the cessation of smoking improves wound healing and reduces the risk of postoperative pulmonary complications ^[35], although there are studies with contradictory results showing no benefit to preoperative smoking cessation.

The Evidence

In a randomized trial using a preoperative smoking intervention program including counseling and nicotine replacement therapy for 6-8 weeks the wound related complication rate was 5% for the intervention group compared to 31% in controls (p=0.001) ^[36]. Another randomized controlled trial examining the effects of smoking and smoking cessation on wound infection randomized subjects to continued smoking, abstinence while using a nicotine patch, or abstinence with a placebo patch. Punch biopsy samples were taken lateral to the sacrum at 1, 4, 8, and 12 weeks after randomization and the sites were followed for development of wound complications. A statistically significant difference in the rate of wound infection was found between continued smokers and those who abstained starting at 4 weeks, and these differences continued to exist at 8 and 12 weeks. There was no difference in wound infection rates between abstainers with nicotine versus placebo patches [37].

Postoperative pulmonary complications were studied in a retrospective cohort study that evaluated patients undergoing pulmonary surgery according to smoking status and found that recent smokers (those who had smoked within 2–4 weeks of surgery) had an odds ratio of postoperative pulmonary complications of 2.44 compared with never smokers; however, this odds ratio fell to 1.03 for ex-smokers (defined as those who quit for >4 weeks before surgery) leading the authors to advocate a smoking cessation period of at least 4 weeks prior to surgery ^[38].

Contradictory studies exist including one where patients who stopped smoking in the weeks prior to surgery had a higher risk of postoperative pulmonary complications possibly secondary to transiently increased sputum production ^[39]. Other studies have found no difference between those who continue to smoke and those who abstain. One proposed reason for these discrepancies is that patient self-reporting may be inaccurate. A study using end-expired carbon monoxide and serum cotinine (a nicotine metabolite) noted that 50% of self-reported nonsmokers were found to be actively smoking ^[40].

The Recommendations

Overall, the available evidence overwhelmingly supports that cessation of smoking in the perioperative period is advantageous, and though the optimal preoperative duration of abstinence is not clear, it appears that the greater the duration the better, with at least 2 weeks, or preferably 4 weeks for best results. It is imperative that both anesthesiologists and surgeons take advantage of the perioperative period, when patients have a heightened awareness of their health to advise quitting and offer referrals to smoking cessation programs if for no other reason than the patient's general overall health ^[30].

Alcohol Use Disorders

Alcohol use disorders are very common among surgical patients, especially otolaryngology patients, and have important perioperative implications. One study found that 23% of general surgery admissions and 43% of otolaryngology admissions met criteria for alcohol abuse ^[35]. It is well established that acute alcohol intoxication reduces the amount of anesthesia needed by a patient whereas anesthesia and analgesia requirements are increased in chronic alcoholics due to cross-tolerance to both inhaled and intravenous anesthetics. Chronic alcohol users are also at risk for cardiomyopathy and arrythmias, immunosuppression, coagulation abnormalities, malnutrition, withdrawal syndromes, and poor wound healing ^[41].

The DSM-IV definitions of alcohol abuse and dependence are listed in Table 7.7. The preoperative diagnosis of alcohol use disorders allows the opportunity to offer interventions to reduce morbidity perioperatively.

Interventions for patients with alcohol use disorders but without alcohol dependence can include abstinence and referral for substance abuse counseling. A controlled trial of 42 patients with heavy alcohol consumption undergoing colorectal surgery randomized patients to 1 month of abstinence controlled with disulfiram versus continued drinking and found that the abstinence group suffered fewer myocardial infarctions, arrhythmias, infections, and hypoxemic episodes. The stress response during surgery as measured by heart rate, blood pressure, serum cortisol concentration, and

Table 7.7 DSM-IV definitions of alcohol abuse and dependence

Alcohol abuse:

A maladaptive pattern of use associated with one or more of the following:

- Failure to fulfill work, school, or social obligations
- Recurrent substance use in physically hazardous situations
- Recurrent legal problems related to substance use
 Continued use despite alcohol-related social or interpersonal problems

Alcohol dependence:

A maladaptive pattern of use associated with three or more of the following:

Tolerance

- Withdrawal
- · Substance taken in larger quantity than intended
- Persistent desire to cut down or control use
- · Time is spent obtaining, using, or recovering from the substance
- Social, occupational, or recreational tasks are sacrificed
- Use continues despite physical and psychological problems

plasma concentrations of glucose, interleukin 6, and catecholamines was higher in the group that continued drinking, and perioperative immunosuppression as measured by delayed type hypersensitivity was greater in this group as well^[42].

Alcohol-dependent patients may undergo preoperative detoxification and/or receive alcohol withdrawal prophylaxis. Initiation of withdrawal prophylaxis should begin upon cessation of alcohol consumption or admission to the hospital. Delaying the initiation of prophylaxis until the postoperative period places the patient at risk for developing withdrawal during or immediately after surgery, when perioperative stress is at its peak. Prophylactic regimens include diazepam 2.5–10 mg, lorazepam 0.5–2 mg, or chlordiazepoxide 5–25 mg every 6 h titrated by clinical status and not "tapered" until at least the third or fourth postoperative day ^[43]. All patients with a history of heavy alcohol use should receive daily multivitamins and thiamine (100 mg) preferably IM or IV during the perioperative period to prevent Wernicke–Korsakoff Syndrome.

Special Considerations

Do Not Resuscitate Orders

The policy of many institutions in the past was to simply suspend Do Not Resuscitate (DNR) orders in the perioperative period, as the administration of anesthesia necessarily involves some procedures that might be viewed as "resuscitation" in other settings. In 1993 the Ethics Committee of the ASA adopted guidelines for the care of patients with DNR orders that have since been updated ^[44]. These guidelines necessitate a preoperative discussion with the patient or surrogate and offer three alternatives:

- (a) Full Attempt at Resuscitation: The patient or designated surrogate may request the full suspension of existing directives during the anesthetic and immediate postoperative period, thereby consenting to the use of any resuscitation procedures that may be appropriate to treat clinical events that occur during this time.
- (b) Limited Attempt at Resuscitation Defined With Regard to Specific Procedures: The patient or designated surrogate may elect to continue to refuse certain specific resuscitation procedures (e.g., chest compressions, defibrillation, or tracheal intubation). The anesthesiologist should inform the patient or designated surrogate about which procedures are (1) essential to the success of the anesthesia and the proposed procedure, and (2) which procedures are not essential and may be refused.
- (c) Limited Attempt at Resuscitation Defined With Regard to the Patient's Goals and Values: The patient or designated surrogate may allow the anesthesiologist and surgical team to use clinical judgment in determining which resuscitation procedures are appropriate in the context of the situation and the patient's stated goals and values. For example, some patients may want full resuscitation procedures to be used to manage adverse clinical events that are believed to be quickly and easily reversible, but to refrain from treatment for conditions that are likely to result in permanent sequelae, such as neurologic impairment or unwanted dependence upon life-sustaining technology.

The discussion about the patient's wishes should be clearly documented in the medical record prior to the start of the procedure. Concurrence on these issues between the surgeon, anesthesiologist, patient, and if possible primary care physician and other members of the perioperative health care team is preferable.

Preoperative Physical Exam

The preoperative physical exam is a brief, focused exam with attention paid to organ systems relevant to the surgery and anesthetic plan. It includes an assessment of blood pressure, heart rate, respiratory rate, oxygen saturation, height, weight, and auscultation of the heart and lungs. A detailed description of airway evaluation can be found in Chap. 8.

All patients should have a thorough airway evaluation (Table 7.8). The Mallampati classification is performed by having patients maximally open the mouth and protrude the tongue with the neck in neutral position, and without phonation. In class 1, the soft palate, fauces, entire uvula, and pillars

Table 7.8 Components of the preoperative airway examination	Table 7.9 General re
Airway examination	Age <50, healthy
Condition of the teeth, including presence of loose teeth and length of upper incisors	No tests unless speci Electrocardiogram
Ability to protrude lower incisors in front of upper incisors	Age 50 or older
Interincisor distance (<4 cm predictive of difficult intubation)	Hypertension Current or past signif
 Tongue size Mallampati classification Class I: entire uvula visualized Class II: soft palate, fauces, portion of uvula visualized Class III: soft palate and base of uvula visualized 	Cardiothoracic proce Chest X-ray Major respiratory con episode within past 6 Cardiothoracic proce
Class IV: only hard palate visualized Presence of facial hair	Serum chemistry Renal disease
Thyromental distance Length and circumference of neck	Major metabolic disc Diuretic therapy Chemotherapy
Range of motion of neck	Urinalysis Genito-urologic proc
are visualized. In class II, the soft palate, fauces, and a portion of the uvula are seen. In Class III only the soft palate and base	<i>Complete blood cour</i> Hematologic disorde Vascular procedure

of the uvula are seen, and in class IV only the hard palate is visualized. The evaluation should also include the status of the teeth including preexisting abnormalities, neck range of motion, neck circumference, and thyromental distance. In otolaryngology patients particular attention is paid to any upper airway deformities that may make mask ventilation and/or intubation with direct laryngoscopy difficult. These include oropharyngeal masses, neck masses, thyromegaly, and post-radiation changes in soft tissue of the head and neck. Often the otolaryngologist has performed indirect laryngoscopy during the preoperative assessment and discussion of the findings can aid in planning for airway management. Concerns about airway management should be discussed with the patient at this time, including preparing the patient for awake fiberoptic intubation if indicated. Specific physical exam signs important to various subsets of otolaryngologic patients appear in the appropriate chapters throughout the text.

Preoperative Testing

Preoperative testing should be directed by the history and physical exam and the need for any tests should be considered in light of the planned surgery, including expected hemodynamic instability and blood loss. In general, tests should only be ordered if the results have the potential to change management. In the authors' institution only three tests are mandatory: (1) a pregnancy test for all women who have menstruated in the last year, (2) a preoperative blood glucose if the patient is diabetic, and (3) a serum potassium within 24 h of surgery if the patient is on dialysis.

Routine preoperative ECG testing is more controversial. The most recent ACC/AHA guidelines do not consider ECG to be indicated in asymptomatic patients undergoing low-risk

Table 7.9 General recommendations for preoperative testing
<i>Age <50, healthy</i> No tests unless specified by surgeon for specific surgical issues
<i>Electrocardiogram</i> Age 50 or older Hypertension Current or past significant cardiac/circulatory disease Cardiothoracic procedure
<i>Chest X-ray</i> Major respiratory condition with change of symptoms or an acute episode within past 6 months Cardiothoracic procedure
Serum chemistry Renal disease Major metabolic disorders (adrenal, thyroid, diobetics) Diuretic therapy Chemotherapy
Urinalysis Genito-urologic procedure
Complete blood count Hematologic disorder Vascular procedure Chemotherapy
Coagulation studies Anticoagulation therapy Vascular procedure
Pregnancy testing Patients who may be pregnant (i.e. menstruated within previous 12 months)

procedures regardless of patient age. These guidelines also state that evidence for routine ECG testing is not even well established for patients with one clinical risk factor undergoing intermediate risk procedures ^[1]. Many centers use age as a criterion for routine ECG testing—in our center an age greater than 50 combined with a non-minor procedure is an indication for a preoperative ECG. Arguments for this practice include the fact that at least 25% of myocardial infarctions in elderly patients are "silent," and that a preoperative ECG provides a baseline should the patient develop ECG changes, chest pain, or cardiac complications perioperatively. Given that a 12-lead ECG can be obtained without delaying surgery and poses no risk to the patient, and that otolaryngology surgery frequently results in hypertension, and bradycardia is often seen during neck surgery this seems reasonable.

There is no routine indication for preoperative chest radiograph, but one should be obtained to assess abnormalities that are found present by history or physical examination, including auscultation of rales or rhonchi, advanced COPD, suspected pulmonary edema, suspected pneumonia, pulmonary or mediastinal masses, and tracheal deviation. Patients with head and neck masses and large goiters generally have had CT scans performed and they should be available to assess the extent of airway compromise (e.g., collapse, deviation, presence or absence of tacheomalacia) and help plan for appropriate airway management. General recommendations for preoperative testing are listed in Table 7.9.

Conclusion

An evidence-based, goal directed, preoperative assessment with targeted optimization of the otolaryngologic patient is critical to improve patient safety and outcome. Anesthesiologists and otolaryngologists must work together to assure such care is provided to all of their patients.

References

- Fleisher LA, Beckman JA, Brown KA, et al. ACC/AHA 2007 Guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: executive summary: a report of the American college of cardiology/American heart association task force on practice guidelines (Writing Committee to Revise the 2002 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery) developed in collaboration with the American society of echocardiography, American society of nuclear cardiology, heart rhythm society, society of cardiovascular anesthesiologists, society for cardiovascular angiography and interventions, society for vascular medicine and biology, and society for vascular surgery. J Am Coll Cardiol. 2007;50:1707–32.
- Lee TH, Marcantonio ER, Mangione CM, et al. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. Circulation. 1999;100: 1043–9.
- Lindenauer PK, Pekow P, Wang K, Mamidi DK, Gutierrez B, Benjamin EM. Perioperative beta-blocker therapy and mortality after major noncardiac surgery. N Engl J Med. 2005;353:349–61.
- 4. Gibbons RJ, Abrams J, Chatterjee K, et al. ACC/AHA 2002 guideline update for the management of patients with chronic stable angina–summary article: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on the Management of Patients With Chronic Stable Angina). Circulation. 2003;107:149–58.
- 5. Hunt SA, Abraham WT, Chin MH, et al. ACC/AHA 2005 Guideline update for the diagnosis and management of chronic heart failure in the adult: a report of the American college of cardiology/american heart association task force on practice guidelines (Writing Committee to Update the 2001 Guidelines for the Evaluation and Management of Heart Failure): developed in collaboration with the American college of chest physicians and the international society for heart and lung transplantation: endorsed by the heart rhythm society. Circulation. 2005;112:e154–235.
- Devereaux PJ, Yang H, Yusuf S, et al. Effects of extended-release metoprolol succinate in patients undergoing non-cardiac surgery (POISE trial): a randomised controlled trial. Lancet. 2008;371: 1839–47.
- Dunkelgrun M, Boersma E, Schouten O, et al. Bisoprolol and fluvastatin for the reduction of perioperative cardiac mortality and myocardial infarction in intermediate-risk patients undergoing noncardiovascular surgery: a randomized controlled trial (DECREASE-IV). Ann Surg. 2009;249:921–6.
- Howell SJ, Sear JW, Foex P. Hypertension, hypertensive heart disease and perioperative cardiac risk. Br J Anaesth. 2004;92: 570–83.
- Whinney C. Perioperative medication management: general principles and practical applications. Cleve Clin J Med. 2009;76 Suppl 4:S126–32.
- Rosenman DJ, McDonald FS, Ebbert JO, Erwin PJ, LaBella M, Montori VM. Clinical consequences of withholding versus admin-

istering renin-angiotensin-aldosterone system antagonists in the preoperative period. J Hosp Med. 2008;3:319–25.

- Tirumalasetty J, Grammer LC. Asthma, surgery, and general anesthesia: a review. J Asthma. 2006;43:251–4.
- Rock P, Passannante A. Preoperative assessment: pulmonary. Anesthesiol Clin North Am. 2004;22:77–91.
- Silvanus MT, Groeben H, Peters J. Corticosteroids and inhaled salbutamol in patients with reversible airway obstruction markedly decrease the incidence of bronchospasm after tracheal intubation. Anesthesiology. 2004;100:1052–7.
- Rabe KF, Hurd S, Anzueto A, et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. Am J Respir Crit Care Med. 2007;176:532–55.
- Shafazand S. Perioperative management of obstructive sleep apnea: ready for prime time? Cleve Clin J Med. 2009;76 Suppl 4:S98–103.
- Chung F, Yegneswaran B, Liao P, et al. STOP questionnaire: a tool to screen patients for obstructive sleep apnea. Anesthesiology. 2008;108:812–21.
- 17. Gross JB, Bachenberg KL, Benumof JL, et al. Practice guidelines for the perioperative management of patients with obstructive sleep apnea: a report by the American society of anesthesiologists task force on perioperative management of patients with obstructive sleep apnea. Anesthesiology 2006;104:1081–93; quiz 117–8.
- van den Berghe G, Wouters P, Weekers F, et al. Intensive insulin therapy in the critically ill patients. N Engl J Med. 2001;345: 1359–67.
- Finfer S, Chittock DR, Su SY, et al. Intensive versus conventional glucose control in critically ill patients. N Engl J Med. 2009;360: 1283–97.
- Moghissi ES, Korytkowski MT, DiNardo M, et al. American association of clinical endocrinologists and American diabetes association consensus statement on inpatient glycemic control. Diabetes Care. 2009;32:1119–31.
- Kavanagh BP, McCowen KC. Clinical practice. Glycemic control in the ICU. N Engl J Med. 2010;363:2540–6.
- 22. Udelsman R, Norton JA, Jelenich SE, et al. Responses of the hypothalamic-pituitary-adrenal and renin-angiotensin axes and the sympathetic system during controlled surgical and anesthetic stress. J Clin Endocrinol Metab. 1987;64:986–94.
- 23. Salem M, Tainsh Jr RE, Bromberg J, Loriaux DL, Chernow B. Perioperative glucocorticoid coverage. A reassessment 42 years after emergence of a problem. Ann Surg. 1994;219:416–25.
- Goodman LS, Gilman A, Brunton LL, Lazo JS, Parker KL. Goodman & Gilman's the pharmacological basis of therapeutics. 11th ed. New York: McGraw-Hill; 2006.
- Rizvon MK, Chou CL. Surgery in the patient with liver disease. Med Clin North Am. 2003;87:211–27.
- Douketis JD, Berger PB, Dunn AS, et al. The perioperative management of antithrombotic therapy: American college of chest physicians evidence-based clinical practice guidelines (8th edition). Chest. 2008;133:299S–339.
- Jaffer AK. Perioperative management of warfarin and antiplatelet therapy. Cleve Clin J Med. 2009;76 Suppl 4:S37–44.
- Newsome LT, Weller RS, Gerancher JC, Kutcher MA, Royster RL. Coronary artery stents: II. Perioperative considerations and management. Anesth Analg. 2008;107:570–90.
- Tomar SL. Trends and patterns of tobacco use in the United States. Am J Med Sci. 2003;326:248–54.
- Warner DO. Preoperative smoking cessation: how long is long enough? Anesthesiology. 2005;102:883–4.
- Kaplan ED, Rozen WM, Shayan R, et al. Preventing postoperative haematomas in microvascular reconstruction of the head and neck: lessons learnt from 126 consecutive cases. ANZ J Surg. 2008;78: 383–8.

- Clark JR, McCluskey SA, Hall F, et al. Predictors of morbidity following free flap reconstruction for cancer of the head and neck. Head Neck. 2007;29:1090–101.
- Marin VP, Pytynia KB, Langstein HN, Dahlstrom KR, Wei Q, Sturgis EM. Serum cotinine concentration and wound complications in head and neck reconstruction. Plast Reconstr Surg. 2008; 121:451–7.
- Garces YI, Schroeder DR, Nirelli LM, et al. Tobacco use outcomes among patients with head and neck carcinoma treated for nicotine dependence: a matched-pair analysis. Cancer. 2004;101:116–24.
- Moore RD, Bone LR, Geller G, Mamon JA, Stokes EJ, Levine DM. Prevalence, detection, and treatment of alcoholism in hospitalized patients. JAMA. 1989;261:403–7.
- Moller AM, Villebro N, Pedersen T, Tonnesen H. Effect of preoperative smoking intervention on postoperative complications: a randomised clinical trial. Lancet. 2002;359:114–7.
- Yang GP, Longaker MT. Abstinence from smoking reduces incisional wound infection: a randomized, controlled trial. Ann Surg. 2003;238:6–8.
- 38. Nakagawa M, Tanaka H, Tsukuma H, Kishi Y. Relationship between the duration of the preoperative smoke-free period and

the incidence of postoperative pulmonary complications after pulmonary surgery. Chest. 2001;120:705–10.

- Warner MA, Divertie MB, Tinker JH. Preoperative cessation of smoking and pulmonary complications in coronary artery bypass patients. Anesthesiology. 1984;60:380–3.
- Hald J, Overgaard J, Grau C. Evaluation of objective measures of smoking status–a prospective clinical study in a group of head and neck cancer patients treated with radiotherapy. Acta Oncol. 2003;42:154–9.
- Tonnesen H, Kehlet H. Preoperative alcoholism and postoperative morbidity. Br J Surg. 1999;86:869–74.
- 42. Tonnesen H, Rosenberg J, Nielsen HJ, et al. Effect of preoperative abstinence on poor postoperative outcome in alcohol misusers: randomised controlled trial. BMJ. 1999;318:1311–6.
- 43. Spies CD, Nordmann A, Brummer G, et al. Intensive care unit stay is prolonged in chronic alcoholic men following tumor resection of the upper digestive tract. Acta Anaesthesiol Scand. 1996;40:649–56.
- 44. Ethical Guidelines for the Anesthesia Care of Patients with Do-Not-Resuscitate Orders or Other Directives that Limit Treatment. 2008. Accessed at www.asahq.org/publicationsAndServices/ standards/09.pdf. Accessed 1 May 2011.

Airway Management, Emergencies and the Difficult Airway

Irene P. Osborn, Andrew J. Kleinberger, and Vivek V. Gurudutt

Introduction

Modern surgical practice necessitates safe and efficient management of the airway in order to administer anesthesia in a controlled manner. It is the primary role and responsibility of the anesthesiologist to assess, secure, and monitor a patient's airway in the perioperative setting. Although performed on a daily basis in seemingly routine fashion, unanticipated problems in airway management will be encountered by even the most skilled anesthesiologist at a rate from 1% to 3% of patients undergoing general endotracheal anesthesia ^[1]. The effective identification of such potentially "difficult" airways should be the focus of the preoperative assessment by both surgeon and anesthesiologist in order to develop a coordinated plan of care and minimize the risk of precipitating a true airway emergency.

The practicing otolaryngologist by nature has unique insight into the anatomy and pathophysiology of the upper aerodigestive tract as well as endoscopic equipment and techniques for airway access. As a result, situations involving acute airway management will often require involvement by the otolaryngologist. Particularly in the perioperative setting, otolaryngologists will routinely care for patients with complex upper airway anatomy and pathology including acute infection, angioedema, trauma, malignancy and prior surgery and radiation therapy of the head and neck. These cases demand meticulous preoperative evaluation and communication between the otolaryngologist and anesthesiologist to devise and safely employ effective airway management strategies.

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Preoperative Assessment of the Airway

The preoperative evaluation of the surgical patient is the cornerstone in prevention of potential unanticipated airway emergencies. It is important to note that this is not only the responsibility of the anesthesiologist, but also the surgeon to engage in this process by actively participating in the patient's perioperative care. Such an assessment should begin with a thorough history and physical exam targeted to determine those patients at risk for difficult intubation (Table 8.1) and difficult ventilation (Table 8.2). Patients at risk for perioperative aspiration (Table 8.3) as well as those who may present problems in general with cooperation should also be identified early. Determining these parameters will have significant impact and will help in guiding coordinated patient management plans.

Particular factors pertinent to airway management include a history of head and neck malignancy, surgery, or trauma; requiring prolonged time on a ventilator or history of tracheostomy; reported "difficult" intubations during previous surgeries; problems with cervical spine mobility or mouth opening; the presence of loose or missing teeth; and a history of personal or familial congenital or genetic syndromes. Additionally, a thorough history to determine aspiration risk must also be ascertained. These factors include a diagnosis of gastroesophageal reflux disease and/or history of taking anti-reflux medications (e.g., proton-pump inhibitors, H2-receptor antagonists), the presence of hiatal hernia, diabetes mellitus, previous gastric bypass surgery, and Zenkers diverticular disease. In many instances this information will not be freely volunteered by the patient, and thus must be actively sought during preoperative consultation in order to appropriately triage the patient. Although the history of a previously difficult intubation is perhaps the most important factor to consider, any of these predictors may signal a possibly "difficult" scenario for the anesthesiologist and necessitate alternate or advanced strategies in order to safely secure the patient's airway for surgery.

After collecting the patient's history, a targeted physical exam should then be performed with a focus on physical attributes associated with difficult intubation and mask

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Table 8.1 Predictors of difficult intubation		
Small mouth opening (<3 cm)		
Short thyromental distance (<6 cm)		
Inability to protrude the mandible		
Mallampati score greater than 3–4		
Limited neck movement		
History of difficult intubation		
History of radiation therapy to the head and neck		
Large neck circumference (>40 cm)		

Table 8.2	Predictors of difficult mask ventilation
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Age greater than 57 years
High body mass index (>30 kg/m ²)
Mallampati class 3–4
History of snoring
Lack of teeth
Limited jaw protrusion
History of radiation therapy to the head and neck
History of obstructive sleep apnea

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Table 8.3	Risk factors	tor	gastric a	aspiration	during	anesthesia

Obesity/pregnancy
Emergency surgery
Trauma
Airway difficulties
Inadequate anesthetic depth
Gastroesophageal reflux
Gastrointestinal disorders
Intestinal obstruction, hiatal hernia
Opioid use

ventilation including overall body habitus, neck proportion and extensibility, oral excursion and the presence of potential oral cavity and/or oropharyngeal obstruction, any prominent, loose, or missing dentition, thyromental distance and jaw projection.

Several classification schemes have been devised to assist in grading the degree of airway "difficulty" including the widely used Mallampati score (Fig. 8.1)^[2]. This simple grading system functions to correlate the amount of visualization of the posterior oropharynx with a potential site of obstruction and problems with conventional transoral intubation. Many groups have since examined and proposed modified grading scales to improve predictive reliability most notably by Samsoon and Young who expanded the initial Mallampati score in 1987 from three to four grades to account for a complete lack of soft palatal exposure with mouth opening ^[3]. Even more recently, the upper lip bite test (ULBT) has been added to the list of possible predictors of airway difficulty in which the inability to bite the upper lip with lower incisors demonstrates lack of translation of the lower jaw and thus potential problems with mask ventilation. Although the ULBT has been demonstrated to have a higher specificity than others including the modified Mallampati test ^[4], more recent and large-scale prospective studies challenge its use as a single screening tool to predict difficult laryngoscopy ^[5]. It should be noted that no grading system or physical attribute is 100% predictive of a difficult intubation, and they are best applied together and in proper clinical context.

If there is a concerning finding on either history or physical exam, flexible fiberoptic laryngoscopy may be performed to directly evaluate the adequacy of laryngeal access. Often in otolaryngology cases this has already been undertaken during a previous encounter and findings can be relayed to the anesthesiologist preoperatively so that an airway management strategy can be prepared. While this is a useful test for determining the presence of airway abnormalities such as lingual tonsil hypertrophy or other lesions, it may not always predict difficult direct laryngoscopy. A recent paper by Rosenblatt described the technique of preoperative endoscopic airway examination (PEAE) used by anesthesiologists as a component of the routine preoperative assessment ^[6]. The PEAEs were carried out by an anesthesiologist in the preoperative holding area using a flexible fiberoptic bronchoscope after topical vasoconstrictors and anesthetic agents were administered intranasally. With the patient in a semirecumbent position, the upper aerodigestive tract is then carefully inspected for pathology or anatomic variations that may predispose to difficulty with intubation and/or ventilation; a specific airway plan was then devised and implemented based on these findings. When performed immediately prior to airway management, this procedure allows for assessment and confirmation of airway architecture and abnormalities and has the potential to avoid unnecessary awake intubation in some cases. Additional randomized trials are needed to validate this technique that may be utilized by both otolaryngologists and anesthesiologists in the preand perioperative setting.

Perioperative Airway Management

In 2003, a task force for the American Society of Anesthesiologists published revised "Practice Guidelines for Management of the Difficult Airway" ^[7] (first published in 1993). These recommendations are based on the preoperative assessment of difficulty with either ventilation or intubation. A proposed algorithm emphasizes the importance of preserving spontaneous ventilation if problems with ventilation are anticipated as well as the need for alternative strategies in addition to the primary method of airway management. After the induction of general anesthesia and

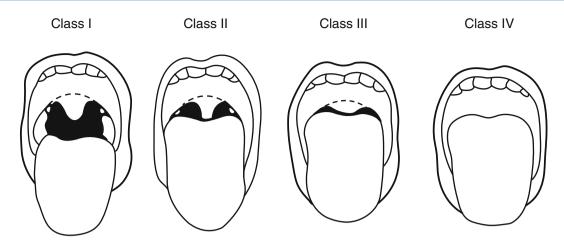


Fig. 8.1 Mallampati classification score

failed intubation, the clinical utility of the laryngeal mask airway (LMA), in cases when mask ventilation is not possible is evident ^[8]. When both strategies fail, a variety of noninvasive and invasive techniques for airway ventilation and access are available. The ASA guidelines represent a systematic schema for the perioperative management of the difficult airway that may assist in preventing unanticipated airway emergencies (Fig. 8.2).

Routine Airway Management

Routine general anesthesia begins with induction and endotracheal intubation or placement of a supraglottic airway. Once the airway is secured with confirmation of device placement (presence of breath sounds bilaterally, absence of gastric sounds, the presence and persistence of expiratory carbon dioxide) and initiation of mechanical or spontaneous ventilation, the surgery may proceed. There are many endotracheal tube (ETT) variations on the market with preconfigured bends and wire reinforcement [Ring-Adair-Elwyn (RAE tube), Anode tube] that decrease kinking and promote airway patency and avoidance of field. These ETT options will be discussed in other procedure specific chapters in the text. Throughout the surgery, both anesthesiologist and otolaryngologist must be careful that the airway device is not inadvertently disturbed, especially in cases where excessive patient repositioning is employed or areas of the head and neck are directly manipulated. Many otolaryngologic procedures require extreme neck extension for surgical exposure including laryngoscopy, tonsillectomy, thyroidectomy, and parathyroidectomy. Neck extension causes the ETT to rise in the airway and may promote premature and inadvertent extubation, while conversely, neck flexion advances the tip of the ETT in the airway potentially causing a mainstem intubation (as a general rule the endotracheal tube moves in the direction of the patient's nose). At the conclusion of the surgery the patient is typically extubated by the anesthesiologist and when clinically stable, transported to the recovery room for postoperative monitoring ^[9].

Endotracheal intubation is most commonly performed by conventional laryngoscopy with either the Macintosh or Miller style blades (Fig. 8.3). The Macintosh blade is curved and designed for insertion into the vallecula anterior to the epiglottis in order to retract upwards thereby revealing the larynx. In contrast, the straight Miller blade is placed posterior to the epiglottis to compress the tongue base and supraglottis anteriorly. This is particularly useful in patients with excessive soft tissue in this region or a floppy, elongated epiglottis as commonly found in pediatric patients.

Adjunctive devices commonly used in airway management are the oral and nasal airways. Inserted blindly following the induction of general anesthesia, these devices can mechanically augment airflow and thereby facilitate mask ventilation or spontaneous breathing. A major advent in modern anesthesiology has been the incorporation of the supraglottic airway (SGA), most commonly the laryngeal mask airway (LMA) for routine care [10] (Fig. 8.4). This device is generally inserted blindly in the oral cavity and can serve as a primary airway during general anesthesia, a conduit for endotracheal intubation, as well as a rescue tool when intubation fails and mask ventilation is difficult or impossible. Although the SGA creates a reliable and effective airway, they do not protect against aspiration and may prove unreliable in patients with decreased lung or chest wall compliance (e.g., asthma, COPD, obesity). A variety of LMAs have also been designed to provide a means for endotracheal intubation (ILMA FastrachsTM) and suction gastric contents (LMA SupremeTM). Similarly, but perhaps less often used is the CombitubeTM that has dual lumens with a proximal and distal cuff to ensure adequate ventilation with transesophageal or tracheal placement ^[11] (Fig. 8.5).



- 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 Tracheostomy
- 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:

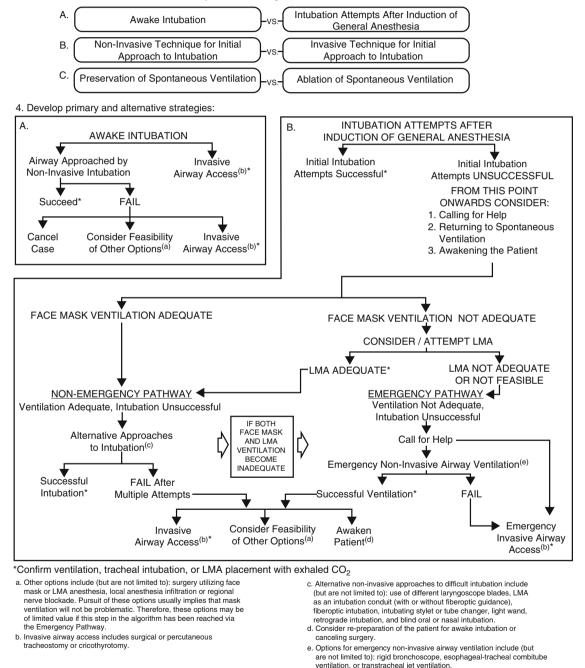


Fig. 8.2 ASA difficult airway algorithm. From practice guidelines for management of the difficult airway: an updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway, Anesthesiology, 2003 May;98(5):1269–77



Fig. 8.3 Macintosh (upper) and Miller (lower) laryngoscopy blades



Fig. 8.4 Supraglottic airways of various types (LMA classic, ILMA Fastrach, LMA Supreme)



Fig. 8.5 Combitube and laryngeal tube

The Anticipated "Difficult Airway"

In cases where a potentially "difficult" airway is identified during the preoperative assessment, the anesthesiologist and otolaryngologist must work as an effective team, and each must be prepared with alternative anesthetic options and intubation strategies.

The Otolaryngologist's Armamentarium

In addition to conventional laryngoscopy, a variety of rigid laryngoscopes and bronchoscopes are often employed by the otolaryngologist and may be incorporated in either planned or emergent airway strategies. These include the sliding Jackson that is equipped with a removable posterior segment that can be detached after intubating successfully through the instrument or the the anterior commissure laryngoscope such as the Benjamin Slimline and Hollinger laryngoscopes. These laryngoscopes have a narrow distal end to better facilitate laryngeal exposure in cases where there is significant obstruction or difficulty with anterior access. The Lindholm and Dedo laryngoscopes are also available providing a wider aperture for laryngeal viewing, instrumentation, or ETT insertion; however, these are often inadequate in cases where exposure is particularly difficult and an anterior commissure scope may be preferred (Fig. 8.6).

In addition to this array of various laryngoscopes, the rigid bronchoscope is another tool commonly used by otolaryngologists in emergent airway situations. In cases where patients can neither be adequately intubated or ventilated, a rigid bronchoscope can be directly inserted through the vocal cords into the trachea to establish a means for ventilation, intubation, airway instrumentation, or as a temporizing measure during placement of a surgical airway.

The Anesthesiologist's Armamentarium

Flexible Bronchoscopy

The most common device used by anesthesiologists to intubate the patient with a difficult airway is a flexible bronchoscope. This instrument has the advantage of being able to be advanced and manipulated in a 360° manner, thus facilitating use in both transnasal and transoral routes depending on clinical and anatomical characteristics ^[12]. Specific indications include patients with limited mouth opening, neck mobility, or airway tissue pliability from previous surgery, radiation therapy, obstructing or friable airway lesions, and anterior or **Fig. 8.6** Array of commonly used rigid laryngoscopes (A. Hollinger, B. Lindholm, C. Sliding Jackson, D. Dedo)



restricted laryngeal and airway anatomy. Because of the ability to use an attached camera and external monitor, intubations with a flexible bronchoscope also allows for team observation and clinical instruction (Fig. 8.7).

Newer technology uses high definition digital optics with a charge-coupled device (CCD) chip embedded in the distal end of the device that eliminates the need for a fiberoptic bundle. Reportedly this technology provides brighter and sharper images than fiberoptic bronchoscopes and is considered more durable since there is no fiberoptic bundle susceptible to damage. Distal chip digital technology also eliminates the need for a separate camera assembly and provides digital photo and video recording capabilities. While extremely useful for many "difficult" airway cases, significant bleeding or secretions greatly diminishes optical visualization despite these technological advances and can make the use of this device less then optimal.

Fixed Video Laryngoscopes

Video-laryngoscopy is the latest technology used for airway management, and a variety of devices offer improved success in difficult and routine intubations. This technique provides airway visualization by eliminating the need to align airway structures and direct line of site for endotracheal intubation. Perhaps the most common videolaryngoscope currently in use by both anesthesiologists and emergency physicians is the GlideScope[®], which incorporates a small camera on the tip of the laryngoscope to a portable bedside monitor ^[13].



Fig. 8.7 Fiberoptic intubation view of larynx

The blade forms a 60° angle to provide a greater and more anterior laryngeal view and facilitate intubation. This is also especially useful for team airway management by having the ability to show other participating practitioners the field of view. There are two main classes of videolaryngoscopes: "blade-type" such as the GlideScope[®], StorzVideoMac[®] and the McGrath[®] series 5 and "channeled" scopes such as the Airtraq[®] and Pentax[®] AWS (Fig. 8.8). In order to achieve success with this new technology, it is important to use proper head positioning techniques and to obtain the "best view" possible before advancing the ETT. This may require that one slightly withdraw the device to reveal more anatomy (e.g., epiglottis, vocal cords, arytenoids) and thus provide more space for safely introducing the ETT (Fig. 8.9). It is also often necessary to utilize a stylette to maintain ETT shape with the "blade-type" videolaryngoscopes; the "channeled" scopes do not require a stylette for ETT placement.



Fig. 8.8 Videolaryngoscopes: channeled and blade type (Pentax AWS, Airtraq, McGrath, and GlideScope)

In a recent retrospective study of 2004 patients the Glidescope had a 98% success rate when used routinely and in patients with at least one known risk factor for a difficult intubation. Impressively, the Glidescope had a 94% success rate when used as a secondary device after traditional laryngoscopy had failed. Risk factors associated with GlideScope[®] failure that are particularly important for anesthesiologists and otolaryngologists include evidence of airway pathology, a local mass, previous surgery, and radiation therapy of the head and neck. Interestingly, limited range of the cervical spine was also associated with failed intubation using the GlideScope[®] ^[14]. For a list of advantages and disadvantages of the videolaryngoscope, see Table 8.4.

Intubating Introducers and Stylettes

There are an array of bougies, stylettes, and introducers that can be used with traditional or video-laryngoscopy for difficult or emergent intubations. They may be placed when there is limited laryngeal visualization and depend on tactile feedback to confirm proper placement within the trachea ^[15]. Once in the airway, they are used as a guide or conduit for the insertion of the ETT by Seldinger technique. These instruments may also serve as a conduit for retrograde intubations when patients with tracheotomies present with difficult airways but require oral airway management for surgical exposure.



Table 8.4	Advantages and	disadvantages of	video-laryngoscopy
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Advantages of video-laryngoscopy
Improved laryngeal view
Alignment of oral, pharyngeal, and laryngeal axes not required
Potentially less movement of the cervical spine
Teaching and demonstration ability
Ability to perform rapid sequence induction and confirmation of ETT placement
Video-imaging of ETT exchange
Disadvantages of video-laryngoscopy
Adequate mouth opening required
Inability to place ETT despite laryngeal view
Hand-eye coordination required
Potential injuries during tube placement

The Difficult Ventilation Patient

The Awake Intubation

In cooperative patients who are likely to be difficult to mask ventilate after the induction of general anesthesia, it is advisable to proceed with an "awake" method of securing the airway^[16]. There are many methods to prepare the patient for an awake intubation, but in general, they include a combination of airway local anesthesia topicalization, local anesthetic nerve blocks, and judicial doses of intravenous sedatives and analgesics, while assuring the patient remains awake, responsive, and spontaneously breathing. Common local blocks will be discussed in detail below, but they include bilateral superior laryngeal nerve blocks along with the transtracheal administration of local anesthesia that targets the recurrent laryngeal nerve and facilitates a safe and relatively well-tolerated manipulation of the airway in the awake patient. This is often combined with a flexible fiberoptic technique especially for cases involving known and significant upper airway obstruction [17].

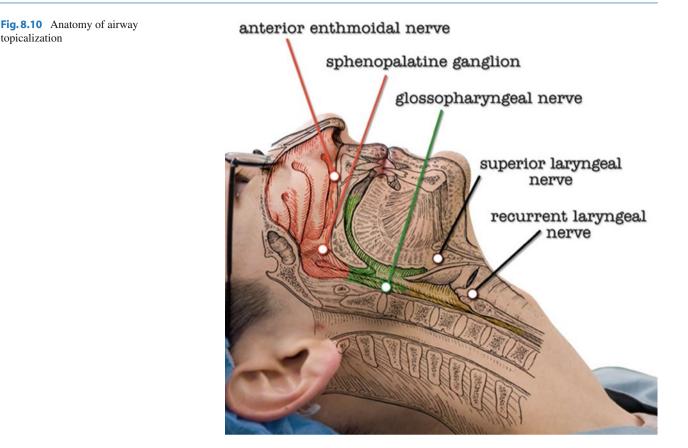
Techniques for Airway Topicalization

There are many published and described techniques that will allow patients to comfortably tolerate awake intubation via the oral or nasal route. This is frequently a combination of topical anesthesia and/or regional blocks in conjunction with sedation as needed. It is important to begin by communicating effectively with the patient and explaining what to expect. Contraindications to awake intubation include patient refusal, the intoxicated/combative patient, or a documented history of allergy to all local anesthetics.

The first step for effective airway topicalization is the administration of an anti-sialagogue to dry secretions and to allow for better mucosal absorption of the local anesthetic. Glycopyrrolate (0.2–0.4 mg) maybe administered intravenously as soon as access is obtained or intramuscularly. With either route the drying effect can be expected to occur within 20 min. Whether the oral or nasal route is chosen, the goal is to apply local anesthetic along the airway passage with the understanding of the relevant anatomy (Fig. 8.10).

Minimal sedation is recommended for most patients to decrease anxiety, provide analgesia, and allow for enhanced cooperation. A detailed description of these agents can be found in Chap. 3. When properly administered, many patients do not recall the experience and if so, do not remember it as unpleasant. There is no sense proceeding with an "awake intubation" only to overly sedate the patient and risk, respiratory depression, airway loss, and cooperation. It is important to remember that the awake, cooperative patient will facilitate the intubation not hinder it and utilizing judicial doses of sedation is advisable. The awake patients can be instructed to stick their own tongue out or to subluxate their own TMJ joint thus opening their own airway. They can also facilitate the placement of the endotracheal tube by actively deep breathing.

A number of agents are acceptable and the choice(s) will depend on the patient's medical condition, degree of anxiety, and airway pathology. Midazolam is most often utilized for its anxiolysis, amnesia, and reversibility but must be used cautiously in patients with obstructive sleep apnea (OSA) (Chap. 9) as well as in elderly and debilitated patients. Midazolam also raises the seizure threshold which may prove useful during the administration of airway topicalization. Fentanyl may also be used for supplemental sedation and provides analgesia, decreases cough response, enhances cooperation, and is readily reversible. Newer agents include the opioid remifentanil, which produces profound analgesia and cooperation but must be administered carefully to avoid bradycardia and respiratory depression. Dexmedetomidine, an alpha-2 agonist, is also useful for awake intubation but requires 10-15 min for onset of action. Its primary advantage is decreased respiratory depression compared to the aforementioned agents and is thus recommended for debilitated patients. Ketamine is a potent hypnotic and analgesic which could prove a useful supplement for emergency patients or those who are unable to cooperate, but increased secretions and potential agitation minimizes its usefulness. All agents should be administered in divided doses with careful monitoring of the patient and the use of supplemental oxygen.



Nasal Approach

Nasal intubation requires blockade of the nasal passages as well as vasoconstriction to prevent bleeding. Vasoconstriction of the nasal mucosa is generally achieved with either cocaine or phenylephrine and generally decongests the nasal mucosa, which widens the space and reduces the risk of bleeding during manipulation; oxymetazoline (Afrin) is also an effective and long-acting agent available as an intranasal spray. Cocaine has been used in concentrations as high as 10% but commonly the 4% solution is recommended. It has the advantage of providing excellent topical anesthesia as well as local vasoconstriction of the nasal mucosa and is typically applied via soaked pledgettes or cotton-tipped applicators along the nasal passages to target the anterior ethmoid and nasopalatine nerves. A nasal trumpet lubricated with viscous lidocaine can then be gently inserted into the wider of the two nares to further dilate the nasal passage and prepare the patient for introduction of the ETT and fiberoptic bronchoscope. In our practice we recommend using a handheld atomizer attached to a supplemental oxygen source. Here the atomizer bulb is removed and replaced with oxygen tubing. Using 6-10 L flow the local anesthesia is finely atomized and the patient is encouraged to breathe deeply while this is administered nasally. Figure 8.11 shows an atomizer used to provide a continuous spray of local anesthetic.

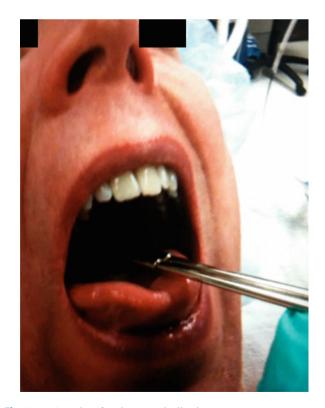


Fig. 8.11 Atomizer for airway topicalization

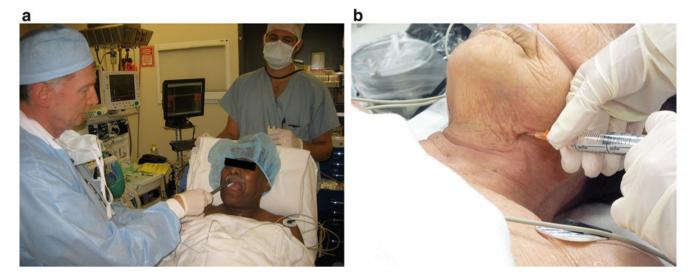


Fig. 8.12 (a) Kraus forceps for pledgette application. (b) Superior laryngeal nerve block by external approach

Oral Approach

A combination of techniques is required to adequately anesthetize the oral cavity, oropharynx, larynx, and trachea for awake intubation. The broadest coverage is provided by the inhalational technique, which involves nebulized 4% lidocaine via a facemask or oral nebulizer. This technique requires time (15-30 min) to achieve an adequate analgesic effect and requires active and deep respirations by the patient. Local anesthetic can also be sprayed directly onto the desired mucosa; a pressurized solution of lidocaine pointed into the oropharynx and sprayed generously will also anesthetize the tongue base and supraglottis innervated by the glossopharyngeal and vagus nerves. This technique is generally faster and can also be performed with either a handheld atomizer and a supplemental oxygen source as described above (our preferred method) or the newer mucosal atomization devices. Alternatively, a 10-ml syringe can be filled with lidocaine 2–4% and sprayed attached directly to the the scope or using a small-bore catheter (our preferred method and described below) via the working channel of the fiberoptic bronchoscope; this is known as the "spray as you go" technique and requires patience and timing.

Regional Nerve Blocks

Regional nerve blocks are useful to provide reliable analgesia and supplement topical anesthetic especially in patients with eroding necrotic oral lesions where local topicalization will be inadequate due to acidotic tissue conditions. The superior laryngeal nerve innervates the base of the tongue, posterior surface of the epiglottis and the arytenoids. It can be blocked internally in the piriform fossa by using 90° Kraus or Jackson forceps to hold a lidocaine-soaked pledgette against the mucosa for approximately 60 s (Fig. 8.12a). Alternatively, this block can be performed using an external approach by direct infiltration with a 25-gauge needle at the level of the thyrohyoid membrane inferior to the cornu of the hyoid bone. After negative aspiration, 2 ml of lidocaine 2% is injected bilaterally (Fig. 8.12b).

The recurrent laryngeal nerve provides primary sensory innervation to the glottis and trachea, and successful blockade facilitates comfortable passage of the ETT into the airway. It can be targeted via topicalization techniques mentioned previously; however, the translaryngeal block provides reliable conditions for airway inspection and patient comfort. In this technique, a 5-ml syringe with a 20- or 22-gauge needle (or catheter) is advanced through the cricothyroid membrane until air is aspirated into the syringe, the patient is instructed to take a deep breath and then 4 ml of lidocaine 4% is then rapidly injected, inducing coughing that further disperses the local anesthetic (Fig. 8.13). The recurrent laryngeal nerve can also be blocked by the "spray as you go" method using the fiberoptic bronchoscope as described in the previous section. For improved control of local anesthesia administration using the "spray as you go" method we prefer using a pre-threaded epidural catheter through the working channel of the bronchoscope and advancing the tip of the catheter during local anesthesia administration. It is important to proceed with intubation shortly thereafter and to observe closely for any signs of local anesthetic toxicity, sedation, nystagmus, change in mental status, and agitation. If impending local toxicity is suspected, judicial administration of midazolam is recommended and securing the airway in a timely fashion must be accomplished before frank seizures and or cardiovascular effects occur.



Fig. 8.13 Transtracheal block

Emergent Airway Strategies

Nonsurgical Airway Management

Otolaryngologist's Perspective

The otolaryngologist can consider placing a rigid bronchoscope to provide ventilation in emergent situations. If successful there will be more time to acquire additional equipment and/or personnel necessary for subsequent attempts to secure the airway or to prepare for placement of a surgical airway. If unsuccessful, endotracheal intubation via rigid laryngoscopes, bougies, and introducers may be employed as long as adequate ventilation is delivered between laryngoscopy attempts. When available, flexible fiberoptic equipment may also be employed in the emergent setting.

Anesthesiologist's Perspective

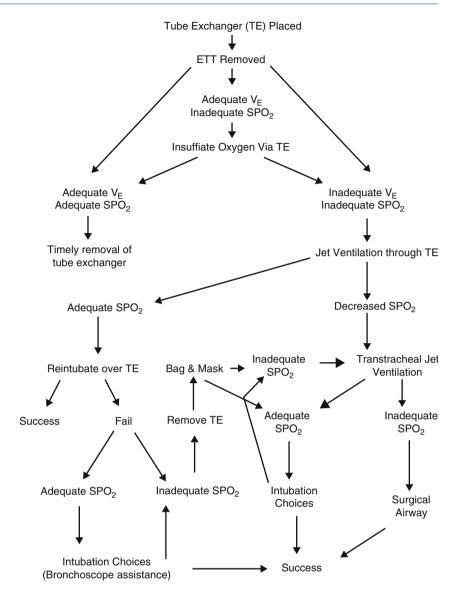
Despite a thorough preoperative assessment with a wellplanned and executed perioperative airway management strategy, unanticipated events may still transpire. Acute airway compromise may also be encountered in a variety of nonoperative settings including the emergency department and inpatient wards. Principles for management of airway emergencies are similar to that of the difficult airway with an emphasis on the need for invasive techniques. Airway management strategies should follow a systematic protocol depending on the clinical scenario and acuity of the patient ^[18]. For the purposes of discussion, an airway emergency will be defined as a situation in which a patient cannot be adequately ventilated or intubated by conventional means. In these instances, the clinical stability of the patient and availability of resources will dictate necessary management. As described previously, adjuncts to mask ventilation including oral and nasopharyngeal airways as well as the insertion of a supraglottic airway (LMA or combitube) should be attempted ^[19]. If successfully placed, the LMA can provide rescue ventilation as well as a conduit for intubation as previously described ^[20]. It can also be used to provide temporary ventilation during placement of a surgical airway.

Extubation of the Difficult Airway

Management of the difficult airway does not end with placement of an ETT; the anesthesiologist and otolaryngologist must share in the decision-making process and strategy for the management of tracheal extubation. The ASA Task Force has recommended a preformulated strategy for extubation of patients with difficult airways (Fig. 8.14). Risk factors for difficult tracheal reintubation include a history of previous difficult intubation, airway edema secondary to surgical manipulation or volume resuscitation, morbid obesity, inexperienced personnel, airway injury, burns or smoke inhalation, limited access or anatomical derangement, and an immobilized or unstable cervical spine.

A team approach is essential as the otolaryngologist has knowledge of the anatomy and structural integrity of the surgical site, and the anesthesiologist has experience and techniques for reintubation if necessary. This team effort may be required at the end of surgery or hours to days later in the intensive care unit. It is important that a plan be constructed in the event of airway failure and need for rapid securing of the airway. If extubation complications are a significant concern, continued endotracheal intubation during the immediate postoperative period should be considered. Following the termination of anesthetic agents, the patient is expected to emerge and begin spontaneous ventilation. Once a regular pattern is established and the patient is responding appropriately to commands, it is possible to determine if airway reflexes are intact. The extubation sequence requires careful observation and is best accomplished with the patient's head elevated to facilitate spontaneous ventilation.

The cuff leak test is sometimes performed in which the ETT cuff is deflated to determine whether the patient can breathe around the outer diameter of the tube. Theoretically, if the patient can breathe with the ETT cuff deflated, then there likely is no clinically significant edema or other airway obstruction to preclude a safe and successful extubation. The qualitative test involves observation of the presence of leak around the ETT after cuff deflation and manually occluding the tube lumen. The quantitative test is performed by measuring the actual delivered tidal volume; a cuff leak of **Fig. 8.14** Guidelines for extubation of the anticipated difficult airway



more than 110 ml has been shown to predict the absence of post-extubation stridor (Table 8.5).

Unfortunately, there are many confounding variables to consider and the test is not consistently reliable. Several factors may account for the lack of a cuff leak: excessive hydration, airway trauma (which may be intubation-related), poor patient or ETT positioning, generalized edema, systemic reactions, sepsis, angioedema, infections, impingement of head and neck venous drainage, and an oversized ETT. In general, the lack of a cuff leak may suggest a higher likelihood of post-extubation stridor or the need for reintubation or tracheotomy, although this remains controversial. When faced with this scenario, many choose to give steroids for 24 h after a failed cuff leak test if there is a known or suspected reason for upper airway edema. Conversely, if there is no clinical suspicion and intubation was not initially difficult,

Table 8.5 Cuff leak test

- 1. Qualitative test: observation of presence of leak around the tube after cuff deflation and occluding the lumen of endotracheal tube
- 2. Quantitative test: record the exhaled tidal volume of 6 consecutive breaths with cuff deflated. Choose the lowest values and take the average. Subtract the average from the delivered tidal volume. Cuff leak of more than 110 ml predicts the absence of post-extubation stridor. Another study showed that the cuff leak of less than 10% of pre-deflation tidal volume predicts the occurrence of post-extubation stridor

one may choose to extubate and observe the patient. Direct or indirect visualization of the airway may provide additional clinical information useful in determining the cause of the airflow obstruction and allowing a strategy for extubation to be developed, although this can be difficult in intubated or uncooperative patients.

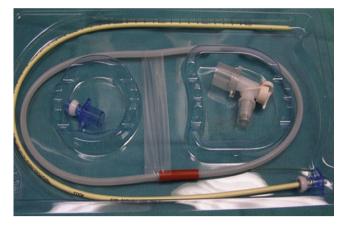


Fig. 8.15 Airway exchange catheter

When airway edema or other pathology is suspected, there are several techniques for evaluating the anatomy prior to tracheal extubation. The fiberoptic bronchoscope may be utilized via the nasal route to advance to the level of the glottis and inspect the upper airway. Appropriate patient selection, optimal positioning (upright), and counseling (in the non-sedated patient) to promote cooperation are key. Innovative and judicious regimens for sedation and analgesia (e.g., dexmedetomidine or remifentanil) may be required, coupled with an anti-sialagogue and effective suction capabilities. The newer videolaryngoscope devices may allow visualization of the glottis when carefully placed into the topicalized or sedated patient. Advanced laryngoscopic techniques offering "around the corner" visualization may overcome many of the limitations of conventional laryngoscopy's "line of sight." Mort compared the use of three different devices (Airtrag, GlideScope, and McGrath videolaryngoscopes) for airway inspection prior to ETT exchange and found that the devices provided an adequate view of most laryngeal structures ^[21]. Fiberoptic evaluation via the SGA is another described method for airway evaluation, but the full view of the immediate supraglottic and pharyngeal structures may be restricted. This technique does require extubation of the patient, and therefore the safety of extubation must be balanced against the clinical information that the technique may yield.

Use of an airway exchange catheter (AEC) to maintain access to the airway has been described and offers some advantages (Fig. 8.15). The AEC is based on the Seldinger technique and was originally described by Bedger and Chang, who passed a premarked 65-cm hollow "jet stylet" with distal side ports and a removable 15-mm adapter via an ETT, advancing it to the lower trachea, after which they removed the ETT ^[22]. A variety of AECs are currently available and anesthesiologists, otolaryngologists, and intensivists should

become familiar with their use, indications, advantages, limitations, and potential complications. Evidence-based studies that support a safe time for keeping the AEC in place are scarce; however, Mort described use of AECs in 51 patients with predicted difficult extubation. Forty-seven of the patients were able to be successfully re-intubated over the AEC; important factors for success were careful placement and appropriate internal diameter ^[23].

Invasive Airway Management

Otolaryngologist's Perspective

The Surgical Airway

The two surgical methods for obtaining emergent access to the airway are cricothyroidotomy and tracheotomy. Cricothyroidotomy is discussed in detail below while tracheostomy is discussed in detail in Chap. 18. Indications for an emergent "surgical" airway are severe laryngeal trauma and failed intubation. Relative indications that may lead secondarily to unsuccessful attempts with intubation are significant maxillofacial trauma, cervical spine injury, and the presence of any obstructing material such as blood, secretions, emesis, edema, or mass. In general, a surgical tracheotomy set should be available anytime a potentially difficult intubation is encountered.

In true airway emergencies a cricothyroidotomy is the favored technique for airway access ^[24]. The cricothyroid membrane is superficially located in the midline neck just above the readily palpated cricoid cartilage and therefore can be readily entered quickly and safely with minimal surgical instruments. If a relatively controlled setting is available with more time and personnel present an emergent tracheotomy may also be attempted. Cricothyroidotomy should not be performed in cases of laryngeal trauma, suspected subglottic obstruction, or in pediatric patients. In preparation for cricothyroidotomy the patient ideally should be placed in a supine position with neck extended to best expose the surgical landmarks; however, in emergent cases the head is often kept elevated to prevent acute airway obstruction. After the laryngeal landmarks are identified by palpation, a vertical stabbing incision is made through the skin and subcutaneous tissues at the level of the cricothyroid membrane. A vertical skin incision is preferred in order to enter a relatively avascular midline plane of dissection and avoid the anterior jugular veins. The horizontal incision of the membrane should be carried out directly into the cricothyroid space at which point the scalpel handle or a tracheal dilator can be used to expand

this opening. More often used during formal tracheostomy, a cricoid hook or retractors placed into the opening can be used to stabilize the airway at this time. Once the airway is successfully entered, either a small endotracheal or tracheostomy tube can be inserted into the cricothyroid space under direct visualization. After confirmation of placement by auscultation, end tidal CO_2 , tidal volume, or fiberoptic tracheoscopy, the tube should be secured to the patient's neck with sutures and a tracheal tie. Hemostasis should be obtained after the airway is effectively secured. Once stabilized, the patient should return to the operating room for conversion to a formal tracheotomy to avoid adverse long-term sequelae of cricothyroidotomy.

Cases in which anatomy is altered either by previous surgery, trauma, or bleeding may prove difficult especially in emergent situations when circumstances are less than ideal. As mentioned previously a cricothyroidotomy may be the preferred technique for these patients because of its relative ease^[25]. If complications are encountered during this procedure however, there are several useful strategies that may be employed. For instance, a syringe filled with saline and 18-gauge needle attached can be used to identify the tracheal lumen by entering the airway and then aspirating air bubbles as confirmation. The needle can then be cut down upon with a scalpel and airway successfully entered. If there is difficulty with endotracheal or tracheostomy tube placement, it may be entering a false passage around the tracheal lumen. Successful entry can be facilitated in these cases by placing retractors into the tracheal opening to stabilize the airway, inserting the tracheostomy tube over a guide wire, suction catheter, or flexible fiberoptic endoscope, or by using a smaller sized endotracheal tube.

Anesthesiologist's Perspective

Percutaneous Procedures

In cases when noninvasive strategies fail or the patient is clinically unstable, invasive means of ventilation and airway access are required ^[26]. These include transtracheal needle ventilation in which a large bore (14-gauge) angiocatheter is inserted directly into the tracheal lumen. There are a number of commercially available cricothyrotomy kits (H & H Emergency Cricothyrotomy Kit[®] and Rusch Quicktrach Emergency Cricothyrotomy Kit[®]) that are inserted based on the Seldinger Technique. Although the point of entry is midline and may be performed through the anterior tracheal wall, the cricothyroid membrane is preferred in emergent situations due to the presence of the complete ring of cricoid cartilage that can prevent unintended puncture through the posterior tracheal wall and resultant esophageal injury or medistinal insufflation. During placement it is critical to confirm that the needle or device is actually in the airway by the presence of the ability to aspirate air. Care must also be taken to ensure that the needle has not inadvertently entered either the internal jugular, carotid, or thyroid vessels.

Once properly placed, the means to supply effective jet ventilation must be available. Although the literature offers several suggestions on how to "make shift" jet ventilators from operating room or hospital supplies, it is the authors' recommendation that commercially produced manual or mechanical jet ventilators with pressure regulators be available and used exclusively for this purpose (Instrumentation Industries Manual Jet Ventilator with Regulator[®]) (Chap. 6). Although potentially useful in emergent situations, this technique can only typically be used as a temporizing measure until a secure airway can be obtained which may include a surgical airway placed by the otolaryngologist ^[27].

Management of the Pediatric Airway

Although a detailed discussion can be found in Chap. 19, the authors believe that for completion a description of the pediatric airway and the unique aspect of management should also be done here. The pediatric airway has several key anatomical differences pertinent to its management. The pediatric larynx is composed of relatively immature and therefore excessively floppy cartilage including the epiglottis that may prolapse into the airway causing obstruction and may impede intubation. For this reason a Miller laryngoscope may be more useful for intubation by compressing these structures anteriorly in order to better visualize the laryngeal inlet. In addition to being more cephalad, the narrowest portion of the pediatric airway is the cricoid ring compared to the glottis in the adult. Due to the small diameter, anything that decreases the airway caliber further (e.g., edema, mucous, foreign body) has significant impact on airway patency. The pediatric patient is unlikely to be cooperative for awake or sedated airway management, and the safest techniques involve deep general anesthesia with maintenance of spontaneous ventilation. Fiberoptic intubation may be performed in this manner with the instillation of local anesthetics prior to intubation as similar to adult patients. A frequently employed technique in failed or anticipated difficult pediatric airways is fiberoptic intubation via the LMA or other type of SGA. Currently, videolaryngoscopes are available in pediatric sizes and have been proven successful in difficult and abnormal pediatric airways ^[28].

Airway Emergencies

There are several classic clinical entities deserving of mention that commonly will require acute airway management. These patients will typically present to the emergency room with the anesthesiologist and otolaryngologist serving as consulting specialists. Successful management in each case depends on efficient triaging of the patient, initial stabilization and treatment, as well as effective communication between various participating disciplines.

Acute Epiglottitis

Although its incidence has dramatically declined over the past decades due to the advent of the H.influenza vaccine, cases still occur necessitating prompt and appropriate management ^[29]. Most commonly an infectious disease of children aged from 2 to 6, adult supraglottitis can also develop particularly in the immunocompromised population. Pediatric patients usually present with a rapid onset of febrile illness associated with sore throat, odonyphagia, and progressive difficulty eating and breathing. Signs on physical exam may include inspiratory stridor, drooling, and sitting in the classic "tripod" position while leaning forward. Ancillary studies showing an elevated white blood cell count or characteristic "thumbprint sign" on a lateral plain film from epiglottic edema may also prove helpful. During this acute presentation it is important not to excessively manipulate the child's airway with tongue depressors or flexible fiberoptic exams as this may precipitate laryngospasm and subsequent acute airway obstruction. The diagnosis of epiglottitis in these cases is clinical and appropriate management should ensue with broad spectrum antibiotics after blood cultures are taken. The OR should be immediately prepared for an emergent laryngoscopy and intubation with the possibility for bronchoscopy and tracheostomy if a surgical airway is needed. The diagnosis should be definitively confirmed upon visualization of a bright red and beefy supraglottis. After securing the airway (for the details of the anesthetic and airway management, see Chap. 19), the child should be transferred to the ICU for further monitoring and management.

In contrast to children, adults with acute supraglottitis typically have more insidious onset and presentation. Presenting symptoms are usually less severe and may include progressive odonyphagia and dysphonia, but overt respiratory distress is rare. Fiberoptic examination is safely performed in these patients demonstrating a boggy and erythematous epiglottis. Depending on clinical acuity, these cases can most often be managed conservatively with broad spectrum intravenous antibiotics and close observation ^[30].

Angioedema

Angioedema refers to the acute development of a non-tender and well-circumscribed area of mucosal swelling in the head and neck. Various forms of angioedema include hereditary, acquired, drug-induced, urticaria-associated, or idiopathic and is based on whether or not the reaction is mast-cell mediated. Regardless of the etiology, all cases involve an increase in vascular permeability caused by a release of specific factors such as histamine, serotonin, and bradykinin. When angioedema is associated with urticaria and anaphylaxis there is often a causative food or drug that can be identified. Common non-mast-cell mediated episodes are triggered by medications such as angiotensin-converting enzyme (ACE) inhibitors or C1 esterase deficiencies as found in hereditary cases [31]. Whatever the inciting factor, the presence of angioedema necessitates a systematic approach for evaluation and medical and airway management.

In a stable patient a detailed history may be taken to identify possible previous episodes and causative agents. Physical examination should include a careful inspection of all head and neck mucosal surfaces and particularly the oral cavity, oropharynx, hypopharynx, and larynx. The localization and extent of edema by fiberoptic laryngoscopy as well as overall clinical status should dictate subsequent management. Patients should be treated with intravenous steroids, H1 and H2 antihistamines, and epinephrine if anaphylaxis is present. Closely monitoring by continuous pulse oximetry and humidified oxygen by facemask should be administered. A compromised airway should be managed aggressively with urgent intubation to prevent acute obstruction. For extensive oropharyngeal edema, a nasotracheal approach while sitting upright may be the most prudent strategy ^[32].

Ludwig's Angina

Initially described by Wilhelm von Ludwig in 1836, this clinical entity represents an acute spreading cellulitis in the floor of mouth ^[33]. Most commonly of odontogenic origin, this lifethreatening infection can rapidly progress causing acute airway compromise. Ludwig's angina develops when infection occurs bilaterally in the sublingual and submandibular spaces. The mylohyoid line demarcates the path of spread depending on the site of odontogenic origin. Patients will typically present with signs of acute infection, throat pain, muffled voice, and difficulty breathing. On examination of the oral cavity, there will be firm and woody edema of the floor of mouth with posterosuperior displacement of the tongue. Prompt airway assessment and management is required to prevent asphyxiation, while close monitoring with serial fiberoptic examination may be adequate in fewer cases ^[34]. Surgical drainage is needed only in failures of intravenous antibiotic therapy or in the presence of a true neck abscess.

Maxillofacial and Laryngeal Trauma

As described previously, cases of maxillofacial and laryngeal trauma may pose unique challenges for airway management. As the first step in comprehensive trauma protocols, securing the patient's airway is of primary importance before resuscitation and secondary surveys can commence. In most of these cases, associated cervical spine injury will be suspected limiting the ability to extend the patient's neck for intubation. Alternate techniques such as in-line jaw stabilization and fiberoptic endoscopy must therefore be employed [35]. Maxillofacial trauma often leads to abundant oropharyngeal hemorrhage and secretions that obscure the laryngoscopist's view and impedes intubation attempts. In addition, due to the risk of skull base fracture associated with significant mid-facial trauma, nasotracheal techniques are contraindicated. Often these challenges may lead to failed translaryngeal intubation and the need for a surgical airway.

Laryngeal trauma presents different difficulties for airway management ^[36]. In cases of obvious laryngeal trauma characterized by stridor, dysphonia, palpable cartilaginous fractures, and cervical crepitus, traditional translaryngeal intubation attempts are discouraged. Most likely the patient's clinical condition will be deteriorating at this time and a surgical airway will most likely be needed. As previously mentioned, in these cases a tracheotomy is preferred over cricothyroidotomy in order to avoid possible further injury to subglottic structures.

Conclusion

Systematic identification and management of patients who may possess a "difficult airway" can often prevent airway emergencies. The most reliable predictor is a history of problematic intubation or airway crisis. Unfortunately, despite significant advances made in airway assessment and management, airway crises still occur in common clinical practice. Preparing for such emergent situations with standardized protocols and training may prevent negative and potentially disastrous outcomes from occurring. These include ready access to emergency equipment including standard and customized intubating laryngoscopes, flexible and rigid bronchoscopes, and tracheotomy sets in case a surgical airway is required. Key personnel must also be available including skilled nursing staff, surgical technicians, anesthesiologists, and otolaryngologists. Only through thoughtful preparation and efficient coordination of action can airway emergencies be successfully managed and defused appropriately. This is performed most effectively when close collaboration exists between specialists such as anesthesiologists and otolaryngologists.

Clinical Insights

For the Otolaryngologist (from the Anesthesiologist)

- Systematic preoperative assessment may identify patients with a potentially difficult airway
- A variety of noninvasive emergency ventilation techniques are available prior to the initiation of a surgical airway
- It is possible to combine techniques such as videolaryngoscopy with the fiberoptic bronchoscope
- Awake fiberoptic techniques can be safe, efficient and prove to be the best option for optimal patient outcome

For the Anesthesiologist (from the Otolaryngologist)

- Preoperative communication with the otolaryngologist particularly in complex airway cases is key to avoiding emergent situations
- For anticipated difficulty with ventilation, an "awake" method of intubation is preferred, as emergency tracheostomy is rarely a simple task

Clinical Pearls

For the Otolaryngologist (from the Otolaryngologist)

- Despite thorough clinical evaluation and preoperative planning, acute airway emergencies will still occur
- Effective preparation and collaboration with the anesthesiologist and airway team is key to the successful management of the difficult airway
- For cases with known difficult airways assure proper equipment is available (i.e., rigid bronchoscope, tracheostomy set)

For the Anesthesiologist (from the Anesthesiologist)

- Patients with head and neck pathology or status post radiation therapy are generally difficult to intubate, ventilate, and have a high GlideScope[®] failure rate
- An awake intubation, should be done awake with excellent airway local anesthesia
- When in doubt, maintain spontaneous ventilation of the patient
- ALWAYS have a plan B, plan C, etc.

References

- Wilson ME, Spiegelhalter D, Robertson JA, et al. Predicting difficult intubation. Br J Anaesth. 1988;61(2):211–6.
- 2. Mallampati SR, et al. A clinical sign to predict difficult intubation: a prospective study. Can Anaesth Soc J. 1985;32:429.
- Samsoon GL, Young JR. Difficult tracheal intubation: a retrospective study. Anaesthesia. 1987;42:487–90.
- 4. Hester CE, Dietrich SA, White SW, et al. A comparison of preoperative airway assessment techniques: the modified Mallampati and the upper lip bite test. AANA J. 2007;75:177–82.
- Myneni N, O'Leary AM, Sandison M, et al. Evaluation of the upper lip bite test in predicting difficult laryngoscopy. J Clin Anesth. 2010;22:174–8.
- Rosenblatt W, Ianus AI, Sukhupragarn W, et al. Preoperative endoscopic airway examination (PEAE) provides superior airway information and may reduce the use of unnecessary awake intubation. Anesth Analg. 2011;112:602–7.
- 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; 98:1269.

- Benumof JL. Laryngeal mask airway and the ASA difficult airway algorithm. Anesthesiology. 1996;84(3):686–99.
- 9. Cooper RM. Safe extubation. Anesth Clin North Am. 1995;13: 683.
- Brain AIJ. The laryngeal mask: a new concept in airway management. Br J Anaesth. 1983;55:801.
- Agro F, Frass M, Benumof JL. Current status of the Combitube: a review of the literature. J Clin Anesth. 2002;14:307–14.
- Edens ET, Sia RL. Flexible fiberoptic endoscopy in difficult intubations. Ann Otol Rhinol Laryngol. 1981;90(4 Pt 1): 307–9.
- Cooper RM. Use of a new videolaryngoscope (GlideScope[®]) in the management of a difficult airway: [L'usage d'un nouveau videolaryngoscope (GlideScope[®]) pour une intubation difficile]. Can J Anesth. 2003;50:611–3.
- 14. Aziz MF, Healy D, Kheterpal S, et al. Routine clinical practice effectiveness of the Glidescope in difficult airway management: an analysis of 2,004 Glidescope intubations, complications, and failures from two institutions. Anesthesiology. 2011;114:34–41.
- 15. McCarroll SM, Lamont BJ, Buckland MR, et al. The gum-elastic bougie: old but still useful. Anesthesiology. 1988;68:643–4.
- Benumof JL. Management of the difficult airway with special emphasis on awake tracheal intubation. Anesthesiology. 1991;75:1087–110.
- 17. Reed AP, Han DG. Preparation of the patient for awake fiberoptic intubation. Anesth Clin North Am. 1991;9:69.
- Henderson JJ, Popat MT, Latto IP, Pearce AC. Difficult airway society guidelines for management of the unanticipated difficult intubation. Anaesthesia. 2004;59:675.
- Bigenzahn W, Pesan B, Frass M. Emergency ventilation using the Combitube in cases of difficult intubation. Eur Arch Ororhinolaryngol. 1991;248:129–31.
- Ferson DZ, Rosenblatt WH, Johansen MJ, Osborn I, Ovassapian A. Use of the intubating LMA-Fastrach in 254 patients with difficultto-manage airways. Anesthesiology. 2001;100:114–50.
- Mort TC. Tracheal tube exchange: feasibility of continuous glottic viewing with advanced laryngoscopy assistance. Anesth Analg. 2009;108:1228–31.
- Bedger Jr RC, Chang JL. A jet-stylet endotracheal catheter for difficult airway management. Anesthesiology. 1987;66:221–3.
- Mort TC. Continuous airway access for the difficult extubation: the efficacy of the airway exchange catheter. Anesth Analg. 2007;105:1357–62.
- Morain WD. Cricothyroidostomy in head and neck surgery. Plast Reconstr Surg. 1980;65(4):424–8.
- 25. Bainton CR. Cricothyrotomy. Int Anesthesiol Clin. 1994;32(4): 95–108.
- Altman KW, Waltonen JD, Kern RC. Urgent surgical airway intervention: a 3-year county hospital experience. Laryngoscope. 2005;115:2101.
- Benumof J, Scheller MS. The importance of transtracheal jet ventilation in the management of the difficult airway. Anesthesiology. 1989;71:769–78.
- Redel A, Karademir F, Schlitterlau A, Frommer M, Scholtz LU, Kranke P, Kehl F, Roewer N, Lange M. Validation of the GlideScope video laryngoscope in pediatric patients. Paediatr Anaesth. 2009;19(7):667–71.
- Guldfred LA, Lyhne D, Becker BC. Acute epiglottitis: epidemiology, clinical presentation, management and outcome. J Laryngol Otol. 2008;122(8):818–23. Aug.
- Al-Qudah M, Shetty S, Alomari M, Alqdah M. Acute adult supraglotittis: current management and treatment. South Med J. 2010; 103(8):800–4. Aug.

- Seidman MD, Lewandowski CA, Sarpa JR, Potesta E, Schweitzer VG. Angioedema related to angiotensin-converting enzyme inhibitors. Otolaryngol Head Neck Surg. 1990;102(6): 727–31. Jun.
- Andreassen UK, Baer S, Nielsen TG, Dahm SL, Arndal H. Acute epiglottitis: 25 years experience with nasotracheal intubation, current management policy and future trends. J Laryngol Otol. 1992;106(12):1072–5. Dec.
- 33. Von Ludwig WF. Über eine in neuerer Zeit wiederholt hier vorgekommene Form von Halsentzündung. Medicinisches

Correspondenzblatt des Württembergischen ärztlichen Vereins, Stuttgart. 1836;6:21–5.

- Schwartz HC, Bauer RA, Davis NJ, et al. Ludwig's angina: use of fiberoptic laryngoscopy to avoid tracheostomy. J Oral Surg. 1974;32(8):608–11.
- Mulder DS, Wallace DH, Woolhouse FM. The use of the fiberoptic bronchoscope to facilitate endotracheal intubation following head and neck trauma. J Trauma. 1975;15(8):638–40.
- 36. Mace SE. Blunt laryngotracheal trauma. Ann Emerg Med. 1986;15(7):836–42.

Adult Sleep Apnea and Related Procedures

Menachem M. Weiner, Fred Y. Lin, David W. Jang, and Benjamin D. Malkin

Introduction

Obstructive sleep apnea (OSA) is the most prevalent breathing disorder in sleep, affecting up to 20% of the population, with approximately 5% experiencing excessive day-time sleepiness ^[1, 2]. The total economic burden of OSA including health-care costs, lost productivity, accidents, and loss of quality of life is substantial, accounting for billions of dollars per year, and is expected to increase ^[3]. As such, procedures related to improvement of OSA symptoms, as well as unrelated procedures in patients with OSA will likely increase in prevalence. Anesthesiologists and otolaryngologists will therefore face the challenges inherent to this patient population and a thorough understanding of the scope of the disease state is crucial.

Overview

OSA is characterized by the presence of inspiratory effort against a partially or completely obstructed upper airway, differentiating it from central sleep apnea, in which no inspiratory effort occurs. Repetitive airway obstruction during sleep is associated with episodic hypoxemia and sympathetic activation that is terminated by repeated arousals secondary to vigorous breathing efforts made to open the collapsed airway. The resulting compromise in inspiratory airflow is divided into distinct types of respiratory events: apneas, hypopneas, and respiratory effort-related arousals. Apnea is defined as a complete cessation of airflow lasting at

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least 10 s in duration whereas hypopnea is defined in two ways: (1) a 30% or greater reduction in airflow for at least 10 s with at least a 4% oxyhemoglobin desaturation; (2) a 50% or greater reduction in airflow for at least 10 s with at least a 3% oxyhemoglobin desaturation or an electroencephalogram (EEG) arousal. Respiratory effort-related arousals are sequences of breaths for at least 10 s with increasing respiratory effort or flattening of the nasal pressure waveform leading to an arousal from sleep, but do not meet the other criteria for apnea or hypopnea ^[4]. These arousals disrupt sleep and are responsible for excessive daytime hypersomnolence and memory loss. The degree of hypoxia varies with several factors including the length of the disturbance, lung volumes and oxygen reserves, and the degree of intrapulmonary shunt ^[5].

Although the magnitude of hypoxemia and the extent of the symptoms are important, the severity of OSA is usually expressed in terms of the number of apneas and hypopneas per hour of sleep, the apnea-hypopnea index (AHI). The American Academy of Sleep Medicine defines mild OSA as AHI 5–15, moderate OSA as AHI 15–30, and severe OSA as AHI > 30^[6].

The morbidity and mortality associated with OSA results from the multiple physiologic sequelae of repetitive airway obstructions followed by states of arousal ^[7]. These physiologic changes include reductions in oxygen saturation, increases in sympathetic output and tone, and arousals from sleep. The hypoxemia and hypercapnia occurring during apneic episodes not only cause arousal and disturbed sleep, but can also lead to significant cardiovascular derangements cardiac arrhythmias and ischemia, pulmonary hypertension and right ventricular hypertrophy, and systemic hypertension and left ventricular hypertrophy. Episodic arousal leads to a rise in sympathetic tone resulting in an increase in blood pressure, heart rate, and cardiac output. The reduced oxygen saturation can directly contribute to the incidence of cardiac arrhythmias, myocardial infarction, and stroke ^[7].

Risk factors for OSA include increased age, obesity, male gender, and the presence of craniofacial or soft tissue anomalies affecting the upper airway. Males are affected nearly

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twice as often as females with an incidence estimated in the range of 1 in 4 men and 1 in 10 women^[8]. Other factors such as nasal obstruction, diabetes, and smoking have also been found to be associated with OSA [1]. Although obesity is arguably the strongest predictor for OSA, clearly not all obese patients suffer from the disease and, conversely, not all patients with OSA are obese. Rather, OSA is a complex disease with multiple etiologic factors to consider. Certainly, anatomic abnormalities are usually identifiable and are the target for sleep surgeons. In children, adenotonsillar hypertrophy is the overwhelming cause of OSA and adenotonsillectomy has been shown to be an effective treatment (Chap. 19)^[9]. In contrast, adults may have anatomic obstructions at multiple levels including the nose and nasal cavity, soft palate, base of tongue, and pharyngeal walls. Obstructions may be soft tissue in origin as is the case in patients with enlarged uvulas, tonsils, or tongues. Obesity clearly contributes to this problem as fat can deposit along the soft tissues of the upper aerodigestive tract. However, anomalies of the craniofacial skeleton, including the size, shape, and position of the maxilla, mandible and hyoid bone, may be at least partly responsible and should also be considered.

Abnormally low neuromuscular tone is another major pathophysiologic mechanism, leading some to believe that OSA is largely a neurological, as opposed to a structural disorder ^[10]. Airway patency when awake is partially dependent on the resting tone of muscles associated with the palate, tongue, and hyoid. Muscle tone naturally decreases during sleep, leading to greater propensity for airway collapse during both inspiration and expiration. Abnormally low muscle tone may contribute to excessive collapse, leading to sleepdisordered breathing. Sleep muscle tone is determined by sleep stage and activation of airway mechanoreceptors by negative pressure and can be affected by medications such as sedatives. All these factors should be carefully scrutinized in the diagnosis and management of a patient with OSA ^[11].

Preoperative Considerations

Otolaryngologist's Perspective

The diagnosis of OSA should be considered when seeing a patient who seeks treatment for heavy snoring. Sometimes, bed partners will report that the patient gasps for air or stops breathing intermittently when asleep. Patients can be asked to produce an audio recording of their snoring habits to find preliminary evidence of apneic or hypopneic episodes. Patients should also be asked about excessive daytime sleepiness and whether they have difficulty concentrating and performing daily tasks. Care should be taken to assess whether daytime somnolence may pose a risk to the patient or others; M.M. Weiner et al.

for example, there should be a low threshold for initiating and expediting a workup in patients who operate heavy machinery. Quantifying the degree of sleepiness and screening for OSA may be accomplished in the office by using the Epworth Sleepiness Scale (ESS). Published in 1991, the ESS is a validated questionnaire asking patients to rate how likely they are to fall asleep or doze in certain situations. Their responses are scored, with higher scores corresponding to more severe disease ^[12].

Once OSA is suspected, the patient can be referred for a polysomnography (PSG), the gold standard for OSA diagnosis. As its name implies, this test measures several physiological parameters in the sleeping subject. Although variations exist, the standard PSG is performed at an overnight sleep center and has the following components: EEG, electro-oculogram (EOG), electromyogram (EMG), ECG, pulse oximetry, and airflow monitor. EMG leads are placed in the chin, but may also be used to monitor respiratory effort in the chest wall. Airflow is typically measured with a temperature or air pressure probe at the nose or mouth. Overall, the PSG provides insight into the complex interplay of events that occur during sleep. It also monitors for concurrent cardiovascular abnormalities that may occur. In a "split-night" PSG, patients receive CPAP titration during the second half of the study. When a diagnosis of OSA is confirmed, patients are often initially referred for CPAP therapy, which has been shown to be highly effective [13]. However, many patients find the therapy uncomfortable and have difficulty with adherence, eventually being referred for surgery.

The sleep surgeon faces the critical and difficult task of localizing the obstruction in the patient with OSA. Just as the pathophysiology of OSA is multifactorial, the sites of obstruction may be multiple. The physical examination is of utmost importance and a complete head and neck examination should be performed, including flexible fiberoptic laryngoscopy and nasal endoscopy. Although its utility has been questioned, the Müller maneuver, in which a patient forces inspiration against a closed airway, can help to determine the site of obstruction ^[14]. An early classification scheme was proposed by Fujita, who categorized patients into three types based on the site of obstruction. He found that type 1 patients. who had palatal obstruction only, responded well to the uvulopalatopharyngoplasty (UPPP), as compared to types 2 and 3 patients, who had multilevel and hypopharyngeal obstruction alone, respectively ^[15]. Later, Friedman published similar results and offered a more detailed staging system based on palate position, tonsil size, and body mass index (Fig. 9.1, Table 9.1). A variation of the Mallampati score, the Friedman stage is useful when deciding which procedure would be beneficial for a patient [16].

Beyond the physical examination, imaging and additional endoscopic studies may be useful in localizing the site of obstruction. Lateral cephalometry uses standardized

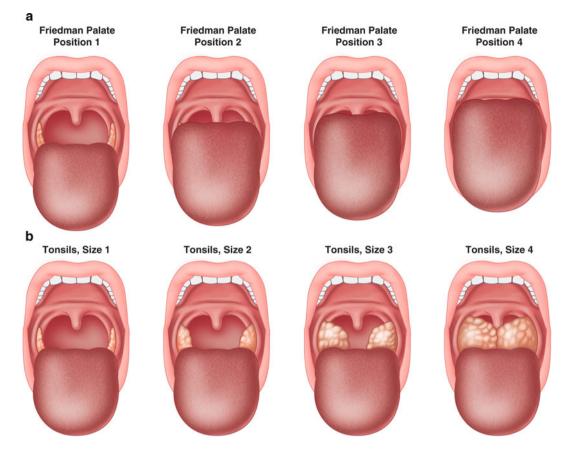


Fig. 9.1 (a) Friedman staging for sleep-disordered breathing. (Modified from Otolaryngol Clin North Am, 40, Friedman M, Schalch P, Surgery of the palate and oropharynx, 835, Copyright 2007, with permission from Elsevier.) (b) Tonsil staging for sleep-disordered breathing

	Friedman palate position	Tonsil size	Body mass index (kg/m ²)
Stage I	1	3, 4	<40
	2	3, 4	<40
Stage II	1, 2	0, 1, 2	<40
	3, 4	3, 4	<40
Stage III	3	0, 1, 2	Any
	4	0, 1, 2	Any
	Any	Any	>40

Table 9.1 Friedman staging system for OSA

anatomic measurements to determine whether the relative positions of the maxilla and mandible contribute to narrowing of the airway. Because this technique assesses bony structures, it can potentially identify patients who would benefit from skeletal surgery, such as maxillomandibular advancement (MMA). Imaging dynamic changes in the soft tissues of the upper airway in a sleeping patient through CT, MRI, and fluoroscopy is also an option, as is airway manometry, which measures pressure changes at various points in the airway. However, these techniques are not widely available. Drug-induced sleep endoscopy has recently emerged as a promising method to evaluate the dynamic airway. Upon being sedated with a low dose of propofol, the "sleeping" patient undergoes flexible endoscopy of the upper airway in order to identify areas of collapse and obstruction. Sleep endoscopy findings have been shown to positively correlate with the presence of OSA and with outcomes after palatal surgery ^[17].

Once a surgical plan is formulated, it is important for a patient to be optimized for surgery from a general medical standpoint. As previously noted, it is well documented that OSA is associated with hypertension, coronary artery disease, cerebrovascular disease, congestive heart failure, and cardiac arrhythmias. Although the mechanisms for this association are unclear, it is thought that chronic intermittent hypoxemia with reperfusion produces increased oxidative stress, vascular endothelial injury, increased sympathetic tone, and hypercoagulability, eventually leading to cardiovascular complications ^[18]. OSA has also been linked to gastroesophageal reflux disease (GERD). It has been speculated that acid reflux into the upper airway causes edema and contributes to airway obstruction, whereas the negative intrathoracic pressure produced during apneic episodes impairs

lower esophageal sphincter function and facilitates acid reflux into the larynx ^[19].

Anesthesiologist's Perspective

From the anesthesiologists' perspective, patients with OSA pose significant problems and incur increased risk during the perioperative period, including management of pulmonary and cardiovascular comorbidities, difficulties with bag mask ventilation and endotracheal intubation, increased sensitivity to anesthetic agents, and postoperative adverse events usually related to reduced ventilatory drive from anesthetic agents. In 2006 The American Society of Anesthesiologists (ASA) issued practice guidelines for the perioperative management of OSA patients. The purpose of the guidelines was to reduce the risk of adverse outcomes in patients with OSA and to improve perioperative care ^[20]. It was their opinion that perioperative risk increases in proportion to the severity of OSA.

Patients with OSA have an increased sensitivity to respiratory depressive agents compared to the normal population. This increased sensitivity to anesthetic and analgesic agents used during the perioperative period can further decrease pharyngeal tone and depress ventilatory responses to hypoxia and hypercapnia. The resulting effect can exacerbate the underlying anatomical and physiologic abnormalities associated with OSA. This propensity to obstruct the upper airway makes patients with OSA susceptible to a variety of serious complications during and after surgery including difficulty in delivering bag mask ventilation during induction and airway obstruction upon extubation. Additionally, patients with OSA are more likely to have comorbidities including uncontrolled hypertension, gastroesophageal reflux disease, coronary artery disease, congestive heart failure, and obesity. In one study, 24% of patients with OSA had significant postoperative complications, compared with 9% of patients in the control group^[21]. OSA has been associated with an increase in perioperative risk and postoperative complications ^[22, 23], especially after upper airway surgery ^[24], which is an independent risk factor for increased morbidity and mortality^[25]. In addition to the identified cardiovascular comorbidities, several investigators have reported that patients with OSA are also predisposed to cerebrovascular insufficiency and intracranial hypertension.

Optimal patient care begins with a tailored preoperative assessment to facilitate patient risk stratification and optimization, followed by formulation of an individualized perioperative management plan. Preoperative evaluation and formulation of an anesthesia management plan may decrease or eliminate the perioperative morbidity associated with patients with OSA. Bolden and colleagues ^[26] concluded that developing and following a perioperative anesthesia protocol is the best way to avoid an adverse outcome in patients suffering from OSA.

The ASA guidelines include recommendations to assess patients who may be at high risk based on clinical suspicion preoperatively ^[20]. The gold standard for the diagnosis of OSA is polysomnography (an overnight sleep study); however, it is expensive, time-consuming, and clearly unsuitable for general screening purposes. Typical symptoms of OSA are heavy snoring, witnessed apneas, lack of refreshing sleep, fatigue, and excessive daytime sleepiness. Patients may also have the characteristic stigmata of OSA on examination including obesity, short thick neck, nasal obstruction, tonsillar hypertrophy, narrow oropharynx, and retrognathia.

Due to the significant issues involved in anesthetizing a patient with OSA, anesthesiologists need to be knowledgeable of a practical preoperative screening tool for identifying patients more likely to have true OSA. The ASA has published a set of guidelines that help diagnose OSA based on elevated BMI, neck circumference, craniofacial abnormalities that obstruct the airway, snoring, airway exam anomalies, and hypersomnolence ^[20]. The Berlin questionnaire is another aid in diagnosing OSA preoperatively using three categories: snoring, sleep and fatigue, and arterial pressure, weight, and height. A patient is at increased risk of OSA if significant symptoms are present in two out of the three categories ^[27].

A recently developed concise and easy-to-use clinical screening tool for anesthesiologists is known as the STOP (snoring, tired, observed, pressure) questionnaire (Table 9.2) ^[28]. When incorporating four additional variables with the acronym BANG (BMI, age, neck circumference, gender) (Table 9.3), the sensitivity of the screening test improved. A patient is at high risk of OSA if 2 or more items score positive on the STOP questionnaire, or three or more items score positive on the STOP questionnaire with AHI greater than 5, greater than 15, and greater than 30 as cutoffs were 65.6%, 74.3%, and 79.5%, respectively. When incorporating the BANG variables into the STOP questionnaire, sensitivities were increased to 83.6%, 92.9%, and 100% with the same AHI cutoffs ^[28].

Application of continuous positive airway pressure (CPAP) is the most widely used and primary treatment for OSA because of its efficacy and low level of invasiveness. CPAP acts as a pneumatic splint to prevent occlusion of the airway during sleep, thereby significantly reducing apneas and hypopneas and the associated hypoxic and hypercapnic events. CPAP has been shown unequivocally to alleviate the symptoms of OSA. CPAP is an effective treatment modality for OSA, improving symptoms (e.g., excessive daytime sleepiness, quality of life) and reducing cardiovascular mortality ^[29]. CPAP has the potential to reduce cardiac rhythm abnormalities, stabilize variability of blood pressure, and improve the hemodynamic profile ^[30]. In an 18-year follow-up cohort study, CPAP improved overall survival and was

 Table 9.2
 STOP questionnaire
 [28]

S	Snoring	Do you snore loudly?
Т	Tired	Do you often feel tired or sleepy during the daytime?
0	Observed	Has anyone observed you stop breathing while sleeping?
Р	Pressure	Do you have or are you being treated for high blood pressure?

Table 9.3 BANG questionnaire [28]

В	BMI	Is your body mass index greater than 35 kg/m ² ?
А	Age	Are you older than 50 years old?
N	Neck circumference	Is your neck circumference greater than 40 cm?
G	Gender	Male gender?

found to be protective against cardiovascular death [1]; however, high level evidence is lacking in the perioperative context. It is unclear whether the use of CPAP therapy reduces adverse events attributed to OSA in rigorous randomized controlled trials. Taking into account the low level of invasiveness of CPAP therapy, its short-term use immediately preoperatively may be considered, particularly in patients with severe OSA ^[20]. Based on consensus opinion, patients already on treatment with CPAP should be advised to continue the treatment perioperatively, and to bring their CPAP machine to the hospital on admission keeping in mind that the use of nasal CPAP will not be possible in several situations due to the use of nasal packing or nasal casts ^[20]. Preoperative initiation of CPAP should be considered especially in cases of severe OSA. If possible, the patient should be treated with 4-6 weeks of CPAP before surgery because an increase in pharyngeal size and a decrease in tongue volume have been demonstrated on magnetic resonance imaging after CPAP therapy [31]. Patients with moderate and severe OSA who have been on CPAP therapy should continue CPAP therapy in the preoperative period ^[20].

The severity of OSA may be assessed from the patient history or from previous polysomnography results. Long-standing OSA may have systemic complications, which should be ascertained and evaluated preoperatively. These complications include hypoxemia, hypercarbia, polycythemia, and cor pulmonale. Electrocardiogram and pulse oximetry in the preoperative clinic can serve as simple screening tools.

As with all patients a thorough airway assessment is mandatory, along with an increased appreciation that patients with OSA are associated with difficult bag mask ventilation and may have anatomic features suggestive of a difficult intubation including obesity, increased neck circumference with limited neck mobility, retrognathia, micrognathia, macroglossia, tonsil and uvula hypertrophy, nasal obstruction, abnormal epiglottis position, anterior positioning of the larynx, and elongation of the airway (Chap. 8).

OSA is associated with several comorbidities. Cardiovascular disease ^[32, 33] including uncontrolled hypertension, heart failure, and arrhythmias ^[34] is common as well as cerebrovascular disease ^[35], metabolic syndrome ^[36], obesity, and gastroesophageal reflux ^[37]. It has also been associated with sudden death ^[38]. Preoperative risk stratification and patient optimization are required.

A consultation with the primary physician, cardiologist, or other appropriate specialists should be considered in patients with complicated comorbid conditions or multiple comorbidities. The purpose of the preoperative consultation is to optimize control of the comorbidities before surgery and to reduce the risk of surgical complications.

Upper airway surgery in sleep apnea patients can temporarily worsen the sleep apnea and lead to serious and potentially fatal complications. Early detection of impending airway problems may prevent these complications. Patients with more severe sleep apnea are at greater risk for perioperative respiratory complications and therefore must be admitted for postoperative monitoring with pulse oximetry.

Although laboratory tests should be ordered based on the history and physical, given the pathophysiology of OSA, it is the authors' opinion that a hemoglobin and electrocardiogram be obtained for all patients. The presence of polycythemia is suggestive of a more severe form of OSA, as is an Electrocardiogram (ECG) consistent with right axis deviation from right ventricular hypertrophy. Such ECG findings should initiate a cardiology consult and a workup for pulmonary hypertension. An arterial blood gas or chest roentgenogram is generally not necessary for preoperative assessment.

Intraoperative Considerations

Otolaryngologist's Perspective

Surgery of the Uvula and Palate

When patients fail CPAP therapy or are unable to tolerate it, a surgical option may be considered. The uvulopalatopharyngoplasty (UPPP), first described by Fujita et al., is the most common procedure performed to address snoring and OSA ^[39]. The UPPP involves amputation of the uvula and removal of redundant mucosa from the tonsillar pillars and soft palate with electrocautery or cold-knife technique (Fig. 9.2). Tonsillectomy is usually performed concurrently. The UPPP has been shown to be effective in carefully selected patients, namely non-obese patients with redundant mucosa in the soft palate and uvula and mild to moderate OSA ^[21]. Other techniques have recently been developed to

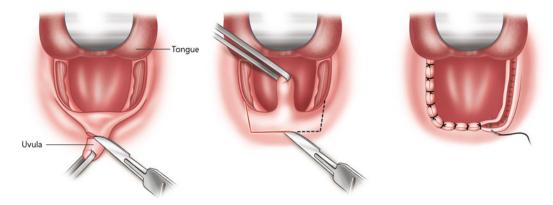


Fig. 9.2 Uvulopalatopharyngoplasty. (Modified from Otolaryngol Clin North Am, 40, Friedman M, Schalch P, Surgery of the palate and oropharynx, 835, Copyright 2007, with permission from Elsevier.)

address redundant soft tissue in the upper pharyngeal airway, but the underlying principles remain the same. A more aggressive resection involves palatal shortening with a Z-plasty or transpalatal advancement procedures. Laserassisted uvulopalatoplasty (LAUP) and the reversible uvulopalatal flap (UPF), as well as radiofrequency ablation, commonly performed in the office setting, are variations of the UPPP. More recently, palatal stiffening with electrocautery or implants has also been performed to achieve the same purpose. Regardless of the technique, atraumatic handling of soft tissue is required to minimize postoperative edema.

Although minor surgery of the upper pharyngeal airway is frequently performed in the office setting, general anesthesia should be considered for the majority of these procedures, as heavy bleeding and airway compromise may occur. Perioperative antibiotics with anaerobic coverage and dexamethasone should be given. After induction and endotracheal intubation, the surgeon stands at the head of the operating table with the patient in the supine position. A Crowe-Davis retractor is used to open the patient's mouth, pushing the endotracheal tube and the tongue inferiorly and providing excellent exposure of the tonsillar pillars and soft palate. The oropharynx is carefully examined and the amount of excess soft tissue is assessed. With cold knife or electrocautery, mucosal incisions are made along the soft palate and a tonsillectomy is performed. The mucosa is carefully dissected off, preserving muscle fibers. After hemostasis is achieved, the remaining mucosa and muscle is folded anteriorly and sutured with absorbable sutures. UPPP with tonsillectomy is typically performed in less than 30 min. Blood loss is typically minimal, but hemorrhage is a risk given the rich blood supply to the tonsillar bed. Appropriate laser precautions should be used for LAUP.

Surgery of the Base of Tongue and Hypopharynx

The major difficulty of surgically treating obstructive sleep apnea is that obstruction sites are often multilevel, with narrowing occurring at the hypopharynx and base of tongue reported to be a factor in up to 50% of patients ^[40]. Although the UPPP is the most commonly performed procedure for sleep apnea, failure to address the tongue base and hypopharynx in certain patients can limit the success of sleep apnea surgery. In their series, Riley et al. found retroglossal obstruction to be the most common factor for UPPP failure ^[41]. Many techniques have been described to treat the tongue base and hypopharynx, but the most common are hyoid suspension, genioglossus advancement, and MMA.

Hyoid suspension was first described by Riley et al. in 1986 in conjunction with an inferior sagittal split of the mandible to move the hyoid superiorly and anteriorly ^[42]. The hyoid was originally suspended to the mandible with fascia lata, but the procedure was later modified to suspend from the upper border of the thyroid cartilage. Although it is possible to perform this under local anesthesia and sedation, the procedure is often combined with other sleep surgeries and is therefore performed under general anesthesia. Perioperative antibiotics and steroids are given, and local anesthesia with epinephrine is injected transversely above the hyoid to minimize bleeding. The skin incision is then made above the hyoid and subplatysmal flaps are raised. The strap muscles are split, exposing the thyroid cartilage. A steel wire suture is then used to pierce the thyroid cartilage on one side and exit out the contralateral side. Taking special care not to enter the pharynx, the suture is then passed under the hyoid and fixated once the thyroid cartilage and hyoid have been brought together, with the overall effect of moving the tongue base and stiffening the upper airway. A drain is often placed and the skin incision is then closed. Postoperative dysphagia is often encountered and self-limited aspiration is sometimes seen [43].

Genioglossus advancement involves advancement of a portion of the mandible containing the genial tubercle with the attached genioglossus muscle to prevent the posterior collapse of the base of tongue ^[44]. The major risks of the procedure include bleeding, damage to the tooth roots and mental nerve, mandibular fracture and aesthetic changes to the chin. The procedure is performed under general anesthesia and patients stay as an inpatient overnight for observation (this will be likely for all patients with OSA severe enough to warrant surgery). Perioperative antibiotics and steroids are given and local anesthesia with epinephrine is injected into the gingivobuccal sulcus to reduce bleeding. Palpation of the floor of mouth is then performed to estimate the location of the genioglossus attachment and an incision is then made from canine to canine in the gingivobuccal sulcus. The dissection is then made in a subperiosteal plane, exposing just enough bone to perform the osteotomy. A 2-cm×1-cm rectangular window should be planned approximately 4-5 mm below the tooth roots. After the bone cuts are made, a temporary screw is placed to assist in the advancement of the segment. The advancement is complete when the inner cortex of the segment is anterior to the outer cortex of the mandible. The segment is then rotated approximately 30°, the outer cortex and marrow are removed and the segment is secured in placed with a lag screw (Fig. 9.3). The incision is then closed in layers with reconstruction of the mentalis muscle and a dressing is applied. Postoperative tongue and floor of mouth edema as well as floor of mouth hematomas can be seen, therefore necessitating close observation overnight in the intensive care or step-down unit [45].

Maxillomandibular advancement remains the most successful operative procedure to treat OSA after tracheostomy, with a long-term success rate of approximately 90% ^[46]. The advancement of the maxillary and mandibular complexes enlarges the airway through expansion of the surrounding skeletal framework. It also improves the tension and collaps-ibility of the suprahyoid and velopharyngeal musculature as well as reducing lateral pharyngeal wall collapse ^[47]. The procedure is performed under general anesthesia with nasotracheal intubation. Arch bars are placed on the

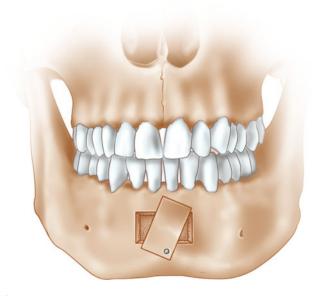


Fig. 9.3 Genioglossus Advancement. (Modified from Genioglossus advancement and hyoid myotomy under local anesthesia. *Otolaryngol Head Neck Surg* 2003;129(1):85–91.)

maxillary and mandibular teeth and a Le Fort I maxillary osteotomy is performed. After identification of the descending palatine artery, the maxilla is advanced approximately 10-14 mm anteriorly and then stabilized with four plates. The mandibular osteotomy is then performed via the sagittal split technique, with careful preservation of the inferior alveolar nerve; the mandible is advanced the same distance as the maxilla, thereby maintaining normal occlusion. The mandible is then stabilized by intermaxillary fixation with wires and rigidly fixated with lag screws and plates (Fig. 9.4). The wired intermaxillary fixation is then replaced with heavy dental elastics to guide the teeth into proper occlusion. Postoperatively, the patients are extubated awake in the operating room and monitored for at least a day in the intensive care unit. Facial and airway edema are often common and necessitate close monitoring [48].

It should be noted that tracheostomy (Chap. 18) is considered the only definitive corrective surgery and the gold standard procedure to cure OSA, as it allows complete bypass of the upper airway. However, given the poor patient acceptance of having a tracheostomy, it is usually reserved for treatment of patients with severe OSA with medical sequelae and lack of other viable surgical options.

Anesthesiologist's Perspective

Airway reconstructive surgery for sleep apnea often causes blood to enter the airway. In addition, OSA patients are particularly sensitive to respiratory depressant and airway effects of anesthetic medications. It is therefore believed to be safest to perform these surgeries under general anesthesia with a secure airway.

Use of sedative hypnotics, anxiolytic agents, and narcotics should be avoided before surgery in patients with OSA. Patients with OSA are very sensitive to the effects of sedation ^[49]. These agents have been reported to lead to sudden death, even in the preoperative holding area ^[50]. Opioids suppress respiratory drive, blunt the arousal response, and may lead to life-threatening hypoxemia. Benzodiazepine agonists effect upper airway dilator muscle tone and worsen sleepdisordered breathing ^[7]. If an OSA patient requires sedation or an anxiolytic immediately before surgery, these patients should be given supplemental oxygen and monitored with continuous pulse oximetry.

Obesity is common in patients with OSA leading to increased intra-abdominal fat, intra-abdominal pressure, higher incidence of hiatal hernia, and an increased risk of GERD. Obese patients tend to have a larger volume of gastric fluid, a lower gastric pH, and are at increased risk of aspiration during anesthesia induction or on extubation. To reduce the risk of aspiration, the use of proton pump

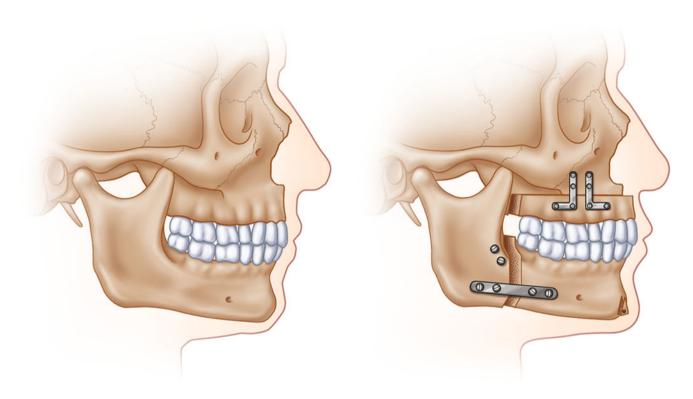


Fig. 9.4 Maxillomandibular Advancement. (Modified from Otolaryngol Clin North Am, 40, Li KL, Hypopharyngeal airway surgery, 849, Copyright 2007, with permission from Elsevier.)

inhibitors, non-particulate antacids, rapid sequence induction, and cricoid pressure may be considered. Following surgery, the stomach should be suctioned.

OSA is a risk factor for difficult endotracheal intubation ^[51]. In a retrospective matched case–control study of 253 patients, difficult intubations were found to occur eight times as often in the patients with OSA versus the control group (21.9% vs 2.6%, P < 0.05)^[51]. In patients undergoing uvulopalatopharyngoplasty (UPPP), an AHI greater than 40 was a predictor for difficult intubation ^[52]. Adequate airway preparation should be made according to ASA difficult airway management guidelines [53]. Some measures to deal with this are to ensure that the patient is positioned in the sniffing position, building a ramp from the scapula to the head and adequate preoxygenation. If a difficult airway is suspected, the patient should be managed conservatively; an awake flexible bronchoscopic intubation is considered optimal^[31]. If induction before intubation is considered appropriate, a rapid sequence induction is advisable; the risk of aspiration in obese and OSA patients is high as a result of poor lower esophageal sphincter tone. For tonsillectomies or intraoral resections, the use of RAE or wire reinforced anode tubes are ideal to permit exposure while minimizing endotracheal tube kinking when a Crowe-Davis retractor is used. If lasers are to be used, airway fire precautions and the use of laser safe endotracheal tubes must be deployed (Chap. 10).

Edema caused by upper airway surgery or a difficult intubation may cause airway compromise, especially in those patients with severe apnea ^[54]. Tissue edema occurs in all surgeries, even after laser and radiofrequency procedures. Administration of systemic steroids is a reliable method of reducing edema in the upper airway. The preferred corticosteroid agent is dexamethasone (10–15 mg/ dose in adults) because it has the lowest sodium retention of all the steroid agents. For optimal effect, dexamethasone should be administered before surgery and several times postoperatively ^[7].

Monitoring during surgery for OSA is dependent on patient-related factors. In addition to the standard ASA monitors, invasive monitoring of arterial blood pressure and central venous pressure should be undertaken if the patient's comorbidities warrant it.

OSA predisposes to the respiratory depressant effects of sedatives, opioids, and inhaled anesthetics. This sensitivity is largely a result of the propensity of airway collapse, sleep deprivation, and the blunting of the physiologic response to hypercarbia and hypoxia. The use of short-acting agents such as propofol, remifentanil, and desflurane and agents with limited respiratory depression, such as dexmedetomidine, is recommended, with the avoidance or minimization of the use of longer-acting anesthetic drugs that can have carry-over sedation effects. Due to the significant airway issues involved, tracheal extubation should be performed only after the OSA patient is fully conscious, airway patency confirmed, and full reversal of neuromuscular blockade verified. Deep extubation is not an option. The hazards include not only the loss of the airway, but also the risk of post-obstructive (negative pressure) pulmonary edema as the patient generates negative pressure trying to breathe against a closed glottis or collapsed airway ^[55]. The patient should be carefully monitored after extubation to ensure that the upper airway remains patent and unobstructed.

The period immediately after extubation is a potentially hazardous time due to the risks of airway obstruction, narcosis, residual anesthesia, and residual neuromuscular blockade. Placing the patient in a non-supine position (semi-upright or lateral) may be helpful for patients whose airway obstruction is worse in the supine posture. CPAP is widely used to reduce the risk of airway obstruction in postoperative patients. CPAP at the preoperative settings should be instituted after extubation. Administration of CPAP immediately after extubation maintains spirometric lung function at 24 h after laparoscopic bariatric surgery better than CPAP in the postanesthesia care unit (PACU)^[56]. The CPAP pressure may need to be adjusted in the postoperative setting to obtain optimal efficacy. Patients who are unable to sustain spontaneous breathing through an obstructed airway may need reintubation ^[31].

Postoperative Considerations

Otolaryngologist's Perspective

Patients who undergo sleep surgery will experience increased airway edema for the first 24-48 h after surgery. Dexamethasone should be started during the procedure, continued postoperatively and tapered if needed. Other measures include elevating the head of bed and the use of ice chips and ice packs. Generally, patients should be allowed to use CPAP or bilevel positive airway pressure (BiPAP), except in certain cases where subcutaneous emphysema may develop. In a patient with an already compromised airway who is receiving sedatives, hypoxia and the associated cardiovascular sequelae are potentially serious complications. Although there are no clear guidelines regarding postoperative monitoring, it is clear that some patients require a higher level of monitoring than others. Many non-obese patients with mild OSA undergoing UPPP may safely be discharged after a few hours in the PACU, while others who are at higher risk from more extensive surgeries may remain intubated and require full ICU monitoring for more than 24 h. Patients at an intermediate risk typically stay overnight with continuous pulse

The literature offers a wide range of complication rates after surgery to correct OSA. Earlier studies showed higher complication rates with UPPP. However, a more recent retrospective analysis of 3130 patients in the Veterans Affairs system showed the overall 30-day perioperative complication rate after UPPP to be 1.5%, with a 0.2% mortality rate. The authors reported that severity of AHI, body mass index, and medical comorbidities were predictive factors ^[58]. Other studies have also shown that multi-site surgery is another risk factor. In such patients, preoperative medical optimization and careful postoperative monitoring is recommended.

Anesthesiologist's Perspective

Abnormalities of breathing may persist into the postoperative period in patients with OSA. Studies have shown that the severity of OSA is typically unchanged or worse for the first two nights after UPPP^[59]. The first 24 h after surgery is likely the most critical time for complications, although deaths from complications have occurred later, potentially from the accumulated effects of sleep deprivation, opioid agents, and rapid eye movement (REM) sleep rebound. This could be a critical period for patients with OSA since monitoring may not be as vigorous, the effects of sedatives, anesthetics, and opioid analgesics may persist causing a reduction of the arousal response thereby worsening hypoxemia and hypercarbia during sleep and postoperative swelling in the upper airway may further compromise the patency of the narrowed upper airway lumen^[7]. In OSA patients undergoing UPPP, predisposing factors for respiratory complications included a higher preoperative AHI (AHI 69 vs 43, P<0.008) and lower minimal levels of oxygen saturation (minimal O₂ Sat 71.9% vs 77.8%, P < 0.01)^[24].

Immediate postoperative complications may intuitively be attributed to the negative effects of sedative, analgesic, and anesthetic agents, which can worsen OSA by decreasing pharyngeal tone, and the arousal responses to hypoxia, hypercarbia, and obstruction. Later events are, however, more likely to be related to postoperative REM sleep rebound which typically occurs on the third or fourth postoperative day. In the severe OSA patient, REM sleep rebound could conceivably act in conjunction with opioid administration and supine posture to aggravate OSA. Sleep studies performed in patients undergoing major abdominal surgery and open-heart surgeries have shown the suppression of REM and slow-wave sleep after surgery with a subsequent rebound in the late postoperative period. The return of REM sleep in the late postoperative period, at a time when oxygen therapy would have been discontinued, has been linked to significant respiratory abnormalities in a group of elderly patients who underwent abdominal vascular surgery ^[60].

Postoperative complications for patients with OSA include respiratory problems such as oxygen desaturation, apnea, respiratory arrest, and cardiovascular problems such as hypertension, arrhythmias, and cardiac arrest [61]. In order to minimize such complications, the ASA guidelines recommend supplemental oxygen until the patient is able to maintain their baseline oxygen saturation on room air. CPAP has been advised in patients who were receiving treatment preoperatively, but remains controversial in those patients who were not. If frequent or severe obstruction or hypoxemia occurs during postoperative monitoring, initiation of CPAP should be considered ^[20]. Either a semi-upright or lateral position can prove beneficial. The AHI and hypoxemia tend to improve when OSA patients sleep in the lateral or prone position, or with the head of the bed elevated. Sleep apnea is usually worse when supine, because of posterior collapse of the base of tongue. Following surgery, elevation of the head of the bed reduces soft tissue edema, turbinate swelling, and typically improves the nasal airway. Although the literature is insufficient to provide definitive guidance in the postoperative period, most physicians agree that after airway surgery, the head of the bed should be elevated and the supine position should be avoided ^[20].

All opiates including morphine, meperidine, hydromorphone, and fentanyl lead to a dose-dependent reduction of respiratory drive, respiratory rate, and tidal volume, which can cause hypoventilation, hypoxemia, and hypercarbia. These effects are of particular concern in the patient with OSA because the frequency and severity of the respiratory events worsen after administration. This poses a dilemma for these patients because upper airway reconstructive surgeries are often quite painful and typically require opioid agents for 10–14 days for adequate pain control ^[59]. It has been assumed that the more potent opioids, which are administered via intramuscular or intravenous routes, cause more respiratory suppression than those given by an oral route. Nonetheless, the literature is insufficient to evaluate the effects of different analgesic techniques ^[12].

A multimodal approach for analgesia is therefore advocated, where a combination of analgesics from different classes is used. Medications such as nonsteroidal antiinflammatory drugs, acetaminophen, tramadol, ketamine, gabapentin, pregabalin, clonidine, and dexamethasone are used to alleviate the opioid-related adverse effects of respiratory depression in susceptible patients. Postoperative oxygen desaturations were 12–14 times more likely to occur in OSA patients receiving postoperative oral or parenteral opioids vs non-opioid analgesic agents ^[62].

After UPPP, it is recommended that all patients be observed in the PACU with continuous pulse oximetry for a longer period than a patient without OSA. Continuous pulse oximetry is believed to be the easiest and most reliable method for early detection of postoperative hypoventilation because it can alert the nursing staff and physician of an impending airway complication. Continuous pulse oximetry with an audible alarm that can be heard by the nursing staff should be used for all OSA patients following non-airway or upper airway surgery. Intermittent checks of oxygen saturation typically have no benefit because putting on the oximetry probe usually awakens the patient. Although there is no consensus as to whether electrocardiographic monitoring affords any protection to the patient with sleep apnea, it should be considered in patients with significant cardiac disease or arrhythmias.

Older publications had recommended intensive care unit monitoring for oxygen saturation and cardiac arrhythmias, whereas others have advocated intensive care unit monitoring because of the high reported incidence of serious airway complications (13–25%) following UPPP. Newer publications have noted a much lower risk of airway complications (1.4%) likely caused by more aggressive perioperative treatment of tissue edema and avoidance of excessive sedation^[63, 64]. Except for the sickest of sleep apnea patients, intensive care unit monitoring is rarely required.

Unfortunately, the literature is insufficient to offer guidance regarding which patients can be safely managed on an outpatient basis as opposed to an inpatient basis or how long the patient should be monitored in the surgical facility ^[20]. In a recent report ASA consultants were surveyed using a non-validated scoring system about opinions regarding outpatient surgery in patients with OSA. The consultant's opinions suggested that a patient with mild OSA undergoing UPPP or nasal surgery was not at increased risk for complications, whereas a patient with moderate OSA undergoing UPPP was at increased risk of complications ^[20].

Depending on the severity of the OSA, the extent of the surgery, the type of anesthetics administered, and the postoperative analgesics required, the patient might shift to the higher end of the risk continuum, increasing the need for step-down care. The anesthesiologist should ensure that a postoperative monitored bed is available for a patient with a high AHI undergoing major surgery or airway surgery. A monitored bed refers to an environment with continuous pulse oximetry monitoring with the possibility of early nursing intervention (e.g., step-down unit or remote continuous pulse oximetry with telemetry). Continuous pulse oximetry monitoring and CPAP therapy may be necessary if recurrent PACU respiratory events occur such as oxygen desaturation, apnea, or pain-sedation mismatch. To summarize patients with mild OSA undergoing UPPP or other pharyngeal airway surgeries may be arranged in an ambulatory setting, but should be observed for at least several hours before discharge. If an apneic or hypoxic event is noted, the patient must be observed for an additional 5 h, but could be discharged if no further episodes are noted and no further opioid pain medicines are required. If an ambulatory or office-based practice does not have such PACU resources, then patients with OSA should not undergo surgery in the facilities. All moderate or severe OSA requiring monitored anesthetic care and sedation or general anesthesia should be admitted overnight with continuous pulse oximetry monitoring and typically include an observation of sleep ^[7].

In general, monitoring of vital signs for the OSA patient should be more frequent. Nursing checks should specifically monitor for respiratory rate, depth of breathing, presence of snoring, and to verify that there is no apnea, hypopnea, or labored breathing ^[7]. CPAP can be safely used after most upper airway surgeries to prevent respiratory events and oxygen desaturation during sleep and should be used in all patients who were able to use it before surgery.

The observation of recurrent PACU respiratory events can be used as a reliable second phase indicator to determine whether the OSA patient requires continuous postoperative monitoring. A PACU respiratory event occurs when a patient has (1) one episode of apnea for greater than 10 s, (2) three episodes of bradypnea of less than eight breaths per minute in 130 min time block, (3) pain-sedation mismatch, or (4) three episodes of oxygen desaturations to less than 90% in 130 min time block. High pain scores and high sedation levels observed simultaneously characterize pain-sedation mismatch. Recurrent PACU respiratory events occur when any one of the PACU respiratory events occurs in two separate 30-min time blocks (not necessary to be the same event) ^[65].

Patients who have recurrent PACU respiratory events are more likely to have postoperative respiratory complications ^[66]. It may be prudent to monitor these patients continuously postoperatively with pulse oximetry in an area where early medical intervention can occur. These patients may also require commencement of postoperative CPAP therapy.

Conclusion

Patients with OSA challenge both the otolaryngologist and anesthesiologist. It is critical that these specialists work together to improve surgical outcome. This chapter highlighted both the surgical and anesthetic concerns in order to improve the collaborative care of these patients.

Clinical Insights

For the Otolaryngologist (by the Anesthesiologist)

- The anesthesiologist should be made aware of the severity of the OSA.
- Although it should be assumed that all OSA patients may be more difficult to ventilate or intubate, there are some with macroglossia, retrognathia, or micrognathia who present an additional challenge to secure an airway. In these patients, the surgeon may request to have a difficult airway set or tracheostomy set in the operating room or to be ready to assist with a fiberoptic intubation.
- Patients should be instructed to bring their own CPAP machine to the surgery facility for postoperative use.
- Initiation of CPAP therapy prior to surgery should be considered especially in patients with severe OSA.
- Patients with mild sleep apnea can likely be cared for in an ambulatory setting.
- Patients with moderate or severe sleep apnea must be admitted to the hospital for overnight pulse oximeter monitoring and sleep observation.

For the Anesthesiologist (by the Otolaryngologist)

- OSA is underdiagnosed, so it is important to recognize risk factors in patients undergoing any type of surgery.
- For UPPP, an oral RAE or anode tube should be utilized to avoid kinking of the endotracheal tube.
- Uncontrolled hypertension can cause excessive bleeding and edema, especially in patients who have had osteotomies.
- Retraction during MMA can often cause a vagal response.
- Although opioids place OSA patients at risk for hypoxia, postoperative pain after sleep surgeries tends to be severe. Pain medications should be carefully titrated to balance these considerations.

Clinical Pearls

For the Otolaryngologist (by the Otolaryngologist)

- Communicating with your anesthesiologist about your preoperative assessment of the patient's airway is of utmost importance.
- The surgeon should be present during induction and awakening to assist in airway emergencies that may arise.
- Obtaining adequate hemostasis is critical in preventing postoperative hematomas that may compromise the airway.
- Panorex films showing the tooth roots and inferior alveolar nerve are essential for preoperative planning in procedures involving the maxilla and mandible.
- Dental occlusion should be maintained after advancement surgery. A dental splint is often helpful in accurately realigning dental occlusion during advancement surgery.
- Identification and control of the descending palatine artery during advancement surgery is crucial prior to distraction.
- Intraoperative use of steroids and postoperative head of bed elevation will help reduce surgical site edema.

For the Anesthesiologist (by the Anesthesiologist)

- Anticipate possible difficult airways. Never underestimate how difficult the airway can be in a patient with OSA.
- OSA patients can have significant associated pulmonary and cardiovascular disease. The preoperative assessment should ascertain OSA severity and comorbidities.
- Avoid or minimize opioids if possible.
- Even a small dose of midazolam can have detrimental effects on postoperative ventilation and pharyngeal patency. Avoid it.
- Consider nerve blocks and/or local infiltration to augment postoperative pain management.
- Full reversal of neuromuscular blockade should be confirmed before extubation, and consider extubation in a non-supine position.

- Postoperative disposition of the OSA patient should be based on the severity of the sleep disorder, recurrent PACU respiratory events, and the need for opioid analgesia.
- The care of these patients requires vigilance before, during, and after surgery to minimize risks associated with their underlying diseases.

References

- Young T, Finn L, Peppard PE, et al. Sleep disordered breathing and mortality: eighteen-year follow-up of the Wisconsin sleep cohort. Sleep. 2008;31:1071–8.
- Young T, Peppard PE, Gottlieb D. Epidemiology of obstructive sleep apnea: a population health perspective. Am J Respir Crit Care Med. 2002;165:1217–39.
- Alghanim N, Comondore VR, Fleetham J, et al. The economic impact of obstructive sleep apnea. Lung. 2008;186:7–12.
- Kushida CA, Littner MR, Morgenthaler T, et al. Practice parameters for the indications for polysomnography and related procedures: an update for 2005. Sleep. 2005;28:499–521.
- Hillman DR, Platt PR, Eastwood PR. Anesthesia, sleep, and upper airway collapsibility. Anesthesiol Clin. 2010;28:443–55.
- Iber C, Ancoli-Israel S, Cheeson A, et al. The AASM manual for the scoring of sleep and associated events, rules, terminology and technical specifications. Westchester: American Academy of Sleep Medicine; 2007.
- Mickelson SA. Preoperative and postoperative management of obstructive sleep apnea patients. Otolaryngol Clin North Am. 2007;40:877–89.
- Young T, Evans I, Finn I, et al. Estimation of the clinically diagnosed proportion of sleep apnea syndrome in middle-aged men and women. Sleep. 1997;20:705–6.
- Mitchell RB, Boss EF. Pediatric obstructive sleep apnea in obese and normal-weight children: impact of adenotonsillectomy on quality-of-life and behavior. Dev Neuropsychol. 2009;34(5): 650–61.
- Broderick M, Guilleminault C. Neurological aspects of obstructive sleep apnea. Ann NY Acad Sci. 2008;1142:44–57.
- Woodson BT, Franco R. Physiology of sleep disordered breathing. Otolaryngol Clin North Am. 2007;40(4):691–711.
- Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep. 1991;14(6):540–5.
- Patel SR, White DP, Malhotra A, et al. Continuous positive airway pressure therapy for treating sleepiness in a diverse population with obstructive sleep apnea: results of a meta-analysis. Arch Intern Med. 2003;163(5):565–71.
- Doghramji K, Jabourian ZH, Pilla M, Farole A, Lindholm RN. Predictors of outcome for uvulopalatopharyngoplasty. Laryngoscope. 1995;105:311–4.
- Fujita S, Conway W, Sicklesteel J, Witting R, Zorick F, Roehrs T, Roth T. Evaluation of the effectiveness of uvulopalatopharyngoplasty. Laryngoscope. 1985;95:70–4.
- Friedman M, Ibrahim H, Bass L. Clinical staging for sleepdisordered breathing. Otolaryngol Head Neck Surg. 2002; 127(1):13–21.
- Iwanaga K, Hasegawa K, Shibata N, Kawakatsu K, Akita Y, Suzuki K, Yagisawa M, Nishimura T. Endoscopic examination of obstructive sleep apnea syndrome patients during drug-induced sleep. Acta Otolaryngol Suppl. 2003;550:36–40.

- Shamsuzzaman AS, Gersh BJ, Somers VK. Obstructive sleep apnea: implications for cardiac and vascular disease. JAMA. 2003;290:1906–14.
- Zanation A, Senior B. The relationship between extraesophageal reflux (EER) and obstructive sleep apnea (OSA). Sleep Med Rev. 2005;9(6):453–8.
- 20. Gross JB, Bachenberg KL, Benumof JL, et al. Practice guidelines for the perioperative management of patients with obstructive sleep apnea: A report by the American Society of Anesthesiologists Task Force on perioperative management of patients with obstructive sleep apnea. Anesthesiology. 2006;104:1081–93.
- Gupta RM, Parvizi J, Hanssen AD, Gay PC. Postoperative complications in patients with obstructive sleep apnea syndrome undergoing hip or knee replacement: a case–control study. Mayo Clin Proc. 2001;76:897–905.
- Hwang D, Shakir N, Limann B, et al. Association of sleepdisordered breathing with postoperative complications. Chest. 2008;133:1128–34.
- 23. Liao P, Yegneswaran B, Vairavanathan S, et al. Postoperative complications in patients with obstructive sleep apnea: a retrospective matched cohort study. Can J Anesth. 2009;56:819–28.
- Kim JA, Lee JJ, Jung HH. Predictive factors of immediate postoperative complications after uvulopalatopharyngoplasty. Laryngoscope. 2005;115:1837–40.
- Marshall NS, Wong KKH, Liu PY, et al. Sleep apnea as an independent risk factor for all-cause mortality: the Busselton Health Study. Sleep. 2008;31:1079–85.
- Bolden N, Smith CE, Auckley D. Avoiding adverse outcomes in patient with obstructive sleep apnea (OSA): Development and implementation of a perioperative OSA protocol. J Clin Anesth. 2009;21:286–93.
- Chung F, Ward B, Ho J, et al. Perioperative identification of sleep apnea risk in elective surgical patients, using the Berlin questionnaire. J Clin Anesth. 2006;19:130–4.
- Chung F, Yegneswaran B, Liao P, et al. STOP questionnaire: a tool to screen patients for obstructive sleep apnea. Anesthesiology. 2008;108:812–21.
- Gay P, Weaver T, Loube D, et al. Evaluation of positive airway pressure treatment for sleep related breathing disorders in adults. Sleep. 2006;29:381–401.
- Kaye DM, Mansfield D, Naughton MT. Continuous positive airway pressure decreases myocardial oxygen consumption in heart failure. Clin Sci (Lond). 2004;106:599–603.
- Jain SS, Dhand R. Perioperative treatment of patients with obstructive sleep apnea. Curr Opin Pulm Med. 2004;10:482–8.
- 32. Shahar E, Whitney CW, Redline S, et al. Sleep-disordered breathing and cardiovascular disease: cross-sectional results of the sleep heart health study. Am J Respir Crit Care Med. 2001;163:19–25.
- Dincer HE, O'Neill W. Deleterious effects of sleep-disordered breathing on the heart and vascular system. Respiration. 2006;73:124–30.
- Mehra R, Benjamin EJ, Shahar E, et al. Association of nocturnal arrhythmias with sleep-disordered breathing: the sleep heart health study. Am J Respir Crit Care Med. 2006;173:910–6.
- Arzt M, Young T, Finn L, et al. Association of sleep-disordered breathing and the occurrence of stroke. Am J Respir Crit Care Med. 2005;172:1447–51.
- Coughlin SR, Mawdsley L, Mugarza JA, et al. Obstructive sleep apnoea is independently associated with an increased prevalence of metabolic syndrome. Eur Heart J. 2004;25:735–41.
- Seet E, Chung F. Obstructive sleep apnea: preoperative assessment. Anesthesiol Clin. 2010;28:199–215.
- Gami AS, Howard DE, Olson EJ, et al. Day-night pattern of sudden death in obstructive sleep apnea. N Engl J Med. 2005;352:1206–14.
- Fujita S, Conway W, Zorick F, Roth T. Surgical correction of anatomic abnormalities in obstructive sleep apnea syndrome: uvulopalatopharyngoplasty. Otolaryngol Head Neck Surg. 1981;89(6):923–34.

- 40. DeRowe A, Gunther E, Fibbi A, et al. Tongue-base suspension with a soft tissue to bone ancjor for obstructive sleep apnea: preliminary clinical results of a new minimally invasive technique. Otolaryngol Head Neck Surg. 2000;122:100–3.
- Riley RW, Guilleminault C, Powel NB, et al. Palatopharyngoplasty failure, cephalometric roentgenograms, and obstructive sleep apnea. Otolaryngol Head Neck Surg. 1985;93:240–4.
- Riley RW, Powel NB, Guilleminault C. Inferior sagittal osteotomy of the mandible with hyoid myotomy-suspension: a new procedure for obstructive sleep apnea. Otolaryngol Head Neck Surg. 1986;94:589–93.
- 43. Hörmann K, Baisch A. How I do it: the hyoid suspension. Laryngoscope. 2004;114:1677–9.
- 44. Lee NR, Woodson T. Genioglossus muscle advancement via a trephine osteotomy approach. Operative Techniques. Otolaryngol Head Neck Surg. 2000;11:150.
- Lee NR. Genioglossus muscle advancement techniques for obstructive sleep apnea. Oral Maxillofacial Surg Clin North Am. 2002;14:377.
- Li KK, Powel NB, Riley RW, Troell RJ, Guilleminault C. Longterm results of maxillomandibular advancement surgery. Sleep Breath. 2000;4:137–9.
- Li KK, Riley RW, Powel NB, Guilleminault C. Obstructive sleep apnea and maxillomandibular advancement: an assessment of airway changes using radiographic and nasopharyngoscopic examinations. J Oral Maxillofac Surg. 2002;67:27–33.
- Li KK, Riley RW, Powel NB, Troell RJ, Guilleminault C. Overview of Phase II surgery for obstructive sleep apnea syndrome. Ear Nose Throat J. 1999;78:851–7.
- Sharma VK, Galli W, Haber A, et al. Unexpected risks during administration of conscious sedation: previously undiagnosed obstructive sleep apnea. Ann Intern Med. 2003;139:707–8.
- Fairbanks DNF. Uvulopalatopharyngoplasty complications and avoidance strategies. Otolaryngol Head Neck Surg. 1990; 102:239–45.
- Siyam MA, Benhamou D. Difficult endotracheal intubation in patients with sleep apnea syndrome. Anesth Analg. 2002;95: 1098–102.
- Kim JA, Lee JJ. Preoperative predictors of difficult intubation in patients with obstructive sleep apnea syndrome. Can J Anesth. 2006;53:393–7.
- 53. American Society of Anesthesiologists Task Force on Management of the Difficult Airway. 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; 98:1269–77.
- Terris DJ, Clerk AA, Norbash AB, et al. Characterization of postoperative edema following laser assisted uvulopalatoplasty using MRI and polysomnography. Laryngoscope. 2009;106:124–8.
- 55. Meoli AL, Rosen CL, Kristo D, et al. Clinical practice review committee; American academy of sleep medicine. Upper airway management of the adult patient with obstructive sleep apnea in the perioperative period–avoiding complications. Sleep. 2003;26: 1060–5.
- 56. Neligan PJ, Malhotra G, Fraser M, et al. Continuous positive airway pressure via the Boussignac system immediately after extubation improves lung function in morbidly obese patients with obstructive sleep apnea undergoing laparoscopic bariatric surgery. Anesthesiology. 2009;110:878–84.
- Mickelson SA, Hakim I. Is postoperative intensive care monitoring necessary after uvulopalatopharyngoplasty? Otolaryngol Head Neck Surg. 1998;119(4):352–6.
- Kezirian EJ, Weaver EM, Yueh B, Khuri SF, Daley J, Henderson WG. Risk factors for serious complication after uvulopalatopharyngoplasty. Arch Otolaryngol Head Neck Surg. 2006;132(10): 1091–8.

- Troell RJ, Powell NB, Riley TW, et al. Comparison of postoperative pain between laser assisted uvulopalatoplasty, uvulopalatopharyngoplasty, and radiofrequency volumetric tissue reduction of the palate. Otolaryngol Head Neck Surg. 2000;122:402–9.
- Kaw R, Michota F, Jaffer A, et al. Unrecognized sleep apnea in the surgical patient: implications for the perioperative setting. Chest. 2006;129:198–205.
- Chung SA, Yuan H, Chung F. A systemic review of obstructive sleep apnea and its implications for anesthesiologists. Anesth Analg. 2008;107:1543–63.
- Bolden N, Smith CE, Auckley D, et al. Perioperative complications during use of an obstructive sleep apnea protocol following surgery and anesthesia. Anesth Analg. 2008;105:1869–70.
- Kezirian EJ, Weave EM, Yueh B, et al. Incidence of serious complications after uvulopalatopharyngoplasty. Laryngoscope. 2004;114:450–3.
- Hathaway B, Johnson JT. Safety of uvulopalatopharyngoplasty as outpatient surgery. Otolaryngol Head Neck Surg. 2006;134: 542–4.
- Seet E, Chung F. Management of sleep apnea in adults functional algorithms for the perioperative period: Continuing Professional Development. Can J Anaesth. 2010;57:849–64.
- 66. Gali B, Whalen FX, Schroeder D, et al. Identification of patients at risk for postoperative respiratory complications using a preoperative obstructive sleep apnea screening tool and postanesthesia care assessment. Anesthesiology. 2009;110:869–87.

Phonomicrosurgery and Office-Based Laryngology

10

Allan P. Reed and Peak Woo

Introduction

Due to the nature of our practices, the concept of collaboration and team work between anesthesiologists and otolaryngologists has been emphasized throughout this book, but because of the very nature of the "shared airway" during procedures such as bronchoscopy, laryngoscopy, or esophagoscopy, the importance of communication between surgeon and anesthesiologist cannot be understated and in fact is critical for patient safety and best patient outcomes.

The practice of laryngology and phonosurgery focuses heavily on the voice. For some patients, such as those with laryngeal cancer, the goal is to restore speech for communication, while in others, such as the professional singer, the goal is to enhance the voice or restore it to its original quality. For the cancer patient whose focus is communication, a hoarse or soft voice and the ability to eat without aspiration are the main goals. In phonosurgery, voice quality takes on greater importance than in most other types of operations. This chapter will outline the major topics related to operative laryngoscopy, bronchoscopy, and esophagoscopy as well as specific issues in phonomicrosurgery. A special section of this chapter is devoted to the expanding field of office laryngology and phonomicrosurgery for those involved with or looking to expand their practice into the office setting.

oxygenation, ventilation, analgesia, amnesia, and a relaxed environment free of motion, the otolaryngologist must work in a constricted space where millimeters of extra room can mean the difference between surgical success and failure. During many of these procedures, a rigid laryngoscope is placed into the airway and the patient's airway is suspended in order to provide binocular view of the larynx. This can produce great demands on the anesthesia team to provide adequate anesthesia and muscle relaxation while adequately ventilating the patient and managing hemodynamic changes. In adults and children, anesthetic techniques may need to be modified to accommodate the needs of sharing the airway between the surgical and anesthesia teams ^[1]. When the procedure is over, these often-short cases require rapid emergence from anesthesia for fast room turnover. The anesthesiologist working with an otolaryngologist should have the confidence to gauge the progress of the surgery and time the medications to correspond to the duration of the surgery. The otolaryngologist should communicate before and during the procedure as to the nature of the airway, the anticipated ease of intubation, the duration of the surgery and their needs for anesthesia care. Lastly, communication regarding the potential of airway compromise at the end of the procedure must also be discussed.

Preoperative Considerations

Overview

Micro-laryngeal surgery means that both the otolaryngologist and anesthesiologist must share the airway. While the anesthesiologist must be secure in providing adequate

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Otolaryngologist's Perspective

Suspension laryngoscopes, rigid bronchoscopy, and painful laser procedures are performed in the operating room with the patient under general anesthesia with a secured airway. These patients, however, initially present for office-based procedures (see special section "Office Based Laryngology and Phonomicrosurgery" later in this chapter), where their vocal cord lesion is first evaluated. Respecting that many of these patients are chronically ill, a thorough preoperative workup is necessary, even if the proposed procedure may take less than an hour.

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There are multiple laryngeal lesions that are managed with phonomicrosurgery; however, they can be divided into two main categories: neoplastic (benign, premalignant, and malignant) and obstructive. The benign neoplasms that affect the larynx are vocal cord nodules, polyps, cysts, and granulomas. These lesions do not have a tendency to become malignant. Premalignant conditions with significant risk of malignant conversion include papillomatosis and leukoplakia. Most malignancies of the airway and vocal cord are squamous cell in origin.

Benign Lesions of the Vocal Cord

Nodules

Vocal cord nodules occur secondary to vocal overuse and are usually at the junction of the anterior one third and posterior two thirds of the vocal folds (Fig. 10.1). This is the point of maximal contact of the vocal folds and the repetitive contact results in localized vascular congestion and eventual hyalinization of Reinke's space and thickening of the overlying epithelium ^[2]. They are analogous to a callous of the vocal cord. The treatment for early small nodules is a trial of voice therapy to cease the vocal behavior that is stimulating nodule formation. Mature and larger nodules require surgery for removal. Indications for surgical removal are longstanding nodules with secondary hoarseness. In addition to surgery, voice therapy is incorporated into the postoperative care to prevent recurrence and scar tissue formation.

Polyps

Vocal cord polyps are more often, but not always, unilateral and tend to occur in males (Fig. 10.2). They are associated with a history of vocal trauma such as intubation, intense vocal abuse, and coexisting anticoagulant use ^[2]. These lesions are secondary to capillary rupture in Reinke's space with extravasation of blood, secondary edema, and development of hyalinized stroma ^[2]. Polyps are surgically excised; however, a subset of patients with small hemorrhagic polyps may be amenable to office pulsed-dye laser treatment ^[3].

Cysts

Cysts of the vocal fold are subepithelial lesions located within the lamina propria and may be mucoid or epidermoid in origin (Fig. 10.3)^[2]. These lesions, similar to polyps, are unilateral. Surgical excision requires removal of the entire sac in order to avoid potential recurrence. These lesions do not respond to nonsurgical treatment and excision may result

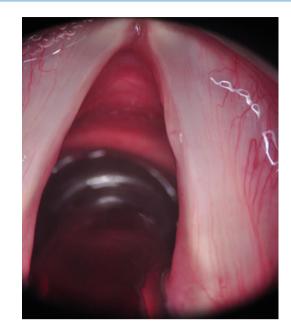


Fig. 10.1 Right vocal cord nodule

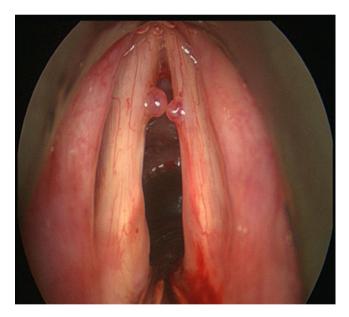


Fig. 10.2 Bilateral vocal cord polyps

in a higher risk of scarring compared to nodules or polyps due to the submucosal location.

Granulomas

Granulomas of the vocal cord occur on the vocal process and are a response to trauma, most commonly from laryngopharyngeal reflux or intubation (Fig. 10.4). These are lesions that are best managed by controlling the inciting event (e.g., reflux control) rather than surgery, due to the potential for



Fig. 10.3 Left vocal cord cyst

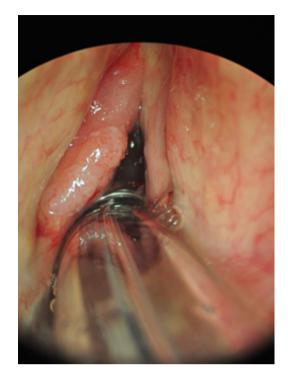


Fig. 10.5 Respiratory papillomatosis of the left vocal cord

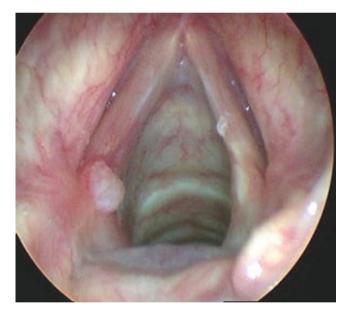


Fig. 10.4 A left vocal cord granuloma overlying the vocal process

recurrence. For this reason surgery is indicated in cases where the lesion is enlarging, compromising breathing, voice, or swallowing or suspicious for malignancy ^[2].

Precancerous Lesions of the Vocal Cord

Respiratory papillomatosis is secondary to infection with the human papilloma virus (HPV) which is the same virus associated with genital warts and cervical cancer (Fig. 10.5). There are multiple serotypes with 6 and 11 being the most

common and 16 and 18 most associated with malignant degeneration in 2% of cases. These lesions are not always unifocal and are associated with recurrence. The mainstay of surgical therapy is the pulsed-dye laser, CO_2 laser and for bulky disease, the microdebrider ^[2].

Leukoplakia is a term that represents a variety of vocal fold changes: hyperkeratosis, dysplasia, and verrucous changes (Fig. 10.6). With the development of leukoplakia, there is an 8–14% chance of malignant conversion ^[2]. These lesions are excised with phonomicrosurgical techniques for diagnostic and therapeutic reasons.

Malignant Lesions of the Vocal Cords

Squamous cell carcinoma is the most common malignancy of the larynx (Fig. 10.7). Early lesions of the glottis are managed by phonomicrosurgery or radiation therapy. Ninety percent of patients have a history of smoking. Alcohol ingestion adds to the risk of carcinoma development ^[2]. Frequently early carcinomatous lesions will have a similar appearance to leukoplakia, thus reinforcing the need to biopsy these lesions especially in the higher risk patient. On preoperative laryngoscopy, vocal cord movement must be evaluated as any alteration in movement especially paralysis would signify a more advance lesion. In general, early lesions confined to the true cords with no palpable cervical lymphadenopathy on physical exam do not require preoperative imaging. This is due to the paucity of lymphatic drainage from the glottis and therefore unlikely dissemination.

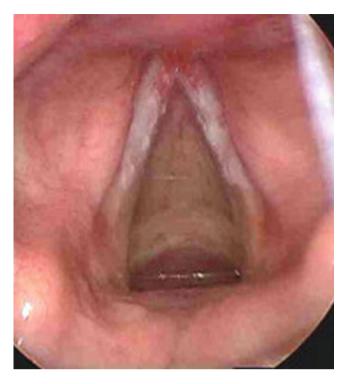


Fig. 10.6 White plaque-like lesions are noted on both vocal cords consistent with leukoplakia. This patient had normal vocal cord movement

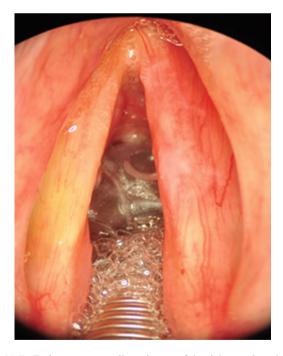


Fig. 10.7 Early squamous cell carcinoma of the right vocal cord

Anesthesiologist's Perspective

Preoperative assessment of the patient presenting for panendoscopy (i.e., laryngoscopy, bronchoscopy, and/or esophagoscopy) requires a focused evaluation of the patient with particular attention paid to the airway pathology and specifically when present, airway lesions, locations, size, and mobility.

General Evaluation

Patients presenting for phonomicrosurgery or laryngoscopic evaluation may often be elderly with a significant smoking and alcohol use history. As such, the expected comorbidities (e.g., coronary artery disease, chronic obstructive pulmonary disease) will necessarily complicate anesthetic management. The presence of a productive cough or adventitious lung sounds should be sought and chest X-ray findings used in establishing a baseline or determining worsening of the patient's condition. Since the surgical stimulus during procedures such as laryngoscopy can be profound, a thorough cardiac evaluation is important. Symptoms consistent with coronary artery disease, congestive heart failure, or arrhythmias means that patients may not tolerate even brief surgical stimuli during these procedures. Where appropriate, a thorough cardiac evaluation including stress testing and/or echocardiography, in addition to an electrocardiogram, should be secured. If the patient endorses a history of alcohol abuse, the anesthetic requirements may be high and this should be considered. Finally, electrolyte and hematologic abnormalities are common in patients presenting with malignancy, chronic disease or chronic alcohol use and thorough lab evaluation will assist in elucidating these abnormalities. We prefer a complete blood count, basic chemistry panel, and coagulation studies for almost all of these patients.

Airway Evaluation

History and physical examination are the screening tools first and foremost to rule out dangerous airway pathology. A discussion with the otolaryngologist concerning the patient's predominant pathology (i.e., location of the lesion and suspected impact on the airway) is critical. For esophageal pathologies, obstructing lesions, GI bleeding, and potential for aspiration should be explored carefully. In adult patients, the presence of inspiratory stridor suggests that the airway diameter is reduced to approximately 4 or 4.5 mm in diameter. However, the absence of stridor does not always indicate a normal size airway and may in fact indicate an extremely stenotic airway with limited air movement. Exhausted patients may also not generate enough airflow to produce stridor, even in the face of significant stenosis. Voice changes such as hoarseness are nonspecific and frequently result from small, non-obstructive lesions or potentially from mediastinal lesions. Dysphagia suggests the possibility of supraglottic obstruction. Inability to lie flat, the need to sit upright, and/or the need to change position frequently to breathe are symptoms of severe airway obstruction.

Supraglottic and glottic masses are evaluated by awake, spontaneously breathing nasopharyngoscopy. Subglottic and tracheal problems are investigated with chest X-rays, computed tomography (CT), and/or magnetic resonance imaging (MRI) and often present with inspiratory and expiratory stridor. In any case, a clear backup plan for airway management must be devised should traditional methods of securing the airway fail (Chap. 8). It is critical that anesthesiologist caring for these patients be familiar with radiographic imaging of the airway (Chap. 2), and all tracheobronchial and airway imaging should be reviewed and discussed by both otolaryngologist and anesthesiologist prior to the case. Previous anesthesia records could reveal prior problems with mask ventilation, laryngoscopy, bleeding, or extubation.

Intraoperative Considerations

Otolaryngologist's Perspective

General Issues

Phonomicrosurgical and certainly panendoscopic procedures are associated with minimal blood loss in general. If a GI bleed is present, this is of course not the case and any suspicion of this should be made clear to the anesthesiologists, so adequate intravenous access can be established and blood be made available. Most of these cases will not require placement of a Foley catheter and as such fluids should be kept to a minimum.

Positioning

Each of these procedures generally requires a 90–180° turn to allow for optimal surgical exposure and access. The anesthesiologist should be prepared for this positioning by having expandable ventilator circuits and monitoring cables long enough for the turn. Also, a plan for airway rescue should be determined as the anesthesiologist may not be able to bag mask ventilate the patient once the bed is turned.

Obtaining the best operative airway exposure has been demonstrated in many textbooks dating back to the early twentieth century and will be briefly described here. The shoulder should be down with the head held approximately 15 cm above the level of the shoulder. Support of the occiput of the head allows the head to be extended relative to the neck. Meanwhile the neck is flexed forward relative to the chest. This neck flexion, head extension position is the classical Bryce Jackson position necessary for direct laryngoscopy^[4]. This is the classic "sniffing position". A properly

executed endoscopy position will allow an adequate view in the majority of laryngeal structures. Counter pressure by pushing down on the cricoid cartilage and the trachea helps to straighten the crico-tracheal angle and view the anterior commissure of the larynx during microlaryngoscopy. This can be accomplished by the assistant or a counter pressure device or taping of the cricoid and trachea downward during micro-suspension laryngoscopy. Suspension of the larynx is directed at 45° in an anterior and inferior angle during supine laryngoscopy. This vector simulates the pull of the handle of the laryngoscope during the exposure of the larynx.

A series of photographs taken during laryngoscopy is shown simultaneously with the internal exposure of the larynx in a difficult to expose patient is shown in Fig. 10.8a–d. Once the patient is in position, one can appreciate the anterior commissure, even in this difficult to expose patient, the careful application of endoscopy exposure and counter pressure has resulted in good exposure adequate for both laser and microsurgical techniques. One can see that as the patient is placed into more neck flexion and head extension while the shoulder is held downward, the exposure improves such that the endolaryngeal structures come into view. With the final suspension apparatus in place and the application of counter pressure, even the most difficult appearing airways may be exposed for micro-laryngeal surgery.

Getting the best exposure while sharing the airway can be challenging. It may mean that both otolaryngologist and anesthesiologist need to work on mutually acceptable compromises. Clearly, an obese patient with pulmonary disease may prove difficult to ventilate through a 5.5 endotracheal tube. Patients with stenosis of the larynx or trachea who do not have a tracheostomy may be difficult to ventilate through a conventional endotracheal tube system. Particularly in patients with posterior glottic stenosis or laser surgery of the larynx for cancer involving the arytenoids cartilage or cricoid cartilage, alternative methods of ventilation have been used. These will be discussed below.

Jet Ventilation Techniques

Options for the difficult exposure in microlaryngoscopy and bronchoscopy have been described since the 1950s ^[5]. The use of jet ventilation has been in the literature for more than 40 years ^[6]. The original description of the Saunders jet ventilation device was a handheld toggle with a high pressure oxygen source. The pressure needed for ventilation was adjustable to deliver jet ventilation through a catheter that was placed either proximally or distally to the larynx. Some bronchoscopes today maintain a Luer lock jet ventilation port for open tube bronchoscopy. The problems of jet ventilation using hand devices were related to the poor reliability of delivering oxygen down a narrowed glottis. The entrainment

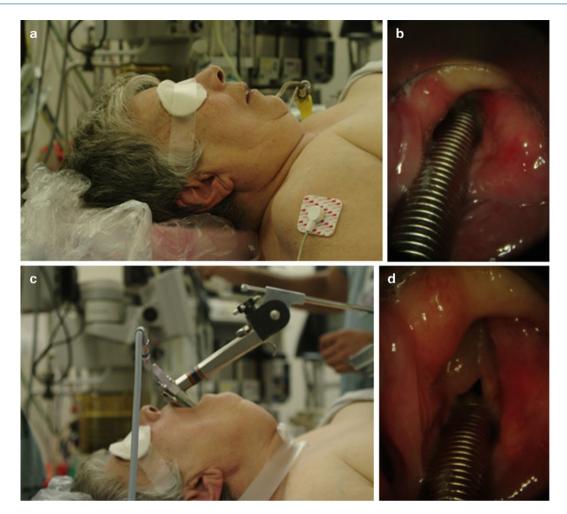


Fig. 10.8 (a) This figure shows the head in neutral position before the laryngoscope has been introduced and the patient has been placed under suspension. (b) This is the view of the larynx. Note that the exposure is inadequate for microlaryngoscopy. The initial endoscopy view is obscured by the laser tube and does not show the laryngeal mass, which

is anterior. (c) After the patient has been suspended, the neck is flexed and the head is extended. There is counter pressure being applied on the anterior neck and the tape is pushing down on the cricoid to straighten out the cervical-tracheal angle. (d) The final position shows a good endoscopy view of the larynx with adequate working room

of air down the path of the jet by the Venturi principle was compromised if there were distal obstructions or lack of alignment of the jetting catheter down the airway. Distal placement of the high pressured jet catheter had its own complications including barotraumas, pneumothorax, and pneumomediastinum^[7, 8]. For the otolaryngologist, the use of the proximal jet catheter within the laryngoscope meant great care was needed to align the catheter down the airway while working. The otolaryngologist has to assure that blood, debris, and secretions do not contaminate the airway during the surgery lest the material be blown down distally. The use of conventional hand-held jet ventilators meant movement of the tissue during micro-laryngeal surgery interfered with the fine microscopic nature of the surgery ^[9]. For these reasons, routine suspension laryngoscopy is usually preferred with a small endotracheal tube in place.

Another technique that has stood the test of time when tubeless procedures are needed has been the combination of high frequency positive pressure ventilation (HFPPV) system with the use a small catheter such as Hunsaker tube (Fig. 10.9)^[10, 11].

The HFPPV ventilator is available as a standalone unit that can deliver gases through a small catheter. The Hunsaker tube is 3 mm wide and has a self-inflating "fin" assembly at the distal end that serves to center itself down the trachea while stabilizing the tube below the level of the vocal cords during jet ventilation. This tube is placed in the subglottis so that there is less risk of displacement as compared to jet ventilation proximally ^[11]. This prevents distal barotrauma while eliminating the need for the otolaryngologist to manage the ventilation tube. The HFPPV machine allows for the ventilation of the patient at 100–500 pulses per minute at lower

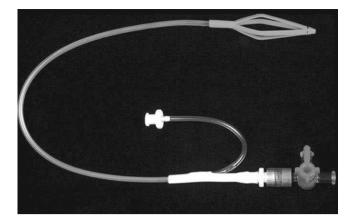


Fig. 10.9 Hunsacker tube

driving pressures than conventional jet ventilation. Entrainment of air around the jetting catheter is similar to conventional jet ventilation but the pressures necessary for ventilation are lower ^[12]. This reduces the risk of barotrauma. The higher frequency and lower driving pressure also means less noticeable patient movement during microsurgery. A typical ventilation parameter used in a healthy adult male or female is 100–120 pulses per minute, with a driving pressure of 20 pounds/inch². The I/E ratio is set at 30% so a longer period is allowed for expiration of air during ventilation. The side port of the catheter has a convenient Luer lock for end tidal CO₂ monitoring.

Despite these conveniences, the anesthesiologists and otolaryngologists must still be aware of several potential hazards during HFPPV ventilation. These include: the surgical team must monitor for progressive hyper or hypoventilation of the patients during surgery. The otolaryngologist must allow gas egress during surgery. If there is any outlet obstruction of exhalation, the lung will progressively distend ^[12]. A similar situation can occur if the ventilation pressure is too great and the lung volume is not allowed to decompress. This can lead to pneumothorax especially in patients with COPD. If the patient is not being ventilated adequately by the HFPPV system, there will be a gradual buildup of CO₂ and resultant acidosis before the onset of hypoxia. The lack of ventilation may be very hard to detect, as the motion of the chest may be barely visible and end tidal CO₂ measurements through the side port inaccurate. The chest may look either too inflated or not inflated enough. In these scenarios, it is important to have a backup method of ventilation by either rigid bronchoscopy or by traditional endotracheal intubation. A tracheostomy may be necessary in some patients. Finally, the use of the Hunsaker tube is not completely fire safe and the use of 100% oxygen through the Hunsaker tube can result in an airway fire if the tube is punctured and there is char present to support combustion. Although complications are unusual, the extra expertise needed for HFPPV means that in the majority of uncomplicated microlaryngoscopy, the author suggests a small endotracheal tube with mass ventilation^[13].

Apneic Ventilation

Appeic ventilation is a technique that is popular in children but not as popular in adults ^[14, 15]. In this technique, the otolaryngologist alternates with the anesthesiologist to share the airway [16]. After general anesthesia and endotracheal intubation by the anesthesiology team, the otolaryngology team then takes over and inserts the laryngoscope. Under direct vision the patients' endotracheal tube is removed while the patient is apneic. This gives the otolaryngologist a period of 2-5 min to work (assuming pre-oxygenation and average lung mechanics). The ETT tube is then replaced through the laryngoscope when the oxygen saturation begins to fall (usually to the low 90s). The patient is then ventilated via the ETT tube until the hypoxia and hypercarbia are corrected and at that point the ETT tube can once again be removed. The cycle is repeated until the surgery is complete. This has the advantage of a completely device-free airway and does not risk blowing blood, papilloma, tumor, or infectious material down the airway. It is, however, technically challenging and time-consuming for both the anesthesiologist and the otolaryngologist.

Managing the Difficult-to-Visualize Larynx

The anesthesiology and otolaryngology team must be knowledgeable and prepared to use rescue techniques during microlaryngoscopy when traditional operating laryngoscopes are not feasible. The following suggestions can be tried in the following escalating format. As each technique is employed, one loses some of the advantages of microlaryngoscopy. Each will be outlined below.

A smaller endotracheal tube or HPPPV with a Hunsaker tube should be used. By using a smaller endotracheal tube or a small bore Hunsaker tube, the operating laryngoscope may be inserted with an adequate view of the larynx. Figure 10.10 is an example how a wide view of the larynx can be attained while ventilating with a small bore jetting tube in a patient being managed for supraglottic stenosis. The otolaryngologist then may use the microscope with traditional binocular microscopy and bimanual instruments. The use of a smaller bore tube may mean that a laser tube cannot be used. When a jet ventilation catheter is used, the otolaryngologist must be aware of the hazards of open tube ventilation discussed in the Jet Ventilation section.

The use of a narrow bore laryngoscope such as the Hollinger laryngoscope may be used. This small bore diagnostic laryngoscope is designed for operative biopsy and not binocular viewing or surgery under the microscope. The special equipment for rescue laryngoscopy is shown in Fig. 10.11. However, the narrow waist of the laryngoscope means that in some patients with a large tongue and small mandible where large bore operating laryngoscopes are

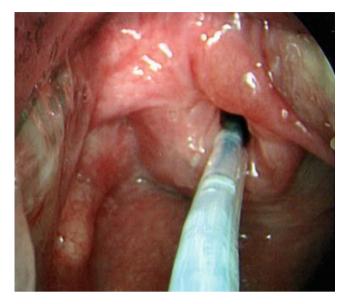


Fig. 10.10 This intraoperative endoscopy view is an example of how a wide view of the larynx can be viewed while ventilating with a small bore jetting tube in a patient being managed for supraglottic stenosis



Fig. 10.12 Photograph of a telescopic laryngoscopy being done through either the Benjamin slim-line laryngoscope or the Hollinger laryngoscope to perform anterior endoscopy and lesion removal to overcome the line of sight problem with microscopic laryngoscopy. This technique allows only one handed instrument through a monocular endoscope that may be angle from 30° through 70°



Fig. 10.11 This is the basic setup for rescue laryngoscopy where the traditional micro-laryngoscopes have failed. The instruments include the laryngeal mast airway, the angled telescope, the Hollinger diagnostic narrow laryngoscope, and angled forceps necessary for doing the procedure

impossible, the diagnostic laryngoscope could be used. The laryngoscope can be suspended using the gallows suspension apparatus and the operating microscope could be brought in. In this way, operating microscopy and bimanual microscope can still be used to complete the procedure. Because of the narrow laryngoscope, the otolaryngologist loses binocular vision. However, magnification, suspension, and bimanual manipulation of the lesion can still be used to complete the operation.

When the rigid operation laryngoscope cannot be placed to expose the lesion other techniques can be tried to overcome the need for direct line of sight when using the microscope. This is usually a problem in patients with a stiff neck

after radiation therapy or due to failure of exposure of the anterior commissure. The lesion of interest is usually anterior to the exposure possible with a rigid scope. To obtain and remove the lesion, this can be accomplished by the use of the telescope and angled instruments. The angled telescopes commonly used for rigid bronchoscopy come in 0°, 30°, and 70° angulations. They may come with integration into a biopsy or foreign body forceps. Figure 10.12 is a photograph of a telescopic laryngoscopy being done through either the Benjamin slim-line laryngoscope or the Hollinger laryngoscope to perform anterior endoscopy and lesion removal to overcome the line of sight problem with microscopic laryngoscopy. To employ this technique, the patient is first suspended while using a narrow bore laryngoscope such as the Hollinger scope. The angled telescope such as the 30° or 70° scope is used to see whether the lesion is able to be visualized. If the lesion can be visualized, an angled instrument such as a cup forceps can be brought in to biopsy or remove the lesion. By combining a small bore laryngoscope, counter pressure, an angled telescope and angled instrumentation, the otolaryngologist may be able to rescue the procedure to obtain an adequate biopsy or remove the lesion. The otolaryngologist can still get magnification through the telescope but binocular visualization and bimanual manipulation is no longer possible.

In the extreme situation where both intubation and exposure with a laryngoscope is not possible under local anesthesia, the use of a laryngeal mask airway (LMA) and the use of interventional flexible laryngoscope through the laryngeal mask may be helpful ^[17–19]. To do this, the laryngeal mask device is placed and good ventilation is verified. Through the



Fig. 10.13 This is a close-up of the biopsy cup forceps being placed through the LMA. This affords the otolaryngologist the ability to biopsy a lesion under anesthesia but does not give magnification or bimanual manipulation of tissue

working channel of the LMA, a channeled chip-tip interventional laryngoscope or a bronchoscope can be brought into the field. The endoscope is threaded through the LMA ^[20] to view the lesion for biopsy or excision. Through the working channel of the flexible laryngoscope, a biopsy forceps is used to remove or biopsy the lesion. This affords the otolaryngologist the ability to biopsy a lesion under anesthesia without movement when such an approach is done in the office. With this approach, one sacrifices the advantages of binocular vision, bimanual instrumentation and magnification but retains the advantages of general anesthesia ^[21]. Figure 10.13 is a closeup of the biopsy cup forceps being placed through the LMA.

When the above techniques do not work, the surgery must be abandoned and open techniques should be considered. These include tracheostomy and open laryngotomy.

The equipment necessary for the rescue laryngoscopy includes the narrow laryngoscope, the angled telescope and angled forceps necessary for doing the procedure. It is not a necessary setup for each case, but in cases where difficulty with exposure can be anticipated, a separate rescue laryngoscopy setup can be helpful in completion of the procedure. By using the above techniques in an escalating fashion, the rescue techniques have been successful in achieving operative laryngoscopy without the need for tracheostomy in most cases.

Medialization Laryngoplasty and Arytenoid Adduction

Medialization laryngoplasty (ML) and arytenoid adduction (AA) are procedures used to prevent aspiration and improve phonation in patients with vocal cord (VC) paralysis. ML is a simple procedure that percutaneously provides support to the affected VC. This support or bulk medializes the VC in order to achieve full glottic closure with the mobile VC. ML is a

technically simple, yet exact surgical procedure. The possibility of operative complications such as transient airway edema and hematoma in medically ill patients may make even the simplest surgical procedures inappropriate in the management of patients with vocal cord paralysis. Despite these precautions, the majority of patients with vocal fold incompetence should be able to tolerate a short surgical procedure of medialization laryngoplasty under sedation without adverse effects. In these cases, anesthesiologists should understand that rapid and catastrophic airway compromise is possible; hence, judicious use of sedation is recommended.

This surgery is easily tolerated under local anesthesia. Yet, not all patients should be considered candidates. Patients who should have the simpler procedure of injection laryngoplasty may include patients who have a bleeding disorder or compromised immune status. Patients undergoing chemotherapy have poor wound healing and are poor candidates. Patients with severe pulmonary compromise by previous pneumonectomy and those with severe COPD will need careful assessment of the effect of added laryngeal surgery and change in laryngeal resistance on their pulmonary function.

Because the surgical approaches to medialization laryngoplasty (ML) and arytenoid adduction (AA) are best performed under local anesthesia, the surgical skills necessary for the performance of AA and ML must be developed. The precise location of the small muscular process of the arytenoid cartilage makes the arytenoid adduction procedure a more technically challenging procedure than medialization laryngoplasty surgery. In some centers, medialization laryngoplasty is the only surgery considered for patients with vocal fold paralysis. Some controversy exists even now as to how much the posterior gap can be closed by the medialization laryngoplasty approach. In consideration of the surgery between ML and AA, it is important to have an understanding of the biomechanics of the different operations. By careful preoperative selection of those patients who will benefit from the ML procedure alone, the AA procedure may be avoided. Preoperative video-endoscopy and analysis of phonatory function studies can help to identify these patients who can benefit from the AA-ML combined procedure.

Anesthesia by local block is an important part of being able to do laryngoplasty with patient voice feedback. Sedation with intravenous agents such as propofol or midazolam and opioids such as fentanyl or remifentanil is usually administered by the anesthesiologist. Titration of medication for pain and anxiety management must be balanced against the need to phonate and maintain reflexes to protect the airway. Patient cooperation is necessary at certain times during the procedure. To this end, a pure opioid regimen has the benefit of analgesia with good patient cooperation.

Local anesthesia for medialization laryngoplasty is performed by injection of Lidocaine 1% with 1:100,000 Epinephrine over the thyroid cartilage. A midline injection of three centimeters by three centimeters is adequate for medialization laryngoplasty. Deeper injections to the thyroid perichondrium are performed after the superficial injection. More lateral injections along the thyroid alae are performed for patients undergoing the AA-ML procedure. To do this, the otolaryngologist places the needle at the midline. Injections are done from the midline laterally to the inferior pharyngeal constrictors. This should adequately anesthetize the pharynx and larynx for the ML procedure. Care should be exercised in not injecting local anesthesia into the cricothyroid muscle and into the pyriform sinus area. An adequate local anesthetic should allow the otolaryngologist to manipulate the thyroid cartilage at will. Retraction and rotation of the thyroid cartilage without discomfort should be achieved before and during surgery. The complications of laryngoplasty include airway obstruction, hematoma, and dysphagia. To prevent airway complications during the surgery, the depth of sedation should be monitored carefully and injection of local anesthesia should be limited to avoid recurrent laryngeal nerve block and superior laryngeal nerve block [22].

Local anesthesia is administered in a similar fashion for those patients undergoing AA-ML. The cervical skin block is larger to accommodate a larger incision, the limits of the skin block are from the midline to the posterior border of the thyroid cartilage of the affected side. The skin between the contralateral 1/2 of the thyroid cartilage, the lower border of the hyoid bone, and the superior border of the cricoid cartilage are the other three landmarks that need to be anesthetized prior to surgery. This will insure that retraction will not result in unwanted movement due to patient discomfort. The main difference is the need to inject Lidocaine liberally along the posterior lamina of the thyroid cartilage. The skin and cartilage block necessary for the arytenoid adduction procedure is done by a more lateral approach. The larynx is first rotated toward the midline by pushing inward on the opposite thyroid alae. This will accentuate the posterior border of the thyroid alae on the side that is being treated by surgery. The otolaryngologist uses the injection to come from lateral to medial direction to stab down on the posterior border of the thyroid cartilage. The posterior border is verified by palpating the hard posterior border of the thyroid cartilage. The needle is passed from a lateral to medial direction to stab down on the posterior border of the thyroid cartilage. Care is taken not to inject into the carotid sheath and not to inject posterior to the thyroid lamina. By stabbing down directly on to thyroid cartilage, the entire posterior constrictor is anesthetized. Anesthesia along this area will facilitate the elevation of the posterior musculo-perichondrial flap and allow for adequate retraction to expose the arytenoid cartilage.

The Professional Singer

A singer who must undergo anesthesia is beset by anxiety by the use of instrumentation, specifically by endotracheal intubation ^[8]. Stories abound about singers who could not sing after either brief or prolonged intubation. Intubation injuries may be classified as anatomical disruptions or due to neuro-motor injury. Under anatomical disruption, vocal fold tear, intubation, arytenoid dislocation, and vocal fold edema are the most common. Prolonged intubation may also cause laryngeal stenosis, intubation groove or crico-arytenoid arthrodesis ^[23, 24] and granulomata. Under the possible injuries to the neuro-motor system, the most likely is temporary vocal fold paralysis or paresis due to recurrent laryngeal nerve compression from a high riding cuff. These injuries are uncommon given the numbers of procedures done under anesthesia.

Anesthesiologist's Perspective

General Concepts

Close communication with the otolaryngologist is crucial to formulating a successful anesthetic plan. For the most part, these procedures will require general anesthesia with good access to the airway and aerodigestive structures provided by a small endotracheal tube and adequate muscle relaxation or a plane of anesthesia deep enough to allow for good surgical access. As discussed previously, close maintenance of hemodynamics is crucial given the stimulating nature of these procedures. As such, adequate anesthetic depth and agents that effectively blunt sympathetic nervous system stimulation (e.g., remifentanil and/or beta-blockers) are commonly employed. As most of these procedures are of short duration, judicious use of paralytics and short-acting anesthetic agents are of great utility. For very short procedures the use of neuromuscular blockade is often omitted and larger doses of remifentanil and propofol are employed to achieve excellent laryngoscopic conditions. It is important if this option is selected to discuss this with the otolaryngologist who may expect a completely flaccid patient. If the otolaryngologist is aware of the advantages of avoiding neuromuscular blockade, they will be more likely to work with the planned anesthetic. Obviously if the procedure cannot be accomplished this way, neuromuscular blockade must be employed. Laser surgery portends its own special risks to patients and operating room personnel. Laserrelated issues are discussed later in this chapter.

Anesthetic Induction and Airway Management

Induction of anesthesia is a period of high risk. When there is any question as to adequacy of ventilation or visualization after induction of general anesthesia, an awake fiberoptic intubation should be performed. Mobile supraglottic lesions can obstruct gas flow through the larynx during positive pressure ventilation and obscure the glottis during laryngoscopy. Laryngeal lesions such as large vocal cord polyps and papillomas have the potential to create partial airway obstruction after induction of general anesthesia. They rarely result in total airway obstruction but can make ventilation technically difficult or impossible.

Contrary to classic teaching, obstruction is frequently worse in anesthetized spontaneously breathing patients than in anesthetized patients receiving positive pressure ventilation by facemask. General anesthesia reduces tone in the pharyngeal dilator muscles, allowing soft tissues to collapse into the airway. Positive pressure ventilation tends to stent the airway open more effectively in these cases. If spontaneous ventilation is to be maintained, assistance of ventilation with positive pressure, an oral or nasopharyngeal airway and avoidance of excessive intravenous respiratory depressants is important. Spontaneous ventilation is useful to evaluate airway dynamics. This is particularly helpful in diagnosing tracheomalacia. In this case, inhalation induction is performed with sevoflurane and 100% oxygen. Once a sufficient depth of anesthesia is achieved, laryngoscopy can proceed.

Subglottic lesions do not inhibit laryngeal visualization, but can prevent advancing a tracheal tube beyond the larynx and into the trachea. If subglottic lesions are present, several small tubes should be available. A means to provide jet ventilation should be available if necessary. Space inside the airway is limited, often to a critical extent. Small diameter tracheal tubes are required to allow for optimal surgical visualization and manipulation. Five and one-half millimeter internal diameter tracheal tubes generally satisfy this requirement and allow for adequate gas exchange ^[1]. Controversy exists over the use of such small tracheal tubes in large adults. Adequate minute ventilation delivered through small tracheal tubes is accompanied by high readings on inspiratory pressure monitors. This is generally of little clinical importance because the small tube acts as a resistor across which there is a substantial pressure drop. Actual intra-tracheal pressures approximate those seen with larger tracheal tubes. Consequently, the risk of barotrauma is no greater using small tubes than large ones despite high measured peak inspiratory pressures on the anesthesia machine. Still, increasing time spent in inspiration (i.e., adjusting the I:E ratio from 1:2 to 1:1) will decrease pressures required to achieve adequate tidal volumes.

For non-obstructing airway lesions, standard techniques of induction and maintenance, as well as closed ventilatory techniques work well. It is common practice to administer topical local anesthetics between the vocal cords during laryngoscopy. To reduce the incidence of postoperative cough and laryngeal spasm, topical anesthesia is used routinely by instillation using the laryngo-tracheal anesthesia cannula ^[25]. Topical local anesthetics contribute to smooth, cough-free emergence, protection from laryngospasm after extubation, and provide postoperative analgesia in the surgical field. Also, if muscle paralysis is not used, anesthetized vocal cords help to assure a lack of vocal cord movement. Commonly, 4% lidocaine is used.

Anesthetic Maintenance

Regarding vocal cord immobility, muscle paralysis need not be obtained but is preferred by many. For most cases of relatively short duration, reduced doses of commonly available intermediate acting non-depolarizing neuromuscular blockers are appropriate (i.e., avoidance of "induction" doses of muscle relaxants). Neuromuscular monitoring (especially of the facial nerve which more closely approximates airway recovery) is mandatory as is full reversal of the blockade. A dangerous scenario is the patient who is recovering from laryngeal surgery who is extubated with residual paralysis, increasing the likelihood of airway obstruction and emergent re-intubation for hypoventilation and hypoxia.

An alternative strategy is to use a relatively deep plane of anesthesia that employs either total intravenous anesthesia with high doses of remifentanil (e.g., >0.2 mcg/kg/min) or use of remifentanil in addition to volatile anesthetics that exert slight neuromuscular blocking effects as well. These relatively high doses of remifentanil ensure a quiet surgical field that is "slack" and makes vocal cord movement highly unlikely. The added benefit is that muscular strength is guaranteed and extremely short procedures will not lead to patients having residual paralysis during emergence.

Emergence

Emergence from anesthesia is frequently associated with coughing and straining. These events are particularly deleterious after upper airway surgery. Forceful vocal cord adduction with or without the tracheal tube in place, extends surgical tissue damage, resulting in impaired healing. The result could be vocal cord scarring with adverse voice changes. For this reason, laryngeal anesthesia during airway manipulation and/or emergence from anesthesia with an adequate plasma levels of opioid (e.g., with a continuous remifentanil infusion) are crucial. During emergence, it is common practice to continue a low dose remifentanil infusion (0.05 mcg/kg/min) at the author's institution to allow for a smooth emergence free of coughing or "bucking." Indeed, smooth emergence is needed to avoid any additional trauma of the airway. While many otolaryngologists request a "deep extubation," the risk of aspiration of blood and debris must be considered and high dose opioid techniques often obviate the need for such techniques.

Ventilatory Modes and Open Techniques

A word regarding ventilator modes is warranted given the issues inherent to these surgical procedures. Ventilatory modes for modern laryngology practice have become highly sophisticated. They are divided into two categories—closed systems and open systems. Closed systems are familiar to all anesthesiologists. This approach protects best against aspiration, allows for positive pressure ventilation, and minimizes operating room pollution from anesthetic gases. Closed systems do, however, limit surgical visibility, interfere with surgical manipulations, risk tracheal tube related laryngeal damage ^[26, 27], and predispose to fire during laser applications.

Open systems maximize laryngeal visualization, reduce risk of tracheal tube trauma, and provide the best laser safety. Open techniques of ventilation can include spontaneous ventilation, apneic oxygenation, and jet ventilation. Unfortunately, they require specialized knowledge, training, and equipment. They also fail to protect lower airways against aspiration. The selection of the use of an open system is also determined by the lesion's location, size, mobility, and vascularity. Utilization of an open or closed system may be changed if the intraoperative requirements change. If the lesion is obscured by the endotracheal tube, the closed system may need to be changed to an open system intraoperatively. An open system utilizing jet ventilation on a lesion originally thought to be avascular may be altered to a closed system to protect the airway against aspirating blood, if hemorrhage occurs.

During spontaneous ventilation following a sevoflurane induction, topical lidocaine is preferred by many to reduce sensory input and limit the anesthetic depth required by laryngoscopy. If spontaneous ventilation is chosen, anesthesia can be maintained with intravenous agents or anesthetic gases via nasopharyngeal airway or via laryngoscope/bronchoscope side port while surgery proceeds. Disadvantages of this method include frequent laryngospasm, vocal cord movement, coughing, apnea, blood pressure fluctuations, lack of airway protection, inaccurate monitoring of ventilation via end tidal capnography and operating room pollution with anesthetic gases. For these reasons, intravenous agents may be preferable for maintenance of anesthesia. If titrated appropriately, anesthesia maintained with remifentanil (0.05–0.1 mcg/kg/min) and propofol (50–125 mcg/kg/min) can allow for safe spontaneous ventilation and adequate depth of anesthesia.

Apneic oxygenation may be employed for the removal of foreign bodies, non-obstructing glottic lesions, or nonobstructing subglottic lesions. Oxygenation can be provided with supraglottic devices such as an anesthesia facemask or subglottic devices such as tracheal tubes. This technique works best in patients with fairly normal functional residual capacity (FRC) and pulmonary mechanics (i.e., at low risk for rapid desaturation). Prior to surgical manipulations during apneic periods, the FRC is filled with 100% oxygen (preoxygenation), established by an end tidal oxygen value greater than 90%. Apneic durations are predicated upon oxygen saturations, which are measured by pulse oximetry. Maintenance of anesthesia is provided with intravenous agents and/or anesthetic gases. Time for surgical manipulation is limited by intervening hypoxemia. If the patient's hemodynamics can tolerate it, placing the patient head up in reverse trendelenburg will increase FRC and apneic times before desaturation. In general, as the saturation approaches 90%, resumption of oxygenation by either facemask or endotracheal tube needs to be resumed. This intervening hypoxemia should be agreed upon and communicated to surgical staff loudly and clearly ahead of time.

Jet ventilation usually involves administration of 100% oxygen under approximately 20 pounds/inch² (psi) pressure, via a catheter or blunt needle. Anesthesia maintained with remifentanil 0.05-0.5 mcg/kg/min and propofol 50-150 mcg/ kg/min is used widely. Supraglottic (proximal) jet ventilation entails attaching the jetting needle to a suspension laryngoscope. Oxygen is delivered into the airway and aimed at the larynx. Advantages of supraglottic jet ventilation include unobstructed surgical view and limited risk of laser fires. Potential problems associated with supraglottic jet ventilation include malalignment of jet stream; gastric insufflation; blood, smoke, and infectious debris could be blown into the airway; vocal cord movement; and barotrauma. Subglottic jet ventilation involves placing a 2-3 mm diameter-jetting catheter into the trachea. Oxygen is delivered directly to the trachea. Advantages of subglottic jet ventilation include vocal cord immobility and ample time to establish rigid (suspension) laryngoscopy. Disadvantages of subglottic jet ventilation include slight obstruction to surgical field and barotrauma. Both techniques provide good oxygenation and adequate ventilation. Sanders' manual ventilator valves are applicable to normal respiratory rates. High frequency jet ventilators are set to deliver approximately 110 breaths/minute for this purpose. High frequency jet ventilation provides for continuous gas flow out of the airway, which tends to carry away small amounts of blood and debris as well as reducing peak and mean airway pressures, which could minimize ventilator-related hypotension. Ventilator rate, percent inspiratory time, and driving pressure affect PaCO₂. CPAP and FIO₂ contribute to oxygenation.

Jet ventilation requires total intravenous anesthesia techniques because inhalation anesthesia vaporizers do not adapt to jet ventilator systems. Jet ventilation requires airway patency for egress of gas. If unobstructed egress of gas is not secured by proper patient positioning or clinician maneuvers (e.g., chin lift; jaw thrust), pneumothorax can occur. This adaptation of high frequency jet ventilation also requires an open system, so that room air can augment tidal volumes based on the Bernoulli effect. Adequacy of oxygenation and ventilation are monitored with pulse oximetry and capnography, respectively. Because this is an open system, quantitative analysis of expired carbon dioxide (ETCO_2) is highly unreliable. For procedures of limited duration, qualitative assessment of ETCO_2 is available and suffices to demonstrate proper placement of ventilation catheters in the trachea. Observation of chest wall fluctuations and auscultation of breath sounds serve as further evidence of correct catheter placement. It is recommended, however, to obtain arterial blood gases for ventilation adequacy for procedures requiring prolonged jet ventilation.

Special Considerations

Laser Surgery and Fire Safety

Before beginning any procedure all personnel especially the anesthesiology team must be aware that a laser maybe deployed. Laser safety begins before the case starts and all equipment must be checked and proper function confirmed. Calibration of laser spot size, alignment of the laser, and calibration of laser power density so as to deliver the expected laser energy should all be done before the patient is in the room. Laser eye protection, wet towels to protect the face, and a syringe with water for irrigation should all be prepared.

Laser fire safety is a topic that needs to be addressed by both the otolaryngologist and the anesthesiologist before laser surgery of the upper airway ^[28]. Cooperation and communication between the surgical team and the anesthesia team is mandatory to ensure patient safety when lasers are being used in the upper airway ^[29]. Laser fires can occur with the CO₂ laser and the Nd–YAG lasers since both depend on their laser tissue interactions by thermal means. Laser fire safety should be part of the routine practice in every case.

High energy lasers provide numerous surgical advantages. Their special characteristics require alterations in anesthetic management. Patient-related risks revolve around airway fire. Laser airway surgery is associated with a 0.5-1.5% incidence of fire. These fires typically start at the tracheal tube, tracheal tube cuff, or cottonoids in the airway. Fires can result from direct impact, reflected light, heated dry cloth, or hot dry tissues. Flames on the exterior of tracheal tubes can produce local mucosal damage. If the tube is pierced by the laser or flame, two important problems can arise. First, polyvinyl chloride is degraded into toxic constituents. Second, inspired gases with high oxygen concentrations are easily ignited. Flames are directed down the airway into trachea and lungs, as if a blowtorch was placed in the airway leading to expected thermal and chemical injury. Puncture of the tracheal tube cuff can allow inspired gases to surround laser fields and predispose to fire, in the newly created, local, oxygen- enriched

atmosphere. Laser-resistant tracheal tubes reduce the chances of converting the tracheal tube into a hospitable environment for a fire. Many laser tubes are equipped with two inflatable cuffs. If the upper cuff is punctured by the laser and deflated, the lower cuff should continue to seal the trachea, allowing for positive pressure ventilation and preventing inspired gases from reaching the laser field.

Specially designed tubes are laser resistant. Much information exists on the hazards of use of flammable endotracheal tubes in the environment of oxygen ^[28]. They are less likely than ordinary tracheal tubes to ignite if exposed to laser energy. Laser tubes also have larger external diameters than comparable traditional tracheal tubes of the same internal diameter. They tend to be stiffer and bulkier than polyvinyl chloride tubes, so the risk of mucosal abrasion, vocal cord hematoma, and other problems is increased. Furthermore, because the inside is corrugated, they require a higher pressure and have a higher resistance to mass ventilation. Other tubes such as those made with a foam coating must be moistened well in order to be laser retardant. Despite the tube being laser retardant, the cuff still needs to be protected during the procedure by wet pads placed over the cuff. If jet ventilation is being used through a catheter such as the Saunders tube or the Hunsaker tube, the tube may melt in the heat and by laser impact. Any carbon debris in the field may support combustion in the presence of a high oxygen environment. Metal tubes can reflect laser light to other places, resulting in tissue damage. Laser tracheal tube cuffs are inflated with water. In that way, if the laser perforates a cuff, water will flow through the hole and cool the plastic to extinguish a fire, if present. Inflated tracheal tube cuffs do not necessarily create perfect air-tight seals with the trachea. In such cases, inspired gases delivered under positive pressure leak into the upper airway where they can accumulate. This is the same location in which the laser is used. Therefore, the lowest FIO, that provides adequate oxygenation is employed as the inspiratory gas mixture for all surgeries involving lasers. Otherwise, lasers would be used in an oxygen-enriched atmosphere, predisposing to fire. For much the same reason, air is substituted for nitrous oxide. Nitrous oxide supports combustion. Helium has been used to dilute oxygen. It does not support combustion, flows through small tracheal tubes better than other gases, and minimally delays ignition if the tracheal tube is punctured. Potent inhalation agents may undergo pyrolysis to toxic compounds, but this does not seem to be a significant problem in clinical practice. In any case, an FIO, of less than 30% should be maintained. If the patient does not tolerate low FIO₂, the otolaryngologist should be informed, laser use stopped, and a high FIO₂ reinstituted.

In our practice we employ two types of laser-resistant endotracheal tubes, the Mallinckrodt Laser-Flex[®] endotracheal tube and Medtronic LASER-SHIELD II[®]. The LASER-SHIELD II is a flexible endotracheal tube with one cuff (filled with powered methylene blue) and a laser-resistant wrap of aluminum over a silicone shaft (Fig. 10.14).

For most cases the less expensive Mallinckrodt tube is used. This stiff stainless steel metal tube has two cuffs, lacks demarcations and should never be placed nasally. Due to stiffness and the fact that this tube keeps its shape once bent makes it challenging to place via a fiberoptic intubation. For nasal cases or those requiring fiberoptics we use the softer, more pliable Medtronic tube. Both tubes lack length demarcations and we advise marking the tube length with tape or a marker prior to use at or near the intended depth for a proper endotracheal position (Fig. 10.15a-c).

Ultimately it is advised that the cuffs of these tubes be inflated with saline. Due to the increased viscosity injecting saline and the subsequent removal of saline takes more time to assure that an adequate amount is injected before the procedure starts and that all of the saline is removed before extubation. After intubation we recommend inflating the cuff

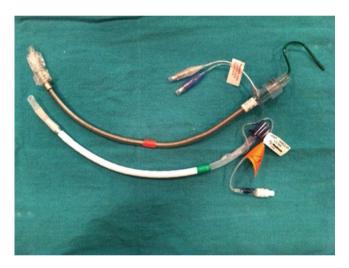


Fig. 10.14 Mallinckrodt Laser-Flex[®] endotracheal tube (*top*) and Medtronic LASER-SHIELD II[®] tube (*bottom*)

with air, confirming proper position via auscultation and visualization. This will help avoid the need for the removal of saline should the tube be malpositioned during the initial intubation. For the double-cuffed tubes one can then inflate the second cuff with saline, and then exchange the air for saline in the original cuff. Before emergence we recommend that the saline be removed from both cuffs, leaving air inflated into one of them. Extubation can then be expeditious without the potential of inadequate cuff deflation.

Lasers also place operating room personnel at risk for injury. Misdirected laser beams have ignited surgical drapes producing fires and they have burned doctors, nurses and patients externally. Viral particles have been identified in smoke plumes, which are easily inhaled and could be infectious and/or carcinogenic. Smoke evacuators at the surgical site are recommended to reduce this problem. Typical surgical masks do not filter viral particles, but specially designed "laser masks" are intended to do so. Laser light can be reflected off smooth metal surfaces into surrounding personnel's eyes. For this reason, eye protectors should provide wraparound coverage. Laser surgery exposes everyone in the operating room to burns from errant beams and this includes the patient. To protect against skin burns, the patient's face is covered with wet towels. The patient's eyes should be protected by taping and covering them with wet gauze or a metal shield. To protect people outside the operating room, windows should be covered when using lasers other than CO₂ types, and the room clearly labeled as involving lasers when in use.

Despite attempts to minimize the risk of laser airway fires, they do occur. Otolaryngologists will most often recognize the problem first, but the anesthesiologist must be prepared to recognize, mitigate, and treat the patient during this catastrophic event. All members of the surgical anesthetic team should be versed in what to do in the event of an airway fire. Upon making the diagnosis of airway fire, the burning element (usually the tracheal tube or cottonoid) should be removed once the oxygen source is eliminated. Oxygen



Fig. 10.15 Sequence for properly demarcating the depth of a laser tube in a female patient: (a) the unmarked tube, (b) the use of a standard endotracheal tube to demarcate 22 cm depth, and (c) the use of tape for demarcation

administration is interrupted, ideally first so as not to remove a flaming tube from a patient's airway. If tissues continue to burn, water should be poured down the airway to extinguish the fire. Oxygenation can be resumed by facemask and the patient should be reintubated as soon as possible. Examination of the airway is then performed. Prophylactic intubation against future airway edema and resulting obstruction is probably prudent but may be difficult. If pulmonary burns are anticipated, intubation is also recommended. When the original intubation was difficult and the fire was small and readily extinguished, extubation may not be the best choice. This is a judgment to be made for each individual case. Tracheostomy may be necessary. The patient should be intubated and a more orderly bronchoscopy and laryngoscopy should be carried out. This second examination is done to assess the extent of damage. If there is evidence of inhalation or smoke damage, the patient should be lavaged of all smoke and soot as well as treated as an inhalation injury by high-dose steroids and positive pressure mechanical ventilation.

The Professional Singer

The professional singer who is to undergo anesthesia via intubation is advised to inform the anesthesiologist of his or her professional status so that extra precautions for gentle intubation and airway manipulation are taken. Topical anesthesia followed by direct visualization of the larynx prior to insertion of the endotracheal tube is optimal. Blind intubation and even intubation over a stiff guide wire is to be avoided due to the possibility of excessive force needed to push the tube through the glottis. The path of the endotracheal tube should be placed through the glottis by using the posterior glottis so as to avoid damage to the membranous larynx. The patient must be fully relaxed and not have an adducted larynx during intubation. When the anesthesiologist is looking for the glottic opening prior to insertion of the tube, it is best to look for the glottis from a proximal to distal direction. One should avoid using a Miller blade that is inserted distal and then pulled back while waiting for the larynx to fall into view. This avoids the possibility of the blade of the laryngoscope injuring the arytenoid and cricoid cartilage. During the procedure, it is important the patient does not have excessive torque on the tube and manipulation of the tube against an unvielding patient. At the time of extubation, the patient should be asked to inhale while the tube is withdrawn. The cuff should always be completely deflated and the patient should not cough during the extubation.

If reasonable, the use of supraglottic airways like the laryngeal mask anesthesia or a combitube(Chap. 8) may be alternatives to laryngeal intubation since both avoid the risk of direct injury by the endotracheal tube being placed between the vocal folds. Selection of endotracheal tubes should not only include the best tube size for ventilation but also the optimum size to avoid laryngeal injury. A 5.0 tube for female and 5.5 tube is recommended for males undergoing short procedures. For longer procedures, a 6.0 tube for females is usually adequate while a 6.5 tube is usually satisfactory for males.

Postoperative Considerations

Otolaryngologist's Perspective

If there is postoperative voice disturbance, swallow disturbance, or airway difficulties, fiberoptic laryngoscopy and a consultation with a laryngologist should be considered early. This is to avoid potentially reversible treatable conditions from becoming permanent. Laryngeal edema due to endotracheal intubation may be treated by short-term systemic steroid administration ^[30].

Intubation Granuloma

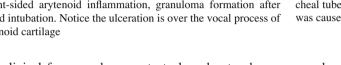
The group of disorders is characterized by vocal process granulomata of the larynx. By histological definition, these lesions are not granulomata at all but are more appropriately termed granulation tissue ^[31]. The lesions are red to tan colored, round masses accumulating over the vocal process of the arytenoid cartilage (Fig. 10.16).

The histology shows acute and chronic inflammation with capillary proliferation. Variable epithelial healing and hyperplasia and metaplasia may be present. Unlike granuloma formation, giant cells are not present.

The common pathophysiology for the beginning of a granuloma in the larynx is an inciting event that promoted mechanical trauma to the posterior larynx [32, 33]. The three most common risk factors are phono-trauma, intubation, and reflux laryngitis. Other unusual causes include osteosclerosis of the arytenoid cartilage, arytenoid mal-rotation after vocal fold paralysis. The arytenoid cartilage, when forcefully adducted as in cough and throat clearing or speaking can be abraded of its mucosa and once abraded, will ulcerate and attempt healing as a secondary healing event. In the event of recurrent trauma, repeated attempts at healing will promote granulation tissue. If successful healing occurs, the site of healing may still look abnormal. It is the process of ulceration to granulation tissue to healing at the vocal process area that may incite signs and symptoms that brings the patient to the otolaryngologists. In all vocal process granuloma, the key event is mucosal injury followed by repetitive trauma, lack of healing followed by secondary healing with granulation tissue formation. It is the clinicians job to differentiate between the



Fig. 10.16 This is the clinical picture during laryngoscopy in a patient with right-sided arytenoid inflammation, granuloma formation after prolonged intubation. Notice the ulceration is over the vocal process of the arytenoid cartilage



various clinical forms such as contact ulcer due to phonotrauma vs. contact granuloma due to acid reflux laryngitis vs. intubation granuloma. The clinical presentation, pattern of involvement on endoscopy, and the response to treatment will define the primary pattern. When the causative pattern is not recognized, the patient may suffer by prolonged treatment, repetitive excision biopsies, and failure to heal. Treatment by surgery or office treatment may be necessary ^[34].

Intubation granuloma is different than contact granuloma due to the specific inciting etiology. Figure 10.16 is the clinical picture during laryngoscopy in one of these patients; reflux or prolonged intubation is usually the inciting injury. The ulceration may then be made worse by acid reflux laryngitis. These patients present with aphonia and dysphonia and less often with throat pain and irritation. If the intubation is associated with pharyngeal tear, there may be pharyngeal findings of hematomata and laceration. Symptoms of dysphagia may also be present. The intubation granulomata may be large and partially obstruct the airway. Despite this, most patients will not complain of pain. This is a distinct pattern from patients with throat pain secondary to voice abuse disorders. The endoscopic finding of patients with intubation granulomata is also different, often appearing undulated and unilateral. Further, these lesions are found at the vocal process and usually associated with variable amounts of reflux laryngitis. These patients are best treated by anti-reflux medication and inhaled or systemic steroids.

Vocal Fold Tears

Vocal fold tear from intubation is uncommon but may be a cause of persistent dysphonia after intubation. Figure 10.17 shows a vocal fold tear caused by the endotracheal tube on the left vocal fold. This resulted during intubation. Notice the linear laceration along the superior portion of the

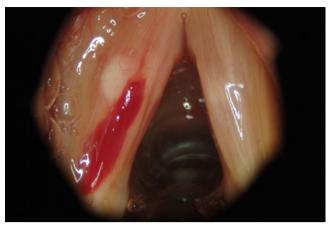


Fig. 10.17 This shows a left vocal fold tear caused by the endotracheal tube on the vocal fold. This linear tear on the superior vocal fold was caused during intubation

membranous vocal fold. Fortunately the injury is on the same side as the offending cyst.

The mechanism of vocal fold tear is the forceful push of the endotracheal tube against the membranous portion of the vocal fold. If the endotracheal tube is not directly inserted into the lumen of the airway, the endotracheal tube may catch the vocal ligament and a tear of the vocal fold cover. If the lip of the endotracheal tube is inserted into the ventricle, this will cause the endotracheal tube to push against the vocal ligament resulting in a mucosal tear. The tear of the vocal fold cover usually does not include the vocal ligament. But a tear usually creates a mucosal flap on the superior surface of the vocal fold. If this mucosal flap is non-displaced it can heal without much phonation difficulty. However, if there is significant mucosal flap displacement, it will result in the appearance of a mass or a vocal fold polyp. Dysphonia is the primary complaint in patients with vocal fold tear as the mucosal flap often stimulates a fusiform polyp. A mucosal flap tear can be differentiated from a vocal fold polyp by stroboscopy. A mucosal flap tear has the appearance of mucosa with variable amounts of inflammation surrounding the site of injury. If the mucosal tear is large, the vocal fold ligament will heal by secondary intention and scar with fibrosis will result.

Surgical treatment is used to create a straight vocal fold. Scar may be softened by steroid injection and or mucosal flap elevation. Rarely, augmentation laryngoplasty may be considered.

Arytenoid Dislocation

A particularly challenging situation for management is in the patient with suspected arytenoid dislocation. It is difficult to assess arytenoid dislocation after intubation. The clinical signs and symptoms may mimic vocal fold paralysis. There are two major types of arytenoid dislocations. The anterior type of dislocation results in airway obstruction as the posterior suspensory ligament of the crico-arytenoid joint is torn, allowing the arytenoids to fall forward and partially obstruct the airway. The lateral dislocation results from tear to the anterior suspensory ligament of the crico-arytenoid joint resulting in lateral displacement. This results in aphonia and aspiration. Clinical examination usually shows glottic incompetence with immobile vocal fold. The patient may or may not have airway distress. In these patients, a high index of suspicion, CT scan of the larynx with 2 mm cuts through the area of interest will give the highest yield to establish the diagnosis. In the management of arytenoid dislocations, early detection, stabilization, and reduction will give better outcome than delayed management.

Vocal Fold Paralysis

Intubation of the larynx with the endotracheal tube cuff placed high in the trachea may cause a compression and result in recurrent laryngeal nerve paresis or paralysis. The patient will have breathy dysphonia with occasional diplophonia. The examination will show immobile vocal fold with occasional partially mobile vocal fold. If the search for arytenoid dislocation or laryngeal stenosis is negative, the presumptive diagnosis of vocal fold paresis or paralysis may be made. Confirmation of the findings may be made by laryngeal electromyography performed 6 weeks to 3 months after injury.

The differential diagnosis of the patient presenting with vocal fold immobility is complex. Some possible diagnoses include: vocal cord paralysis, vocal fold paresis, arytenoid fixation, arytenoid dislocation, and subglottic or glottic stenosis. A carefully done endoscopic examination using constant and using a stroboscopy light source can often separate between these entities without the need for operative laryngoscopy. A carefully done endoscopic examination in the office of laryngeal function serves as the cornerstone to triage patients who may need to undergo operative intervention versus office rehabilitation.

Pseudo-paralysis of the larynx due to nasogastric tube injury is an interesting iatrogenic injury of the larynx from indwelling nasogastric tubes ^[35]. Nasogastric tubes, especially large ones, can cause injury to the posterior cricoid plate and result in pseudo-paralysis. Local mucosal ulceration and injury of the pyriform sinus occurs when the patient repeatedly swallows against the nasogastric tube. Mucosal ulceration of the pyriform sinus and the post-cricoid plate occurs as the larynx is repeatedly rubbed against the nasogastric tube. With ulceration and edema, the posterior cricoarytenoid muscle becomes dysfunctional. Endoscopic examination of the larynx will show a lack of abduction on the side of the NG tube. The patient will complain bitterly of pain on swallowing. There is often arytenoid edema and post-cricoid edema with pooling on the side of the nasogastric tube. This condition appears more commonly in patients who have mucositis after chemotherapy and in those with poorly controlled reflux disease.

Paradoxical vocal fold motion disorder and laryngeal spasm may be a temporary feature after intubation ^[25]. This is a feature of the irritable larynx syndrome that may include: cough, laryngeal spasm, and paradoxical adduction during inspiration. The diagnosis is made by fiberoptic laryngos-copy that shows adduction during inspiration with paradoxical closure seen on flexible laryngoscopy. They can be quite frightening as laryngeal spasm may result in sustained dyspnea with paradoxical closure of the larynx during inspiration. Treatment is by local and systemic administration of Lidocaine and by oxygen support ^[36].

Anesthesiologist's Perspective

Upper airway surgery is associated with increased risk of postoperative airway resistance ^[4]. After extubation, blood, secretions, or debris as well as pain in the distribution of the superior laryngeal nerve can precipitate laryngospasm. It can produce partial or total airway obstruction. Strategies for preventing laryngospasm include topical local anesthesia, intravenous lidocaine, and adequate analgesia. Treatment revolves around suctioning the airway, 100% oxygen by face mask, positive pressure to splint the airway open, and/or a small dose of succinylcholine (10–20 mg). Alternatively, induction agents such as propofol may help "break" the laryngospasm. Other causes of post-extubation stridor include laryngomalacia, tracheomalacia, vocal cord paralysis, airway edema, hematoma, soft tissue obstruction, and retained foreign body (cottonoid, throat pack).

One of the more dramatic signs of upper airway obstruction is stridor. Stridor is a predominantly high pitched, inspiratory noise emanating from the airway. It can result from partial obstruction or near complete obstruction. Treatment revolves around 100% oxygen by face mask, sitting position, and steroids. Some patients may benefit from nebulized racemic epinephrine. For selected patients, Heliox, a mixture of helium and oxygen, can improve gas flow through a narrowed airway. Stridorous noise can diminish because the airway diameter is improving or because it is getting worse and less gas is passing through it. When in doubt it may be prudent to reintubate the patient and allow for decreased swelling or resolution of persistent neuromuscular blockade.

Complications of endotracheal intubation and endoscopic surgery of the larynx have been well documented ^[13]. These complications can occur regardless of whether spontaneous ventilation, mechanical controlled ventilation, or apneic ventilation methods are used. Such risks in singers and professional voice users are added burdens to the patient already anxious due to surgical considerations. Managing complications from intubation and anesthesia require a high index of suspicion and early intervention by laryngeal endoscopy.

Office-Based Laryngology and Phonomicrosurgery

With changes in demographics of the population and reimbursement, office-based procedures are increasingly more attractive as an option for the otolaryngologist ^[37]. Laryngoscopy, bronchoscopy, and esophagoscopy can be safely performed in this setting by the well-trained otolaryngologist. In general, most of the patients presenting for such procedures are elderly and have a positive history of smoking or chronic alcohol use and subsequent squamous cell carcinoma formation. Malignant lesions may be already identified or suspected. Foreign bodies represent another indication for performance of these procedures.

Procedures such as biopsies, injection laryngoplasty, and Cidofovir injections give the laryngologist new options in the office management of laryngeal lesions. Office-based steroid injection is one method for local delivery of antiinflammatory medication to the vocal folds without the complications of systemic steroids ^[26]. Increasingly the management of scar, vocal fold paralysis, and granulomas can be managed in the office setting as well as biopsies of malignant lesions. The use of lasers in the office setting has also increased ^[27].

Since office-based procedures often occur in outpatient offices without easy access to the emergency room and hospital, the need for safe yet effective local anesthesia is paramount. Chapter 20 discusses the logistics and administrative aspects of office-based practice for both the otolaryngologist and anesthesiologist. Acquiring the skills needed for good topical anesthesia of the upper airway is possible with proper patient selection, understanding of topical anesthetics and acquisition of skills in topical application. Unless the otolaryngologist has advanced ACLS life support training and access to full resuscitation support with adequate staff training, the use of office-based intravenous or intramuscular sedation in the clinic is not advised. The judicial use of local anesthetic during airway topicalization is not without risk, and the office-based otolaryngologist must also be knowledgeable about detection and treatments for local anesthesia toxicity^[29]. Although this can vary by practice and region, in closely monitored situations such as an appropriately equipped office (Chap. 21), the otolaryngologist may elect to

Table 10.1 Indications for office-based tracheoscopy and bronchoscopy

Larynx
Glottic stenosis
Subglottis stenosis
Amyloid tumor invasion of trachea
Wegener's granulomatosis
Hemoptysis
Cough
Trachea and bronchi
Tracheostomy granuloma
Tracheomalcia
Tracheal stenosis
Tracheal tumor
Tracheitis
Complications of tracheostomy
Assessment of airway after tracheostomy change
Foreign body obstruction

use intramuscular or intravenous anti-anxiety medication or analgesics to support the patient during office procedures or may seek the assistance of specially trained office-based anesthesiologists. They may also elect to perform the procedure in an outpatient surgery center or the ambulatory care center at a hospital that would have ready access to emergency support, and appropriately trained personnel. For the average busy clinic, however, this is not practical. A good topical anesthetic applied judiciously suffices in the majority of patients. Some of the indications for office-based tracheal bronchoscopy are listed in Table 10.1.

Preoperative Considerations

Otolaryngologist's Perspective

Laryngoscopy is a procedure by which the pharynx, hypopharynx, and larynx may be visualized for diagnostic and therapeutic benefits. Bronchoscopy is used for examination of the tracheobronchial tree and esophagoscopy to examine lesions of the esophagus. In the office, use of "suspension" laryngoscopy or rigid bronchoscopy is impractical given the lack of availability of general anesthesia or deep sedation. Hence, video and fiberoptic devices are crucial to the office setting as a way of doing these fairly invasive procedures in a comfortable way. Topical anesthesia procedures for the upper airway have become popular as flexible endoscopic procedures now allow the endoscopist to perform routine investigations of the upper aerodigestive tract in the office without sedation ^[38].

Selecting patients appropriate for office-based procedures should be considered of paramount importance. Some insight by the patient as to the procedure being done is also helpful. Informed consent that describes the airway topicalization process is necessary. It is helpful, for example, to describe for the patient what it will feel like to have local anesthesia of the larynx and pharynx. The taste of the local anesthetic, the discomfort with local anesthesia injections, and the loss of sensation with topicalization may be distressing for some patients. An accurate description of the duration of the procedure and how it will feel will help to allay the fears surrounding instruments in the mouth and nose. At the end of the informed consent process, some patients will decline the procedure. It is better to err on the side of caution and not start the local procedure than to spend a long period of time in an anxiety-ridden patient only to have the procedure fail because of patient reluctance to undergo the procedure in the first place. It is given that thorough trust in the otolaryngologist's ability by the patient will reduce the fear factor. If the otolaryngologist is confident, calm, and has good expectations for the patient to do well, it will set the tone for a positive experience. Forcing the issue is rarely successful if ever.

Anesthesiologist's Perspective

Anesthesiologists are often not involved in the management of brief office-based procedures such as laryngoscopy, bronchoscopy, and esophagoscopy. However, the assessment of patients and those that can and those that should not be managed in the operative setting is an important topic of discussion. Patients with significant comorbidities of the cardiovascular, pulmonary, hematologic, hepatic, and renal systems or those at significant risk of aspiration or those with significant psychologic or psychiatric disorders (Chaps. 7 and 20) are generally not good candidates for office-based procedures. Obviously any patient with a compromised airway (e.g., active stridor) or with an airway likely to be compromised by manipulation despite adequate topicalization should of course be evaluated and managed in the hospital setting.

Intraoperative Considerations

Otolaryngologist's and Anesthesiologist's Perspectives

Since much office-based laryngoscopy, bronchoscopy, and esophagoscopy may not be accompanied by intravenous anesthetics, anesthesiologists may not have direct involvement in these procedures. However, the largest obstacle to doing an interventional procedure of these kinds is the failure to obtain adequate local anesthesia of the larynx and pharynx and these considerations are important to both



Fig. 10.18 Setup for basic local anesthesia of the larynx and pharynx for rigid and fiberopic interventional laryngeal procedures: local anesthesia such as Pontocaine or Lidocaine; large particle nebulization sprays with directional control for local anesthetic spray of the nasal, oral, and hypopharyngeal area with the topical anesthetic; right-angled cotton ball carrier with cotton ball for direct application of topical anesthetic into the laryngeal introitus; kidney basin; long tipped cotton ball applicator for nasal anesthesia and decongestant application; disposable tissue

anesthesiologists and otolaryngologists alike. A well-done local anesthesia will allow the otolaryngologist to obtain reliable results with little variability. The duration of local anesthetic application is generally 10–15 min and should not be hurried.

Airway Topicalization

The materials necessary for local anesthesia of the larynx and pharynx are dependent on whether the oral approach or the nasal approach is to be used for the procedure. If the procedure is to be done by rigid endoscopy, only the oral cavity, oropharynx, hypopharynx, and larynx need to be anesthetized. If the procedure is to be done through the nasal cavity by a fiberoptic technique, the nasal cavity must be anesthetized and decongested. The equipment is simple and is shown in Fig. 10.18.

A large particle nebulizer that is powered by a forced air pump is necessary to spray the topical anesthetic into the oral and nasal cavities and hypophayrnx. It is helpful to have a nozzle on the nebulizer that can be directed downward to directly spray the hypophayrnx, base of tongue, and the larynx. From experience, sprays alone may not be adequate and direct topical application of anesthetics on a brush or cotton ball is often more efficient in achieving a fully anesthetized larynx to allow instrumentation. This is achieved with the use of a cotton ball with a long tail tied around a long silk tie. The cotton ball is introduced into the hypophayrnx and larynx by a right- cannula such as a right-angled cup forceps or a Clauert right-angled tonsil sponge carrier. A cotton tipped applicator is helpful for nasal decongestion and shrinkage of the turbinate to allow passage of the therapeutic flexible laryngoscope. The kidney basin and disposable wipes round out the local anesthetic kit. A nasal speculum and tongue blade may be needed to provide exposure with a headlight. If the procedure is to involve local injection of anesthesia, 2% Lidocaine is drawn up separately in a 27-gauge, 1.5-inch. needle with a 5-ml syringe.

The local anesthetic is first applied topically by spray into the oropharynx and hypopharynx. A medical assistant may do this spraying process but the authors insist on doing the direct topical application themselves. At the end of the local anesthetic application process, the expected result is a calm patient that can tolerate touching and manipulation of the vocal folds, larynx, and base of tongue. If the procedure is to include sub-glottis visualization or tracheal manipulation, these areas must also be systematically addressed during the local anesthesia process.

The topical medication that is used is 4% topical Lidocaine. The use of flavors or additives is to be avoided as in some patients it causes more throat irritation and cough. Cocaine has some theoretical advantages in terms of secretion and vasoconstriction but is not used due to fear of central excitation ^[39]. For vasoconstriction of the nasal cavity, 0.25% Neosynephrine spray is used. A topical Lidocaine soaked cotton strip placed in the nose to obtain adequate decongestion of the turbinate structures may be needed in some cases so as to pass the therapeutic scope.

To get adequate anesthesia of the larynx by local application, divide the local anesthesia into three parts. These three parts are: oral cavity and oropharyngeal anesthesia, hypopharynx and supraglottic anesthesia and glottic and subglottic anesthesia. If the procedure is to be successful at the glottic level, three levels must be anesthetized. First, spray the oropharynx with a spray of 4% Lidocaine with the nozzle directed into the oral cavity. Make sure the oropharynx is adequately sprayed. Second, spray the larynx and hypopharynx with a spray of 4% Lidocaine with nozzle of the nebulizer directed inferiorly. Third, perform direct application of the larynx and base of tongue with a cotton ball soaked in Lidocaine. After completion of these three maneuvers, the clinician should be able to perform direct trans-oral instrumentation and manipulation of the laryngeal mucosa without major disturbance to the patient. If airway protective reflexes are triggered, the reason is almost always due to lack of patience in allowing the anesthetic to work and seldom due to improper application. If so the procedures must be repeated so the patient is comfortable with the manipulations. These maneuvers do not ameliorate deep seated pain and cough

that may be elicited with deep injection or biopsy. Therefore, care in selection of patients that are appropriate for office trans-oral procedures under local anesthesia alone should be taken.

Procedures of the larynx involve the use of flexible endoscopy instrumentation with working channels. The flexible interventional laryngoscope has a working channel that can vary. Most channeled scopes have a working diameter of 2.0 mm. This will allow the placement of small cup forceps for biopsy of 1 mm diameter. It will allow the insertion of laser fibers such as optical quartz fibers from 300 to 600 µ. A #5 French pediatric suction catheter can be placed through the channel for the purpose of suction or for laser fiber protection as a sheath against scope damage. The latest generation of CO₂ laser waveguides can also now be placed through the channel of the fiberscope for laryngeal surgery. The main disadvantage of both indirect techniques through the transoral technique using a rigid angled laryngoscope and the trans-nasal technique through the fiberoptic endoscope is the lack of bimanual manipulation. The indirect technique gives the operator some tactile feedback as to the stiffness of the lesion and the mobility of the joint while the fiber optic technique may allow the operator greater access in areas such as the anterior commissure and the undersurface of the vocal fold and trachea. Selection of the technique is based on the site of the lesion, the skill of the operator with each technique and the cooperation of the patient. Prior publications have cited the efficient methods for delivery of office-based anesthesia for office laryngeal procedures ^[40].

The protocol for flexible and per oral laryngo-tracheal anesthesia is as follows. Each nostril is sprayed while the patient is asked to sniff quickly. Follow-up spray into the nasal cavity may be necessary to anesthetize the nasopharynx. Directing the nozzle of the spray straight while the tongue is depressed downward with the tongue depressor will also spray the oral cavity and the oropharynx. When the pharynx is numb, the patient must be asked to assume the endoscopy position. This requires the patient to lean forward with the chin thrust toward the operator. The patient is asked to hold his own tongue, roll the tongue down, and sit forward in a chin forward position. This will train the patient to put his head and neck into an "endoscopy" position for rigid interventional endoscopy. The spray is then directed downward 90°. By asking the patient to take a deep breath, the spray nebulizer is turned in a slow circle to liberally spray the larynx and hypopharynx. It is important to spray during inhalation. This is done twice. If the patient coughs, it must be repeated, as it will mean no anesthesia was delivered to the larynx and trachea. The patient is then observed for 10 min. If there is no further cough, the topical application continues by cotton ball application of the pharynx and the larynx.

The curved cannula with cotton is dipped in the topical 4% Lidocaine. The cotton ball should be 1.5 cm in size. It should have a slight tail so it will trail the anesthesia into the mucosal surfaces of the larynx and pharynx. The cotton ball will be used to paint each tonsil, the vallecula and then directed into each pyriform sinus along the lateral border of the hypopharynx. This anesthetizes the lingual, glosso-pharyngeal, and the internal branch of the superior laryngeal nerves. It is difficult to direct the cannula into the depth of the pyriform sinus to block the recurrent laryngeal nerve and direct application of the laryngeal introitus is preferred. To do this, the refreshed cotton ball is held by the operators' right hand and the cotton ball is directed into the introitus of the larynx by 2-3 cm. Visualization is provided by the left hand holding the video laryngoscope. The rigid technique is performed with the patient holding the tongue while assuming the head forward, neck flexed, head extended position. When the cotton ball is inserted deeply into the larynx, it will partially occlude the airway. The patient is instructed to cough with the cotton ball within the introitus of the larynx. This assures an even topical application to the entire supraglottic and glottic larynx. If additional anesthesia is required in the trachea for fiberoptic tracheoscopy or bronchoscopy, the therapeutic bronchoscope is placed deep into the trachea and additional anesthesia is instilled ^[41]. Alternatively, trans-tracheal block may be used by crico-tracheal puncture.

After 5–10 min, the patient should be ready for the local procedure. A test palpation of the larynx can be done using a blunt probe or the biopsy forceps. Tolerance by the patient to passive palpation should be confirmed prior to invasive procedures that may require suction, biopsy or injection.

The use of superior laryngeal nerve block in the office setting is usually not necessary. Its application is based on sound knowledge of the anatomy of the internal branch as it courses from the neck into the thyro-hyoid membrane. In the OR setting, the use of superior larvngeal nerve block can give a superior block to allow direct laryngoscopy and biopsy or injection laryngoplasty under local anesthesia ^[14]. The total anesthesia time necessary to anesthetize the larynx is about 15 min and cannot be rushed. Therefore it is best to have blocked time for this in the part of the day that is not hurried by other demands for the room. While the patient is undergoing the anesthetic process, the physician should be immediately available for any complications. Smelling salts and the ability to lie the patient completely flat is helpful if the patient should feel faint. Familiarity with the crash cart and the basics of oxygen administration and CPR is advisable but has not been made necessary.

Using this protocol, one can expect good nasal, oral, oropharyngeal, and laryngeal anesthesia for most rigid and flexible interventional laryngeal procedures. Additional supplementation of anesthesia by nerve block or oral medication may be needed but is not usual.

Topicalization for Flexible Bronchoscopy and Esophagoscopy

Fiberoptic tracheoscopy and limited bronchoscopy may be done in the office setting with the right patient selection and emergency cart setup. The anesthesia must be adequate to provide an unhurried view of the larynx and trachea. Patients with critical upper airway obstruction are not candidates for office bronchoscopy but dynamic upper airway obstruction or mild to moderate airway obstruction may be visualized by a fiber optic laryngoscope or bronchoscope. Unlike bronchoscopy done by the pulmonary physician, the otolaryngologist is primarily interested in bronchoscopy for evaluation of the upper aerodigestive tract.

Although therapeutic bronchoscopy can be done in the office setting, the author prefers to do this in the outpatient surgical suite due to the possible need for sedation. More commonly, the primary indication of office-based bronchoscopy is to provide a complete visual diagnosis in the assessment of integrity of the upper airway ^[42]. Thus diagnosis in the dynamic breathing patient is the main advantage of office-based tracheoscopy and bronchoscopy.

With today's modern chip-tip technology and small fiber size, the bronchoscopy can usually be performed through nasal endoscopy. The coupling of chip-tip technology coupled to a high quality video-endoscopy cart has made officebased tracheoscopy and bronchoscopy practical and reliable.

Local anesthesia for bronchoscopy is performed by large particle nebulizer by spraying the nasal cavity and the oral pharynx with the 4% Lidocaine. The nozzle of the nebulizer is then turned 90° and the spray is directed into the larynx while holding the tongue outwards. During the spray of the pharynx and the larynx, the patient is asked to pant back and forth to inhale the large particle Lidocaine into the airway. The patient must do this at least three times. Adequate time must then pass prior to instrumentation.

For tracheoscopy, the fiberoptic laryngoscope is placed through the nose and passed into the supraglottic larynx. At this time, the patient is asked to breath gently in an upright position. The endoscope is used to pass into the trachea. If the patient tolerates this without cough, the procedure proceeds with video recording. If the patient coughs or is unable to tolerate the procedure, the decision must be made to use Lidocaine by nebulization or use trans-tracheal block or superior laryngeal nerve block.

Use of nebulizer with 4% Lidocaine can be done with a standard compressed air nebulizer used for aerosolized medication administration. An Acorn type medication administration kit is hooked up to a compressed air source. And 4% plain Lidocaine is used for aerosol inhalation. The patient is asked to breathe the aerosolized 4% Lidocaine for 2 min. The smaller particle size will give better lower air way anesthesia and allow inspection of the lower airway with less cough than the large particle nebulizer which is excellent for the upper airway and the pharynx but may not get adequate deposit into the distal trachea and bronchi.

Use of trans-tracheal puncture can be performed. This is a convenient way to instill topical anesthesia into the trachea. The need for this is rare. It is best in patients with severe gagging. To do this a 25 gauge 1 inch needle with 3 ml syringe is used. The Lidocaine used is 4%. The needle is placed through the cricothyroid membrane using the midline top of the cricoid cartilage as a landmark. The needle is directed in the midline parallel to the lower border of the cricoid cartilage for a distance of 1 cm. While aspirating on the syringe, the needle is slowly advanced through the tough cricothyroid membrane until the airway is reached. The air will come through the needle indicating the needle is in the airway. The needle with the local anesthetic is used to puncture above the cricoid cartilage in the midline. Once air is aspirated into the needle, the topical anesthetic is injected quickly and the needle is withdrawn. A 2 ml bolus of 4% Lidocaine is instilled. This is to prevent the patient from coughing on the needle and causing bleeding. After injection, the patient is asked to cough. This anesthetizes the trachea and the larynx.

The superior laryngeal nerve block is rarely used in the office. This is because the sequelae from bilateral block are an unpleasant sensation. The patient often complains of not being able to swallow. The copious secretions that result from difficulty swallowing saliva can be bothersome in the office setting.

The anatomical landmarks necessary to do the superior laryngeal nerve block have been published ^[43]. To perform the block of the internal branch of the superior laryngeal nerve, the operator must have a good appreciation of the course of the internal branch of the superior laryngeal nerve as it enters into the larynx between the hyoid bone and the thyroid cartilage. The thyroid cartilage has a lateral depression between the superior thyroid horn and the body, the thyoid alae. This can be found by palpation by grabbing the thyroid cartilage by the posterior border and moving the fingers cephalad along the posterior border until the fingers fall into the thyro-hyoid space.

To perform the injection, 2% Lidocaine in a 5 ml 1 ½ inch 23 gauge needle is used. The approach is from lateral to medial. One hand is used to grasp the thyroid cartilage and rotate the larynx away from the side of nerve block. This will exaggerate the posterior border of the thyroid cartilage and the greater cornu of the hyoid bone. The needle is used to stab down to the posterior superior aspect of the superior thyroid cornu from a posterior to anterior direction. Once the superior cornu of the thyroid cartilage is found, the needle is pulled back and then advanced 1 cm anterior and slightly cephalad to the superior cornu to deposit 2 ml of Lidocaine. This will allow the local anesthetic into the thyro-hyoid membrane and block the internal branch of the superior laryngeal nerve. The same procedure is done on the contralateral side. Use of instilled Lidocaine through the operating channel of the therapeutic laryngoscope or bronchoscope is another way to deliver topical anesthetic into the larynx and trachea; 2 ml of 4% Lidocaine is placed through the instillation port of the therapeutic scope and prepared for instillation. The laryngoscope is placed at the rim of the epiglottis with a wide view of the endo-larynx. The patient is asked to take a deep breath. While the patient is inhaling inward, the 2 ml of Lidocaine is rapidly pushed and instilled into the larynx and trachea. This usually causes a brief cough which further achieves local upper airway anesthesia. As with use of any topical anesthetic, precautions on dosing and knowledge about treatment of anesthetic reaction are necessary.

Topicalization for Esophagoscopy

Anesthesia for office esophagoscopy is different than that for office bronchoscopy. With the TNE equipment, office-based esophagoscopy can now be performed through the nose with a minimal of difficulty under local anesthesia. This approach does not require sedation but requires good nasal decongestion and topical nasal anesthesia. Anesthesia to the hypophayrnx and oropharynx is useful. Laryngeal and tracheal anesthesia is not necessary and should be avoided.

The patient is examined in the sitting position. The nasal cavity is sprayed with topical Phenylephrine (Neosynephrine) (0.25%) and 4% Lidocaine. The nasal passage is inspected with a headlight and speculum. If the nasal cavity is narrow, it may be necessary to further vasoconstrict the nasal cavity with a cotton pledgett (4 cm × 1 cm) placed along the floor of the nose. The cotton pledgett is soaked in equal amounts of 4% Lidocaine and 0.25% Neosynephrine. The bayonet forceps is used to place the pledgett far enough along the entire floor of the nose to vasoconstrict the inferior turbinate. A headlight, nasal speculum, and bayonet forceps would be helpful. The pledgett is left in situ for 15 min until the lip and upper teeth are numb.

Next, the patient is given 2 ml of 2% viscous Lidocaine to gargle and swallow. The viscous Lidocaine should be gargled liberally for at least 2 min before swallowing. Once the pharynx is treated, the office esophagoscopy may commence.

The office esophagoscopy examination introduces a larger and longer scope into the esophagus and the primary site of patient discomfort is during the passage of the scope through the post-cricoid area and through the crico-pharyngeus. Once the endoscope is through the UES, there is surprisingly little discomfort. Two helpful hints for a successful office endoscopy with minimal patient discomfort are to insert the endoscope through the upper esophageal sphincter quickly during active swallow and to avoid excessive instillation of air to avoid esophageal and gastric distention. Suction of the air used for distention should be done in brief spurts to avoid nausea and vagus stimulation.

Postoperative Considerations

Otolaryngologist's and Anesthesiologist's Perspectives

Even though the procedures are more limited and for the most part performed under local anesthesia, the immediate postoperative period is critical and the patients must be evaluated for discharge readiness as they would be in an ambulatory or hospital-based setting. Because of the nature of the procedures many of the postoperative concerns and complications discussed in the previous section are no less relevant for patients cared for in the office setting. It is important to monitor the patients adequately in the procedure room or a monitored setting before they are moved to a non-monitored setting for further observation prior to discharge. Since most of the procedures are performed under local anesthesia, recovery should be expeditious, however due to the degree of topical anesthesia patients must refrain from oral intake due to the anesthesia of the upper airway and risk of aspiration. In addition, patients who have undergone an injection procedure should refrain from anticoagulant medications for 2-3 days to avoid the risk of a hematoma and secondary airway obstruction

Conclusion

Surgery of the larynx is diverse and challenging. Strong collaboration between two teams working in a small space means that a relationship between the anesthesiologist and surgeon is not only necessary, but crucial to optimize patient outcomes. We hope this chapter has shed some light on the major procedures and techniques necessary to this field.

Clinical Insights

For the Otolaryngologist (from the Anesthesiologist)

 Although oxygenation during jet ventilation is adequately measured by pulse oximetry, ventilation cannot be quantitatively assessed by noninvasive means.
 PaCO₂ evaluation requires arterial blood gas analysis.
 If jet ventilation is to be used for extensive periods of time, then arterial catheters are indicated.

- It is best to maintain HFJV catheters as far from the carina as possible to prevent jets from stimulating carina. Immediate consequences of carinal stimulation can be hypertension, tachycardia, and/or bronchospasm. Long-term consequence could be
- When performing apneic techniques, surgical time is related to successful pre-oxygenation of the FRC, which can take a while.

postoperative coughing and/or bleeding.

- Suspending the larynx during microlaryngoscopy is extremely stimulating and requires a deep level of anesthesia, which could take time to titrate off. It is critical to communicate with the anesthesiology team concerning the timing of the procedure and the expected time to completion.
- Patients with glottic or supra-glottic lesions typically progress better with intravenous induction and positive pressure ventilation by face mask than with classic inhalation induction and spontaneous ventilation.
- If apneic ventilation techniques are used with laser surgery, then allow time for washout of 100% oxygen before applying heat/energy sources.
- Before laser use, confirm accurate alignment of laser and sighting beams.

For the Anesthesiologist (from the Otolaryngologist)

- Because of the very nature of the "shared airway" during procedures such as bronchoscopy, laryngoscopy, or esophagoscopy, the importance of communication between otolaryngologist and anesthesiologist cannot be understated and in fact is critical for patient safety and optimized patient outcome.
- If possible avoiding the use of an *endotracheal tube stylet* is preferable when intubating patients with *pedunculated* or friable laryngeal lesions.
- Laser use is a fire risk and avoiding the use nitrous oxide and a high FiO_2 (greater than 30%) is mandatory.
- Constant communication is needed especially if apnea or repeated intubation and extubation will be utilized in the procedure.

Clinical Pearls

For the Otolaryngologist (from the Otolaryngologist)

- Because of the very nature of the "shared airway" during procedures such as bronchoscopy, laryngoscopy, or esophagoscopy, the importance of communication between otolaryngologist and anesthesiologist cannot be understated and in fact is critical for patient safety and optimized patient outcome.
- The classic "sniffing position" when properly executed will achieve an adequate view in the majority of laryngeal structures. Counter pressure by pushing down on the cricoid cartilage and the trachea helps to straighten the crico-tracheal angle and view the anterior commissure of the larynx during microlaryngoscopy.
- Getting the best exposure while sharing the airway can be challenging. It may mean that both otolaryn-gologists and the anesthesiologists may need to work on mutually acceptable compromises.
- Heliox, a mixture of helium and oxygen, can improve gas flow through a narrowed airway, but its administration needs to be made ahead of time. A discussion with the anesthesiologists concerning heliox's benefit and availability needs to be done well in advance of the case.
- Topicalization of the airway in the office takes time and a thorough knowledge of anatomy and pharmacology. Don't rush and make sure you aren't giving toxic levels of local anesthesia.

For the Anesthesiologist (from the Anesthesiologist)

- Small tracheal tubes facilitate surgical visualization and manipulation. They neither increase the risk of pneumothorax nor the work of breathing if controlled ventilation is used.
- Steel encased laser tubes should never be placed nasally and are not reliably placed via a flexible fiberoptic intubation.
- Laser safe tube lengths are generally not demarcated, it is recommended to mark depth prior to their use.
- Some jet ventilation catheters are laser resistant.

- In the event of an airway fire, extubation of patients whose airways are difficult to manage preoperatively must be evaluated on a case-by-case basis.
- Airway surgery increases the risks for postoperative respiratory obstruction. Awake patients can probably maintain airway patency better than anesthetized or heavily sedated ones. Deep extubation should be performed with caution.
- It is best to recover airway surgery patients in close proximity to their anesthesiologists and otolaryngologists.
- Jet ventilation techniques require upper airway patency. It is best to ask an assistant to hold the airway open.
- Coughing, bucking, and straining on emergence can adversely affect vocal cord healing and subsequent voice quality.

References

- 1. Orr R. Anesthesia for microlaryngoscopy. Paediatr Anaesth. 2005;15:81 [author reply 2].
- Altman KW. Vocal fold masses. Otolaryngol Clin North Am 2007;40:1091–108, viii.
- Ivey CM, Woo P, Altman KW, Shapshay SM. Office pulsed dye laser treatment for benign laryngeal vascular polyps: a preliminary study. Ann Otol Rhinol Laryngol. 2008;117:353–8.
- Friedrich G, Gugatschka M, Gugatschka M. Influence of head positioning on the forces occurring during microlaryngoscopy. Eur Arch Otorhinolaryngol. 2009;266:999–1003.
- Patel A, Rubin JS, Rubin JS. The difficult airway: the use of subglottic jet ventilation for laryngeal. Logoped Phoniatr Vocol. 2008;33:22–4.
- Mechenbier JA. Jet ventilator in microlaryngoscopy reduces anesthesia risks. Clin Laser Mon. 1992;10:23–6.
- Sims HS, Lertsburapa K. Pneumomediastinum and retroperitoneal air after removal of papillomas with the microdebrider and jet ventilation. J Natl Med Assoc. 2007;99:1068–70.
- Gaitini LA, Fradis M, Vaida SJ, Somri M, Malatskey SH, Golz A. Pneumomediastinum due to Venturi jet ventilation used during microlaryngeal. Ann Otol Rhinol Laryngol. 2000;109:519–21.
- 9. Biro P. Jet ventilation for surgical interventions in the upper airway. Anesthesiol. 2010;28:397–409 [Epub 2010 Aug 11].
- Davies JM, Hillel AD, Maronian NC, Posner KL. The Hunsaker Mon-Jet tube with jet ventilation is effective for microlaryngeal. Can J Anaesth. 2009;56:284–90 [Epub 2009 Feb 25].
- Hunsaker DH. Anesthesia for microlaryngeal surgery: the case for subglottic jet ventilation. Laryngoscope. 1994;104:1–30.
- Buczkowski PW, Fombon FN, Lin ES, Russell WC, Thompson JP. Air entrainment during high-frequency jet ventilation in a model of upper. Br J Anaesth. 2007;99:891–7.
- Jaquet Y, Monnier P, Van Melle G, Ravussin P, Spahn DR, Chollet-Rivier M. Complications of different ventilation strategies in endoscopic laryngeal. Anesthesiology. 2006;104:52–9.
- 14. Barker I, Bull PD. Tubeless anaesthesia for microlaryngoscopy. Paediatr Anaesth. 1999;9:94.

- Thaung MK, Balakrishnan A. A modified technique of tubeless anaesthesia for microlaryngoscopy and bronchoscopy in young children with stridor. Paediatr Anaesth. 1998;8:201–4.
- Connelly A, Clement WA, Kubba H. Management of dysphonia in children. J Laryngol Otol. 2009;123:642–7 [Epub 2009 Feb 16].
- Windfuhr JP, Remmert S. Intubation laryngeal mask: atraumatic diagnostic tool in suspension laryngoscopy. Acta Otolaryngol. 2005;125:100–7.
- Schindler A, Capaccio P, Ottaviani F. Videoendoscopic surgery for inaccessible glottic lesions. J Laryngol Otol. 2005;119: 899–902.
- Weiss M. Video-intuboscopy: a new aid to routine and difficult tracheal intubation. Br J Anaesth. 1998;80:525–7.
- Pinosky M. Laryngeal mask airway: uses in anesthesiology. South Med J. 1996;89:551–5.
- Ogata J, Horishita T, Minami K. The airway management using laryngeal mask airway and tracheal fiberscopy in a pediatric patient with tracheal stenosis after tracheostomy. Masui. 2004;53:1282–5.
- Friedlander P, Aygene E, Kraus DH. Prevention of airway complications in thyroplasty patients requiring endotracheal. Ann Otol Rhinol Laryngol. 1999;108:735–7.
- Zestos MM, Hoppen CN, Belenky WM, Virupannavar V, Stricker LJ. Subglottic stenosis after surgery for congenital heart disease: a spectrum of of severity. J Cardiothorac Vasc Anesth. 2005;19:367–9.
- Santos PM, Afrassiabi A, Weymuller Jr EA. Risk factors associated with prolonged intubation and laryngeal injury. Otolaryngol Head Neck Surg. 1994;111:453–9.
- Lewis KE. Transtracheal lignocaine: effective treatment for postextubation stridor. Anaesth Intensive Care. 2007;35:128–31.
- Mortensen M, Woo P. Office steroid injections of the larynx. Laryngoscope. 2006;116:1735–9.
- Zeitels SM, Burns JA, Akst LM, Hillman RE, Broadhurst MS, Anderson RR. Office-based and microlaryngeal applications of a fiberbased thulium laser. Ann Otol Rhinol Laryngol. 2006;115:891–6.
- Welty P. Anesthetic concerns and complications during suspension microlaryngoscopy. Crna. 1992;3:113–8.
- Werkhaven JA. Microlaryngoscopy-airway management with anaesthetic techniques for CO(2) laser. Paediatr Anaesth. 2004;14:90–4.

- Hoing R, Loick HM, Anger C. Effect of preventive glucocorticoid administration on edema formation and inflammation susceptibility after microlaryngoscopy. Laryngorhinootologie. 1992;71: 145–8.
- Devaney KO, Rinaldo A, Ferlito A. Vocal process granuloma of the larynx-recognition, differential diagnosis and treatment. Oral Oncol. 2005;41:666–9.
- Tahir MZ, Shamim MS, Jooma R. Vocal fold granuloma after prolonged neurosurgical procedure with wire reinforced. J Pak Med Assoc. 2008;58:658.
- Yilmazer C, Sener M, Yilmaz I. Bilateral giant posterior laryngeal granulomas with dyspnea: a rare complication. Anesth Analg. 2005;101:1881–2.
- Lin DS, Cheng S-C, Su W-F. Potassium titanyl phosphate laser treatment of intubation vocal granuloma. Eur Arch Otorhinolaryngol. 2008;265:1233–8.
- Postma GN, McGuirt Sr WF, Butler SG, Rees CJ, Crandall HL, Tansavatdi K. Laryngopharyngeal abnormalities in hospitalized patients with dysphagia. Laryngoscope. 2007;117:1720–2.
- Mevorach DL. The management and treatment of recurrent postoperative laryngospasm. Anesth Analg. 1996;83:1110–1.
- Blake DR. Office-based anesthesia: dispelling common myths. Aesthet Surg J. 2008;28:564–70 [discussion 71–2].
- Simpson CB, Amin MR, Postma GN. Topical anesthesia of the airway and esophagus. Ear Nose Throat J. 2004;83:2–5.
- Rubin HJ. Toxicity of certain topical anesthetics used in otolaryngology; an experimental. AMA Arch Otolaryngol. 1951;53: 411–20.
- Sulica L, Blitzer A. Anesthesia for laryngeal surgery in the office. Laryngoscope. 2000;110:1777–9.
- Kundra P, Kutralam S, Ravishankar M. Local anaesthesia for awake fibreoptic nasotracheal intubation. Acta Anaesthesiol Scand. 2000;44:511–6.
- 42. Cosano Povedano A, Munoz Cabrera L, Cosano Povedano FJ, Rubio Sanchez J, Pascual Martinez N, Escribano Duenas A. Endoscopic treatment of central airway stenosis: five years' experience. Arch Bronconeumol. 2005;41:322–7.
- Stockwell M, Lozanoff S, Lang SA, Nyssen J. Superior laryngeal nerve block: an anatomical study. Clin Anat. 1995;8:89–95.

Otologic Surgery

Cheryl K. Gooden and Sujana S. Chandrasekhar

11

Introduction

Although otologic surgery encompasses a wide range of procedures from simple myringotomy with tube placement to radical mastoid tumor resections, many of the surgical and anesthetic considerations for these procedures are universal throughout the spectrum of disease prevalence and severity. The seasoned otolaryngologist knows that optimal otologic outcomes will be achieved if there has been clear and thorough communication with the anesthesiologist, as well as appropriate preoperative preparation before the patient reaches the operating room. For otology cases there are requirements to avoid certain anesthetic agents (such as nitrous oxide) and care in patient positioning. In addition, strategies that best reduce the incidence of postoperative nausea and vomiting (PONV), minimize surgical bleeding, and prevent movement in the absence of muscle relaxant must also be considered ^[1]. As with other otolaryngologic procedures, effective communication and planning on the parts of surgeon and anesthesiologist ensure safe and successful surgery.

Overview

In general, otologic procedures are elective surgeries of the external and middle ear structures done to improve patient quality of life whether through restoration of hearing, decrement of infection or improvement of cosmetic defects. Common preoperative diagnoses for otologic surgery patients

C.K. Gooden

include chronic otitis media, cholesteatoma, congenitally deformed ear, conductive hearing loss, neoplasm, or trauma. Figure 11.1 details the components of the external and middle ear.

The most common operation performed in the USA is myringotomy with tube insertion (M&T), which is covered in greater detail in Chap. 20. Other common otologic operations include myringoplasty, tympanoplasty with or without ossiculoplasty, stapedectomy or stapedotomy, middle ear exploration, perilymph fistula repair, chemical labyrinthotomy, canalplasty, mastoidectomy, and implantable hearing devices, such as osseointegrated cochlear conducting devices (bahaTM and PontoproTM), cochlear implants, and implantable hearing aids (Fig. 11.2). The particular considerations unique to neuro-otologic procedures such as acoustic neuroma resection are discussed in Chap. 17.

Age at surgery is an important consideration for otologic procedures. Both otolaryngologist and anesthesiologist must be cognizant of issues relating to both otology and high risk age groups such as children and the elderly. The average age for patients undergoing M&T is 3 years. This procedure is uncommonly performed before age 9 months ^[2]. Adult M&T procedures are often performed outside of an operating room setting. Cochlear implant surgery is FDA approved in the USA for children as young as age 1 year, and is not infrequently performed in younger children, predominantly between the ages of 9 and 12 months. This entirely elective procedure is performed in all age groups, including in the geriatric population.

Acute mastoiditis unresponsive to intravenous antibiotic therapy and/or presenting with a complication such as facial palsy or intracranial infection necessitates either emergency M&T and/or emergency cortical mastoidectomy ^[4, 5]. The median age for acute mastoiditis in children under age 10 years is 14 months (range: 5 months to 8.75 years), with an incidence rate of 7.4 in this age group versus 1.4 in the population overall ^[6]. Although the surgery time for cortical mastoidectomy in these cases is generally less than 60 min, these often very young children are quite sick and may even become septic, adding to the concerns regarding emergency surgery in these patients.

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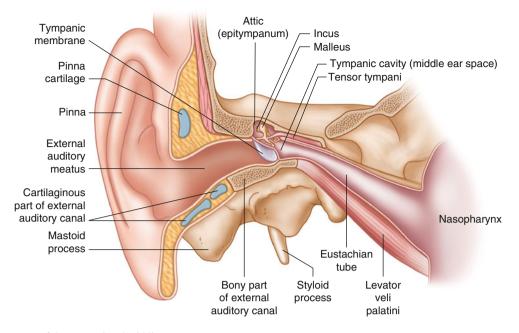


Fig. 11.1 Components of the external and middle ear

Cholesteatoma is an expanding and thus destructive cyst of keratinizing squamous epithelium in the middle ear and/or mastoid process. It may be congenital or acquired and presents in childhood with an incidence of 3 in 100,000 compared with 9 in 100,000 in adults ^[7]. Because of factors relating to eustachian tube development, some surgeons may opt to delay operating on non-cholesteatomatous chronic otitis media (COM) until the child is 9 or 10 years old ^[7, 8]; however, there is no clear evidence that delaying surgery results in better surgical outcomes ^[9]. Non-cholesteatomatous COM includes tympanic membrane perforation, ossicular abnormalities, and chronic mastoiditis. Due to its destructive nature, cholesteatoma is an absolute indication for surgery that must be performed at the time of presentation.

Osseointegrated cochlear conducting device implantation is FDA approved in the USA from age 5 years onward; the surgery has been performed safely in children as young as age 2. Unlike other otologic procedures, this operation does not involve the middle ear or mastoid air cell system and is not dependent on eustachian tube function for healing.

Otologists have developed office-based techniques to manage disabling vertigo, including chemical labyrinthotomy via transtympanic injection. However, certain patients, including a percentage of geriatric patients, will require operative intervention.

Preoperative Considerations

Otolaryngologist's Perspective

Regardless of the surgical procedure planned, a thorough preoperative assessment is important to a successful operation. Otologic surgery is generally elective, quality-of-life surgery. For most healthy pediatric patients, little workup is needed other than a well-child visit and assurance that the child is not currently ill (detailed in Chap. 20). For adults, most of the workup prior to surgery is patient-centered as these procedures tend to have limited blood loss and fluid shifts. For a thorough review of the evidence-based approach to preoperative assessment, see Chap. 7. Since these procedures are generally elective, it is critical that the patient is evaluated and any medical condition optimized preoperatively.

Patient expectations following otologic surgery are for a safe, dry ear with no tympanic membrane perforation, hearing improvement, and no long-term vertigo or dizziness. Surgical outcomes for the following procedures should be: 90% closure of tympanic membrane perforation following myringoplasty or tympanoplasty with closure of air-bone gap (restoration to best nerve hearing level); 85–90% closure of air-bone gap following partial ossicular reconstruction



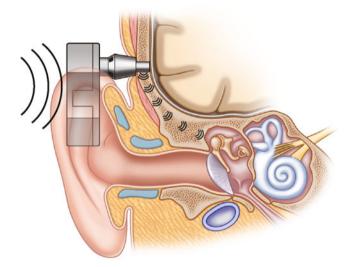


Fig. 11.2 Baha implant location and placement

surgery; 75–85% closure of air-bone gap following total ossiculoplasty; over 80% amelioration of vertigo following middle ear exploration (MEE) with perilymphatic fistula repair or endolymphatic sac operation; and over 90% vertigo relief after MEE with topical aminoglycoside chemical laby-rinthotomy ^[10]. Patients undergoing cochlear implantation expect that the implant will be placed correctly inside the cochlea with minimal damage to the cochlea, and that hearing following surgery is remarkably better ^[11].

A surgery of particular concern is atresiaplasty surgery. This surgery creates a congenitally absent external ear canal. These procedures are generally deferred until at least age 5 or 6, if not later, and are performed well after any auricular reconstruction procedures are complete. As such, these children often have had several surgical experiences in the past, and frequently have body image concerns. These are important factors to take into consideration when preparing them for surgery. Atresiaplasty procedures can take from 3 to 6 h to perform, and success depends on surgical skill, intraoperative facial nerve monitoring, and postoperative ear care ^[12]. The anesthesiologist must be aware about the length of the procedure, the positioning, and the management of the anesthetic without muscle relaxants in a young patient. The patient and his or her family need to be reassured and dealt with in as sensitive a manner as possible.

Anesthesiologist's Perspective

As important as the challenges are to the anesthesiologist providing care to the patient undergoing otologic surgery, so are the patient's expectations. In the past, an overnight hospital stay may have been a requisite following otologic surgery ^[13]. However, for the most part this is no longer the situation. Whether the patient is a child or adult, the expectation is that many of these procedures will be performed on an outpatient basis. This is a realistic expectation since many of these procedures are now considered minimally invasive. Ultimately, what this means for the anesthesia provider is the requirement to develop an anesthesia management plan that will allow the patient to meet discharge criteria in a safe and efficient manner.

The preoperative process for any patient begins once the decision is made to proceed with surgery. For the most part, the anesthesiologist relies upon their surgical colleague for obtaining all the required evaluations and studies prior to the date of surgery, especially for ambulatory procedures. As such, it is important for surgical staff to be aware of the latest guidelines on preoperative test ordering to avoid unnecessary testing and unnecessary case cancellations. The American Society of Anesthesiologists has issued practice guidelines for preoperative testing and both surgeons and anesthesiologists should be familiar (see Chap. 7). Depending on the institution, some patients may be evaluated by an anesthesiologist several days or more before their scheduled surgery ^[14]. Whether or not the preoperative assessment by the anesthesiologist occurs on the day of surgery or several days prior, the key to any assessment is an efficient and evidencebased approach.

The preoperative anesthesia assessment for the patient undergoing otologic surgery will not differ much from most other anesthesia assessments. That being said, there still remains the need to individualize the assessment to this group of patients undergoing middle ear surgery. The anesthesia provider must be cognizant of whether or not their patient has an associated decrease in hearing acuity or utilizes a hearing aid as this can make communication challenging. Common manifestations of ear-related disorders may include nystagmus, vertigo, nausea, and vomiting. As such, patients may be very uncomfortable prior to surgery. An inquiry into a history of motion sickness, or postoperative nausea and vomiting (PONV) is useful for the more targeted anesthesia plan. Ultimately, the information obtained during the assessment will facilitate the decision as to whether the patient is in his/her optimal state of health and whether there are any interventions best suited to the particular patient.

The majority of otologic surgery will be performed under general anesthesia. However, monitored anesthesia care (MAC) for some of these procedures is quite reasonable. In our institution, many stapedectomies are performed under MAC. This provides the surgeon with the opportunity to perform intraoperative hearing tests. Careful selection of patients for sedation procedures is crucial. For example, during an office visit our surgeon will evaluate whether a patient meets the surgical criteria for a sedation technique. What the patient should expect as well as what is required of the patient ought to be discussed in detail. If the patient expresses an interest in sedation for their procedure further evaluation and discussion will take place by the anesthesiologist on the day of surgery. The criteria for a patient undergoing stapedectomy under sedation may include the following: (1) normal or "reassuring" airway, (2) non-obese or without other full stomach/pulmonary aspiration risks, (3) no history of claustrophobia or panic attacks, (4) comfortable with operating room noise, and (5) a complete understanding of what is required on the part of the patient (i.e., stillness, relative awake state. Of course, the decision to perform these surgeries under sedation will vary among anesthesiologists and surgeons.

Intraoperative Considerations

Otolaryngologist's Perspective

Nearly all otologic procedures, including myringotomy surgery (with or without tube insertion), offer unique challenges in terms of patient, operating table, and imaging equipment positioning. The ideal positioning of the "players" in an otology case is as follows (Fig. 11.3)

The patient is placed on the table with his or her head at the foot of the bed. This enables the surgeon to sit comfortably during surgery with his or her feet under the table, unimpeded by the center "core" of the electric/pneumatic table. The head of the operating table is turned 180° away from the anesthesiologist; that is, the patient's feet are toward the anesthesia machine. The surgeon is seated on the side of the operated ear. The scrub technician is positioned directly opposite the surgeon; that is, on the non-operative side, so that instruments can be passed directly to the surgeon. The patient's head is turned away from the operated ear. The operating microscope, if on a floor stand, is brought in from the head of the bed. If it is ceiling-mounted, this is not a concern.

The general surgical approach is transcranial or postauricular (i.e., through the mastoid) for all of these surgeries (Fig. 11.4). Because the operating table is often tilted ("airplaned") rather dramatically, the patient is secured to the table with two extra belts or tape in addition to the usual

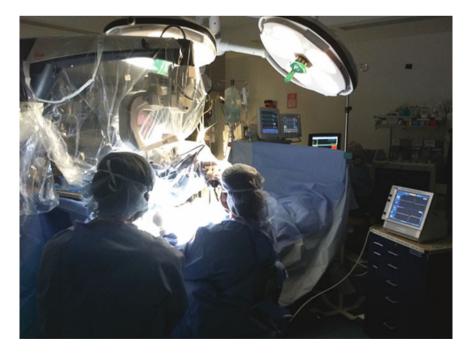
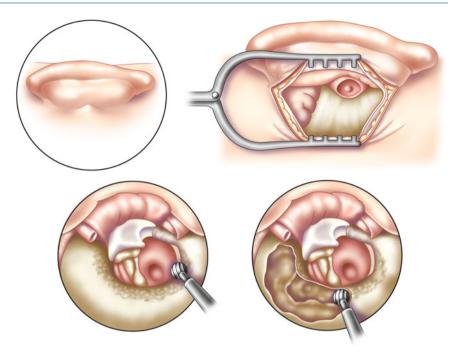


Fig. 11.3 Positioning of operating room staff before and after draping. Note that the facial nerve monitoring system is on the operative side, the blood pressure cuff and electrocautery unit are on the non-operative side, and the anesthesiologist is at the foot of the bed

Fig. 11.4 Surgical exposure for otologic procedures



single belt, and the anesthesiologist is given the bed controller. Care must be taken to tilt the bed slowly throughout the surgery and to make sure the patient is not sliding as the risk of falling from the table is evident.

Because of the delicate microscopic nature of otologic surgery and the position of the surgeon relative to the side of the patient, the blood pressure cuff is best placed on the arm opposite to the operated ear, while the intravenous line is ideally placed in the ipsilateral arm. This is to avoid interference with the surgeon's arm as the BP cuff inflates intermittently which might impair the ability to work in a small surgical space.

Facial nerve electromyography monitoring (FNM) is often used during otologic surgery, especially during mastoid surgery (Fig. 11.5).

FNM needle electrodes are placed into the ipsilateral orbicularis oris and orbicularis oculi musculature, with ground electrodes placed in the neck or chest (Fig. 11.6).

Communication between surgeon and anesthesiologist ensures that this can be accomplished seamlessly, without interference between anesthetic and otologic monitoring equipment. The facial nerve monitor can receive interference from the electrocautery machine and is often monitored auditorially by the surgeon; therefore, the FNM should be placed on the side of the surgeon while the electrocautery machine is on the contralateral side. The FNM has a grounding cable that is placed at the non-sterile part of the cautery cord, in order to mute the monitor during cautery use.

The operating drill is set up near the cautery, with irrigation either from within the drill system or separately from the foot of the bed near the anesthesiologist. Laser equipment, when used, is brought in from the microscope side or from

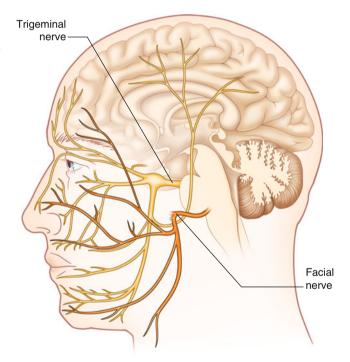


Fig. 11.5 The facial nerve distribution

behind the surgeon. If the microscope is equipped with closed circuit television, it is usually set up so that the screen is visible by the scrub as well as by the anesthesiologist and any observers. The use of endoscopes is expanding in otologic surgery. The endoscope is connected to the same video monitor, which can be moved so that the surgeon can use it for that portion of the operation.

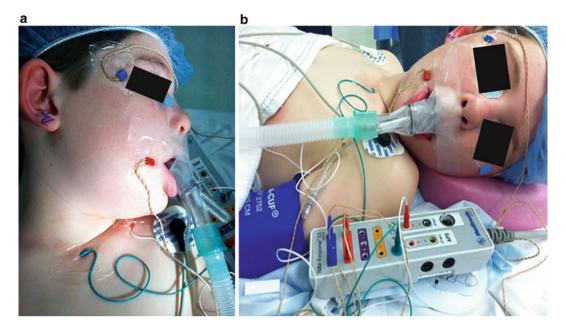


Fig. 11.6 Positioning of facial nerve monitoring equipment and electrodes

Procedure	Average operative time
M&T	15–20 min
MEE	45–60 min
Myringoplasty	30-60 min
Tympanoplasty with or without ossiculoplasty	45 min to 2 h
Tympanoplasty with mastoidectomy	2–4 h
Simple "cortical" mastoidectomy	1 h
Stapedotomy	30-60 min
Cochlear implant	2–4 h
Baha TM or Pontopro TM implant	30-60 min
Implantable hearing aid	4–6 h
Endolymphatic sac procedure	60–90 min
Atresiaplasty	4–6 h
Glomus tympanicum tumors	1–2 h
Glomus jugulare tumors	4–8 h

 Table 11.1
 Common procedures and surgical times

The times (Table 11.1) are gross averages; the surgeon should be able to anticipate his or her own surgical time within a reasonable margin of error. This allows the anesthesiologist to titrate medications appropriately. If, during the course of the surgical procedure, a situation is encountered wherein the surgical time will be significantly longer, the care of the patient is served best if the anesthesiologist is alerted to this fact promptly. In almost all otologic surgery procedures, there are no significant amounts of blood loss or fluid shifts encountered. The anesthesiologist should be aware of fluid used for irrigation during mastoid surgery, which will accumulate in the suction canisters and may lead to an erroneous assumption of blood loss. However, significant blood loss can occur during glomus tumor surgery (Chap. 17). For sizeable glomus tumors, this will have been anticipated and the patient will frequently have undergone preoperative embolization to minimize the amount of blood loss. Additionally, blood should be available for transfusion in the operating room, should the need arise. If a glomus tumor is extending into the neck, a transmastoid and transcervical approach may be required and rapid blood loss is possible.

Significant unanticipated blood loss can also occur if either the sigmoid sinus or jugular bulb is violated during tympanomastoid surgery. The actual blood loss is usually less volume than it appears to be, due to magnification under the operating microscope, but this is nonetheless an alarming situation. In this occurrence, the anesthesiologist should be alerted immediately. Because this is a low-flow venous bleed, it is usually controlled readily with packing and other surgical manipulation, and most often does not require transfusion.

Anesthetic care of the otologic surgery patient is challenging due to limitations imposed by the surgical site and the surgical technique on the medications that may or may not be administered during surgery, as well as by the desire by both surgeon and anesthesiologist to limit postoperative vertigo and nausea, which are much more common problems than is pain. Paralytic agents are contraindicated in most otologic surgeries, whether or not facial nerve electromyography monitoring is being employed. The facial nerve's extracranial, intratemporal course is a convoluted one through the temporal bone, until it exits at the stylomastoid foramen and travels anteriorly and passes through the parotid gland where it divides into five major branches. Injury to the facial nerve can occur anywhere along its course, and, if the patient has received muscular relaxants, this injury may go unrecognized.

Due to the blood gas insolubility of nitrogen compared to inhaled nitrous oxide (N₂O) (34 times less soluble) N₂O will accumulate in air-filled spaces, i.e. the middle ear space, 34 times quicker than nitrogen can exist, increasing the pressure in that space. Additionally, there are concerns regarding postoperative nausea and vomiting following the use of N₂O ^[15]. The mechanical effects of N₂O are of concern to the otologic surgeon, since the increased middle ear pressure can interfere with maintenance of position of graft material for tympanoplasty and/or ossicular reconstruction prostheses. If used, it is common for the anesthesiologist to turn off the N₂O approximately 15 min before the middle ear is packed and prepared for the tympanoplasty and/or ossiculoplasty; it is also common for the anesthesiologist to avoid the use of N₂O entirely during these cases.

One instance in which N_2O may be useful to the otologic surgeon is during M&T surgery or tympanoplasty for a severely retracted tympanic membrane. In these cases, the accumulation of N_2O that increases the pressure in the middle ear space can be used to allow the tympanic membrane to lift itself off of the ossicles and the promontory, allowing the surgeon to identify reversible disease, and enabling placement of a ventilation tube into a previously nonexistent space. The surgeon may ask the anesthesiologist to deliver N_2O for a few minutes while he or she continues to examine the ear under the microscope, and then stop the N_2O once its effect is realized.

Otologic surgery can result in vertigo and nausea as a direct result of manipulation of the structures of the middle and, indirectly, the inner ear. Healing after otologic surgery, however, is greatly dependent on maintaining a steady pressure gradient in the middle ear; that is, avoiding vomiting, nose-blowing, and straining. Intraoperative intravenous steroid administration is employed to reduce nausea and, if there are any concerns, to treat transient facial neuropraxial. However, the anesthesiologist who can deliver the proper "cocktail" of intraoperative medications that minimize or eliminate postoperative vertigo, nausea and vomiting and bucking on emergence contributes greatly to excellent oto-surgical outcomes.

Anesthesiologist's Perspective

The aim of any anesthesia plan is designed to ensure hemodynamic stability, amnesia, and analgesia. While standard ASA monitors are appropriate for all but the most rare otologic surgeries (i.e., large glomus tumor excisions which may require arterial line placement for beat-to-beat blood pressure monitoring), there are other unique features of otologic surgery that must be addressed in the anesthesia plan. First, many of these otologic procedures are performed with the use of a surgical microscope, therefore requiring a surgical field as free of blood as possible. A small amount of blood can obscure the surgeon's view through the microscope. Second, a number of otologic surgeries will incorporate the use of FNM that necessitates a motionless patient in the absence of muscle relaxant. Injury to the facial nerve is an obvious concern. The incidence of iatrogenic injury to the facial nerve has been determined to range from 0.6% to 3.6% ^[16]. Lastly, patient positioning is an important part of these procedures.

During surgery the head of the operating room table may be turned 90-180° away from the anesthesiologist. In addition, the patient's head and neck will be rotated to the opposite side from the operative field. Avoidance of hyperextension of the neck limits the likelihood of nerve injury. Also, the dependent ear and eve should be free of excessive pressure especially in long cases. Because of the unique positioning in otology, the anesthesiologist will be a distance from the airway. Prior to turning, our group positions and drapes intravenous and monitor cables in such a way (down the right side of the patient and coming up from the feet) so that the only thing disconnected prior to the turn is the breathing circuit. This limits the need to disconnect monitors prior to turning and allows for continuous monitoring throughout the 180° turn without tangling monitoring cables. Alternatively, monitors can be disconnected prior to turning and swiftly reconnected once the bed is in its final position. With proper preparation and adequate securing of the airway device, this can be accomplished with both endotracheal tubes and laryngeal mask airways, as well as for monitored anesthetic care (MAC) (i.e., "sedation"). For general anesthesia, long anesthesia circuit tubing is required and it is best to recheck all connections and that gas exchange is optimized in what will be the final surgical position prior to draping.

During MAC cases, claustrophobia, rebreathing carbon dioxide and creating combustible pockets of enrich oxygen, can be avoided or greatly diminished by placing the anesthesia circuit holder, often called a "Christmas tree," next to the patient's head and placing drapes over this, leaving a significant amount of space between the patient's face and the drapes. This space together with the use of as little supplemental oxygen as possible is critical during MAC cases of the head and neck in order to avoid a catastrophic fire. This author uses a cool compress over the patient's eyes to help them relax; some surgeons advocate using an MP3 player with the earphone in the nonoperated ear for the same purpose. The best patient outcomes in otologic surgery under MAC anesthesia occur when the surgeon and anesthesiologist have taken the time to explain the procedure, including anesthetic expectations, to the patient preoperatively, and the patient enters with proper expectations of the anesthesia plan (i.e., not that they will "be asleep").

The choice of inhalational anesthetic agents or total intravenous anesthesia (TIVA) and whether one is more beneficial than the other for otologic surgery is still controversial. Jellish and colleagues ^[17] compared the use of isoflurane to TIVA with propofol and fentanyl in patients who had undergone middle ear surgery. More patients in the TIVA group with propofol had a quicker emergence with less nausea and vomiting. In another study of Jellish and colleagues ^[1], desflurane was compared to sevoflurane for middle ear surgery. Intraoperative hemodynamics was not different between the two groups. Similar results were observed in the desflurane and sevoflurane groups with regards to pain scores, nausea and vomiting, use of rescue antiemetics, recovery scores, and discharge times.

The issue of whether or not N₂O should be used at all during otologic surgery also remains controversial. There are several properties of N₂O that would suggest avoiding its use during middle ear surgery. N₂O is more soluble than nitrogen in blood. As a result, when N₂O is utilized as part of the anesthetic technique it can move more quickly into the middle ear space than nitrogen can move out ^[18]. This is problematic in the presence of a perturbation of the eustachian tube, such as a blockage. This results in an increase in middle ear pressure. For example during tympanoplasty, the tympanic membrane graft may become dislodged due to excessive middle ear pressure from the use of N₂O. N₂O is also considered a risk factor for postoperative (PONV) and postdischarge nausea and vomiting (PDNV)^[19]. In conjunction with the use of N₂O is the associated risk factor of ear surgery for PONV and PDNV. Indeed, consideration must be given to whether there is any benefit derived from the use of N₂O during ear surgery. In general, if tympanoplasty or stapedotomy or stapedectomy is planned, N₂O should be avoided.

Remifentanil is a rapid short-acting selective opioid agonist. The short half-life of remifentanil is secondary to its metabolism by nonspecific tissue and plasma esterases. Remifentanil is suitable for otologic surgery because of its potency, short half-life, and its ability to be easily titrated. Jellish and colleagues ^[20] compared a remifentanil-based anesthetic with supplemental propofol to a propofol-based anesthetic with supplemental fentanyl to determine which provides the best perioperative conditions for otologic surgery. The results of their investigation revealed that the remifentanil technique decreased surgical movement, provided superior hemodynamic stability, and resulted in a more rapid emergence from anesthesia at a cost similar to the propofol-based technique.

Dexmedetomidine is an alpha-2-adrenergic agonist with hypnotic, sedative, and analgesic effects ^[21]. The properties of dexmedetomidine that make it most appealing for otologic surgery are that it provides hemodynamic stability and controlled hypotension. The result is a surgical field with minimal bleeding that makes it quite suitable for the surgeon operating with a microscope. It is most probable that dexmedetomidine can be used as an adjuvant agent during MAC or general anesthesia in otologic procedures. However, currently the experience with the use of dexmedetomidine in middle ear surgery is limited but emerging.

The laryngeal mask airway (LMA) is a supraglottic airway device that may be considered for use during otologic surgery

Table 11.2 Antiemetic drugs and doses			
Timing	Drug	Dose/route	
Preoperatively	Dexamethasone	8–12 mg/IV	
Intra/postoperatively	Ondansetron Droperidol	4–8 mg/IV 0.625 mg/IV	
Night before surgery	Scopolamine	Transcutaneous patch	

instead of the conventional endotracheal tube (ETT) or oral RAE tube. Avala and colleagues [22] evaluated the safety and efficacy of the LMA compared with oral ETT in patients who underwent otologic surgery. The LMA was determined to be a safe alternative to the ETT with no observed increased risk of airway complications in patients undergoing otologic surgery. The decision-making process by the anesthesiologist as to whether or not an LMA is used during otologic cases should be the same as with any surgical procedure. That being said, one major factor to consider is that more than likely the patient's airway will be a distance away from the anesthesia provider. In our practice we do prefer using the LMA for most otologic surgeries unless contraindications to its use prevail (full stomach, restrictive lung disease, etc.). Its reliable and predictable placement without the need for neuromuscular blocking agents and the ability to remove it at the end of the procedure, with minimal stimulation makes it a very favorable and efficient alternative to endotracheal intubation. We do recommend that once placed, proper LMA "seating" is confirmed with the head turned with the operative site up be done before turning away from the anesthesia machine or securing the LMA.

The use of antiemetic therapy in otologic surgery plays a significant role. These procedures tend to be associated with a high incidence of PONV. Several studies suggest that the incidence of PONV after middle ear surgery is between 50% and 80% ^[23, 24]. Therefore it is clear that the incidence of PONV is significant and that patients undergoing middle ear surgery will benefit most from prophylactic treatment. The risk of PONV will depend on patient, anesthetic, and surgical factors. A number of antiemetic drugs are available for single or combination therapy (e.g., dexamethasone, ondansetron, and droperidol). The reader is advised to familiarize themselves with the various antiemetic treatment options including the dose and dose timing, the potential side effects and contraindications. In short, multimodality antiemetic therapy (Table 11.2).

Postoperative Considerations

Otolaryngologist's Perspective

Recovery following M&T surgery is quite rapid. Recovery following all other types of ear surgery is a bit more prolonged. Pain is not usually a significant component of the postoperative course; vertigo, dizziness, nausea, and vomiting are the major concerns. In the recovery room, precautions of positioning and medication are taken to minimize postoperative vertigo, nausea, and vomiting, as well as pain. Meclizine, scopolamine, and even diazepam are used with benefit for vertigo. Ondansetron is very effective for nausea and vomiting.

For the first week after surgery, the patient is advised to avoid increasing head/ear pressure, and is given prescriptions for meclizine, scopolamine skin patch, pain medications and antibiotics where indicated. Infrequently patients require orally disintegrating ondansetron to treat intractable postoperative nausea; however, it is very effective when needed. The advantage of the transdermal scopolamine administration is that the medicine takes effect for 72 h and does not depend on gastrointestinal absorption. The bothersome side effects are those of all belladonna alkaloids, including dilated pupils with blurry vision, dry mouth, and sedating effects.

Injection of lidocaine with epinephrine is routinely performed by the surgeon at the beginning of the case, into the external auditory meatus and canal. There are fissures (of Santorini) in the anterior bony external auditory canal. If the injection is deep anteriorly, the patient can wake up with transient facial nerve palsy. This will generally recover completely within 2 h but can be disconcerting for patients and post anesthesia care unit (PACU) nurses. Driving is permitted after 7 days postoperatively if there is no further vertigo or dizziness. The patient is advised to continue exercising caution in terms of aerobic exercise and head/ear pressure for a full month postoperatively.

Initial hearing improvement is often identified by the patient within 2–4 weeks postoperatively, depending on the type of chronic ear surgery performed. The first audiogram is performed at 6 weeks postoperatively, and the final hearing improvement is evaluated at a 4-month audiogram. These are general guidelines; interpersonal and regional variation is common.

If surgery was performed for amelioration of vertigo, significant improvement may be seen within the first 2–4 weeks, but it may take up to 3–4 months for more chronic disabling dizziness to improve. Continuing to follow and support the patient and their family (medically and emotionally) during this time is crucial.

Anesthesiologist's Perspective

In the PACU following middle ear surgery the most common problems encountered by patients are nausea, vomiting, and vertigo. Although pain management should always be a consideration in the PACU, it tends to be less of an issue for most patients recovering from ear surgery. The most painful experience for patients undergoing middle ear surgery is encountered during the intraoperative period. Despite all of this, the postoperative care in the PACU must be tailored to the individual needs of the patient. The management may include hydration, drug therapy, gradual elevation of the head of the bed, a quiet location and sometimes even some reassurance by the anesthesia provider.

Conclusion

There is no doubt that otologic procedures present a unique set of challenges for the anesthesiologist and surgeon. One method to help improve this situation is through a direct line of communication between the anesthesia provider, surgeon, and other team members involved with the care of the patient. In addition, the knowledge, understanding, and attention to the specific needs of this group of patients are essential for a successful outcome.

Clinical Insights

For the Otolaryngologist (from the Anesthesiologist)

- N₂O is not the only anesthetic agent that can have an effect on middle ear pressure. Desflurane and sevoflurane may increase middle ear pressure. Studies in this area are limited, and further investigation is necessary.
- NMB agents will be avoided, therefore communication with the anesthesiologist is critical prior to initiating painfull stimuli (i.e., Electrode placement of FNM, local injections, surgery)

For the Anesthesiologist (from the Otolaryngologist)

- Middle ear volume is an important consideration in otologic surgery. Avoidance of agents that increase or decrease ME volume, as well as avoidance of immediate postoperative bucking, coughing, and PONV that changes ME volume are of paramount importance.
- Even where the facial nerve EMG monitor is not being used, it is vastly preferable in ear surgery that the patient not be paralyzed, so that even unanticipated FN issues can be recognized promptly.

Clinical Pearls

For the Otolaryngologist (from the Otolaryngologist)

- N₂O can be employed briefly to help elevate a retracted tympanic membrane, before M&T surgery or tympanoplasty. It must be turned off before the middle ear packing and tympanic membrane repair are done.
- Avoidance of bucking and coughing at emergence will be aided by care that is taken by the surgeon while cleaning the area and applying the mastoid dressing. It is important to avoid rubbing the neck vigorously to clean off any povidone/iodine, etc., or torquing the neck while applying the mastoid dressing as the patient is getting "lighter."

For the Anesthesiologist (from the Anesthesiologist)

- Plan strategies to turn 90–180° without disconnecting monitors.
- Middle ear pressure rises with N_2O administration and as it is reabsorbed negative pressure may develop in the middle ear cavity. Tympanic membrane rupture, nausea, vomiting, or serous otitis may result from use of N_2O .
- It is preferable not to use muscle relaxants when FNM is being utilized. At least 10–20% of the muscle twitch response must be present to identify the facial nerve.
- During MAC cases dexamethasone must be given slowly to avoid painful perineal and genital burning and itching

References

- Jellish W, Owen K, Edelstein S, et al. Standard anesthetic technique for middle ear surgical procedures: a comparison of desflurane and sevoflurane. Otolaryngol Head Neck Surg. 2005; 133:269–74.
- Keyhani S, Kleinman LC, Rothschild M, et al. Clinical characteristics of New York City children who received tympanostomy tubes in 2002. Pediatrics. 2008;121(1):e24–33.

- Bhattacharyya N. Ambulatory pediatric otolaryngologic procedures in the United States: characteristics and perioperative safety. Laryngoscope. 2010;120(4):821–5.
- Stenfeldt K, Hermansson A. Acute mastoiditis in southern Sweden: a study of occurrence and clinical course of acute mastoiditis before and after introduction of new treatment recommendations for AOM. Eur Arch Otorhinolaryngol. 2010;267(12):1855–61.
- Acevedo JL, Lander L, Shah UK, Shah RK. Existence of important variations in the United States in the treatment of pediatric mastoiditis. Arch Otolaryngol Head Neck Surg. 2009;135(1):28–32.
- Homøe P, Jensen RG, Brofeldt S. Acute mastoiditis in Greenland between 1994–2007. Rural Remote Health. 2010;10(2):1335. Epub 2010 Jun 18.
- Dornelles C, Costa SS, Meurer L, Schweiger C. Some considerations about acquired adult and pediatric cholesteatomas. Braz J Otorhinolaryngol. 2005;71(4):536–45.
- Sarkar S, Roychoudhury A, Roychaudhuri BK. Tympanoplasty in children. Eur Arch Otorhinolaryngol. 2009;266(5):627–33.
- Chandrasekhar SS, House JW, Devgan U. Pediatric tympanoplasty. A 10-year experience. Arch Otolaryngol Head Neck Surg. 1995;121(8):873–8.
- Brackmann DE, Shelton C, Arriaga M, editors. Otologic surgery. WB Saunders Publ. 2009, Philadelphia.
- Van de Heyning P, Punte AK. Electric acoustic stimulation: a new era in prosthetic hearing rehabilitation. Adv Otorhinolaryngol. 2010;67:1–5. Epub 2009 Nov 25 [Review; PMID: 19955716].
- Chandrasekhar SS, De la Cruz A, Garrido E. Surgery of congenital aural atresia. Am J Otol. 1995;16(6):713–7.
- Megerian C, Reily J, O'Connell F, et al. Outpatient tympanomastoidectomy. Arch Otolaryngol Head Neck Surg. 2000;126:1345–8.
- Maurer W, Borkowski R, Parker B. Quality and resource utilization in managing preoperative evaluation. Anesthesiology Clin N Am. 2004;22:155–75.
- Nader ND, Simpson G, Reedy RL. Middle ear pressure changes after nitrous oxide anesthesia and its effect on postoperative nausea and vomiting. Laryngoscope. 2004;114(5):883–6.
- Wiet R. Iatrogenic facial paralysis. Otolaryngol Clin North Am. 1982;15:773–80.
- Jellish W, Leonetti J, Murdoch J. Propofol-based anesthesia as compared with standard anesthetic techniques for middle ear surgery. J Clin Anesth. 1995;7:292–6.
- Stoelting R, Hillier S. Pharmacokinetics and pharmacodynamics of injected and inhaled drugs. In: Pharmacology & physiology in anesthetic practice, 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2006. p. 3–41.
- Le T, Gan TJ. Update on the management of postoperative nausea and vomiting and postdischarge nausea and vomiting in ambulatory surgery. Anesthesiol Clin N Am. 2010;28:225–49.
- Jellish W, Leonetti J, Avramov A, et al. Remifentanil-based anesthesia versus a propofol technique for otologic surgical procedures. Otolaryngol Head Neck Surg. 2000;122:222–7.
- Firat Y, Selimoglu E. Dexmedetomidine: a novel anesthetic agent for middle ear surgery. Recent Pat CNS Drug Discov. 2007;2: 151–4.
- Ayala M, Sanderson A, Marks R, et al. Laryngeal mask airway use in otologic surgery. Otol Neurotol. 2009;30:599–601.
- Honkavaara P, Saarnivaara L, Klemola U. Prevention of nausea and vomiting with transdermal hyoscine in adults after middle ear surgery during general anesthesia. Br J Anaesth. 1994;73:763–6.
- Fujii Y. Clinical strategies for preventing postoperative nausea and vomiting after middle ear surgery in adult patients. Curr Drug Saf. 2008;3:230–9.

Facial Plastic Surgery

Adam I. Levine, Samuel DeMaria, Jr., Eunice Park, and William Lawson

Introduction

Americans spend nearly 11 billion dollars annually on cosmetic procedures. Of that total, almost 6.6 billion dollars is spent on surgical procedures ^[1]. Beauty is often characterized by a combination of factors that involve symmetry and aesthetically pleasing proportions and relationships. An understanding of the proportions, angles, measurements, and relationships that are considered the standards for the attractive face give the surgeon an appreciation for what is askew when a facial characteristic is outside the considered normal range and the patient is dissatisfied with his or her appearance.

Overview

Rhinoplasty surgery is the sixth most popular cosmetic procedure overall, the second most popular cosmetic surgery in men and the most requested aesthetic surgical procedure by teenagers. Over 130,000 rhinoplasties are performed annually which represents approximately 1/10 of all cosmetic surgeries performed in the USA ^[1]. In 2009 over 58% of the cosmetic procedures were performed in office-based facilities, 23% in freestanding surgi-centers and 18% in hospitals ^[2]. Successful rhinoplasty depends on careful analysis of both the nose and the surrounding facial features.

Rhinoplasty has been described as one of the most challenging procedures in plastic surgery. Gustave Aufricht once said, "Rhinoplasty is an easy operation, it is just difficult to get good results" ^[3]. There is no single rhinoplasty operation, and for that reason the learning curve to perfect the technique is a long and steep one. Each operation is unique in that it must be tailored to the specific anatomic components involved and the desires of the patient. By developing a consistent, meticulous routine in which the patient's nose is analyzed with regard to its anatomic components and their complex interrelationships, the surgeon can best select the appropriate incision, approaches, and techniques ^[4].

The nose plays a functional role in nasal breathing and an aesthetic role because it represents the most prominent and central facial feature. It is indisputable that the nose has enormous psychological, emotional, social, and symbolic importance. Although rhinoplasty can be a satisfying procedure for the patient and the surgeon, the literature reports an incidence of postoperative rhinoplasty complications ranging from 8% to 15% ^[5–8]. The rhinoplasty surgeon must take great care to minimize the incidence of functional and cosmetic complications.

Ultimately, success in rhinoplasty is based on welldeveloped judgment, wisdom, and accumulated knowledge and experience. Similar to most surgeries, rhinoplasty is a science and an art. Skill comes from experience and wisdom, combined with a measure of talent. The surgeon must have a detailed understanding of the multiple anatomic variants encountered. The surgeon also must have accumulated the appropriate surgical techniques and experience. Specifically, the surgeon must acquire knowledge of the surgical alternatives, and how healing forces affect the result. This skill set is acquired by careful follow-up of operated patients over time.

Although one's definition of beauty is subjective, the anesthetic management of patients presenting for cosmetic facial plastic surgery must be objective and evidence based.

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Patients presenting for elective cosmetic surgery are also consenting to undergo "elective" anesthesia. Patients expect that the anesthetic management will be practically "transparent," that is, incredibly safe with limited residual effects. While patients presenting for elective cosmetic surgery will generally be in excellent health, the anesthetic management that optimizes both intraoperative and postoperative conditions is challenging and must be well planned and grounded in physiology and pharmacology.

This chapter will highlight some of the more fundamental surgical maneuvers and anesthetic concepts in basic rhinoplasty and nasal fracture surgery to serve as a basis on which one can build. Full coverage of the entire range of rhinoplasty principles and procedures is beyond the scope of this chapter and much of the anesthetic considerations are similar to those for sinus surgery (Chap. 13). Instead, this chapter will focus on rhinoplasty fundamentals and the close interplay between the anesthesia and surgical teams when operating on a shared airway.

Preoperative Considerations

Otolaryngologist's Perspective

When the patient arrives at the office, he or she is asked to fill out a detailed history form including their past medical, surgical, and anesthetic history, including a list of current medications. Patients who are on aspirin or other medications (e.g., herbal medications such as echinacea, garlic, ginger, ginko biloba, kava kava) that may disturb normal platelet function are informed that these medications should be stopped 2 weeks before the surgery date, assuming it is safe to do so. Also, a history of illicit drugs must be determined during the visit. Patients who smoke are asked to avoid smoking and using other nicotine-containing products for at least 4 weeks before surgery because the effects on wound healing are well documented ^[9].

Detailed anatomic analysis of the nose is an essential initial step in achieving a successful surgical outcome ^[10, 11]. Standardized digital photographs from the frontal, base, lateral, and oblique views are taken (see Figs. 12.1 and 12.2). Care is taken to obtain photographs along the Frankfort horizontal that extends from the top of the tragus to the infraorbital rim. Photographic documentation is helpful both as an accurate reminder of the preoperative state and to support the claim for future surgery should that be necessary.

While reviewing the photographic images, it is critical to review the goals of surgery and ensure that the issues the patient would like to address have not changed. During the





Fig. 12.1 Five standard views for preoperative and postoperative photography used for most facial plastic surgery procedures. It is important to use the Frankfort horizontal line, which extends from the top of the tragus to the infraorbital rim, as a reference to ensure proper head positioning (**a**) anteroposterior (AP); (**b**) left oblique; (**c**) left lateral; (**d**) right oblique; (**e**) right lateral. (Reprinted from Facial Plast Surg Clin North Am, 18(2), Swamy RS, Most SP. Pre- and postoperative portrait photography: Standardized photos for various procedures, pp. 245–52.)

12 Facial Plastic Surgery

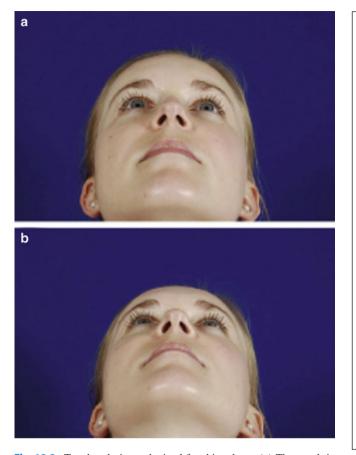


Fig. 12.2 Two basal views obtained for rhinoplasty. (**a**) The nasal tip is aligned with the medial canthi. This view can help with assessing any curvature in the dorsum and its relationship to the alar-columellar complex. (**b**) Aligning the nasal tip to the glabella allows for isolated photodocumentation of the alar-columellar complex. (Reprinted from Facial Plast Surg Clin North Am, 18(2), Swamy RS, Most SP. Pre- and postoperative portrait photography: Standardized photos for various procedures, pp. 245–52.)

preoperative evaluation, the surgeon must possess a mental image of the potential outcome and the surgical limitations inherent in every individual case. In essence the operation is rehearsed as the preoperative evaluation proceeds.

Analysis begins by examining all five views and making an assessment of the overall stature of the patient, the facial skin quality, and the symmetry of the face. The quality of the skin-soft tissue envelope—its thickness, integrity, and mobility in relation to the underlying nasal structures—must be determined because it plays a crucial role in dictating the limitations of what can and cannot be accomplished with aesthetic nasal surgery. After completing the general assessment, the surgeon reviews each photographic image and the major aesthetic and technical points are noted (see Fig. 12.3).

After the surgical goals are reviewed and the plan is mutually understood between the patient and the surgeon, the patient is given a standardized medical procedure informed consent form. Informed consent should include the risks of bleeding, intranasal scarring, worsening of the nasal airway, palpable or visible irregularities, asymmetry, failure to meet

Nasal analysis

General

Skin quality: integrity, vascularity, mobility, skin thickness (thin, medium, or thick)

Identify primary concerns leading patient to seek rhinoplasty (eg, "big," "twisted," "large hump")

Frontal view

Twisted or straight: follow brow-tip esthetic lines Width: narrow, wide, normal, "wide-narrow-wide" Tip: deviated, bulbous, asymmetric, amorphous, other

Base view

commentary.

Triangularity: good versus trapezoidal Tip: deviated, wide, bulbous, bifid, asymmetric Base: wide, narrow, or normal; inspect for caudal septal deflection Columella: columellar-to-lobule ratio (normal is 2:1 ratio); status of medial crural footplates Lateral view Nasofrontal angle: shallow or deep Nasal starting point: high or low Dorsum: straight, concavity, or convexity-bony, bony-cartilaginous or cartilaginous (ie, is convexity primarily bony, cartilaginous, or both) Nasal length: normal, short, long Tip projection: normal, decreased, or increased Alar-columellar relationship: normal or abnormal Nasolabial angle: obtuse or acute **Oblique view** Does it add anything, or does it confirm the other views There are many other points of analysis that can be made on each view, but these are some of the vital points of

Fig. 12.3 Guide to nasal analysis. (Reprinted from Otolaryngol Clin North Am, 39(3), Becker DG, Becker SS, Reducing complications in rhinoplasty, pp. 475–92, Copyright 2006, with permission from Elsevier.)

patient expectations, and the possible need for future revision surgery.

Surgery may be performed under local anesthesia, intravenous sedation, or general anesthesia, depending on patient and surgeon preference. The latter two options are generally preferred.

Anesthesiologist's Perspective

Although it is assumed that all patients presenting for elective cosmetic surgery are in excellent health, all patients must have a preoperative history and physical as defined in the Practice Advisory for Preanesthesia Evaluation published by the ASA ^[12]. Please refer to Chap. 7, for a detailed discussion on preoperative assessment. Most patients presenting for elective cosmetic surgery will be ASA classification 1 or 2, however, patients with more serious systemic illnesses are not precluded from having elective surgery. Patients, though often

medically well, may be taking home remedies or recreational drugs and this must be determined. In these situations it is absolutely necessary to assure that the patient is stable and medically optimized and has received counseling about the risks and benefits of proceeding with elective surgery. Proper documentation of informed consent is always required, but is very important in this situation. The majority of cosmetic procedures are done in an office setting ^[2]. Patients presenting for their cosmetic surgery in an office setting must have a full and complete preoperative evaluation, but must also be assessed and determined to be acceptable candidates for an office-based anesthetic (OBA) and surgery. The assessment of patients for OBA is discussed in detail in Chap. 21.

In the past the geriatric patient was considered a poor risk for good cosmetic outcomes due to a lack of tissue elasticity and poor wound healing. However, recent articles have suggested otherwise, and older patients may be presenting for cosmetic surgery at an accelerated rate ^[13, 14]. Due to the popularity of rhinoplasty amongst the pediatric and adolescent patient populations ^[15], the anesthesiologist must also be knowledgeable and comfortable assessing and caring for patients in these age groups (Chap. 20).

Intraoperative Considerations

Otolaryngologist's Perspective

Surgical Approaches

There are basically two approaches to the nose: endonasal and external. The most important determinant here is the physician comfort level and experience. In general more typical indications for an external approach would include complicated revision surgery, unclear anatomy, severe asymmetry, the need to secure grafts, the crooked nose, and the cleft lip rhinoplasty.

We employ primarily the endonasal approach at our institution. In all cases, the nose is also injected at the time of surgery with 10–15 ml of 1% lidocaine with 1:100,000 epinephrine for additional hemostasis, and decongested with either oxymetazoline or 4% cocaine (maximum of 5 ml) soaked pledgets. The minimum amount of local anesthetic to facilitate hemostasis without overly distorting the nasal appearance is recommended. Access to the dorsum is obtained by elevating the overlying skin and soft tissue envelope through bilateral intercartilaginous (IC) incisions (at the junction of the upper and lower lateral cartilages) that are connected to a full transfixion incision at the caudal septum. Midline dissection should remain in the avascular plane intimate to the perichondrium of the dorsal septum and deep to the periosteum of the nasal bones. Elevate the skin and soft tissue envelope only as much as is necessary for good exposure to facilitate reduction or augmentation, because these periosteal and soft tissue attachments to the nasal sidewall help to provide support and protect from excessive medial collapse of the nasal bones. Figure 12.4 shows a simplified anatomical schema of the nasal architecture.

Access to the nasal tip can be via IC incisions with retrograde dissection or transcartilaginous (splitting the lateral curs of the lower lateral cartilage (LLC)) incision, but both of these imply more limited exposure/visibility and thus a higher risk of asymmetry. A third (and preferred) option if any significant tip surgery is planned endonasally is using an alar delivery approach, whereby the LLC is delivered or pivoted inferiorly through the nostril as a bipedicled chondrocutaneous flap hinged medially and laterally by combining IC incisions with a transfixion incision along the caudal septum (above the LLCs), and marginal incisions along the lower border of the LLC on each side. When bilateral nasal tip adjustment is necessary, both lower lateral cartilages are delivered to one side and any dissection is completed.

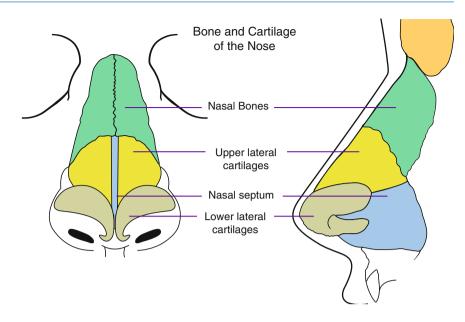
An IC incision is made by everting the alar rim with a wide two prong hook, at which point the caudal border of the Upper Lateral Cartilages (ULC) is apparent as a shelf. A no. 11 blade is then used to make an incision between the ULC and the overlying skin, parallel to the plane of the ULC, between the cephalic margin of the LLC and the caudal border of the ULC. This incision is then carried medially over the dorsal cartilaginous septum and around the anterior septal angle to connect with a transfixion incision along the caudal septum.

The marginal incision is made by everting the alar rim and using a no. 15 blade to incise vestibular skin at the caudal border of the lower lateral carilage, carefully following that border from lateral to medial, beneath the dome of the LLC and along the columella, taking care not to incise the caudal border of the medial crura in so doing. The overlying skin and soft tissue are then dissected from the LLC, which can be pivoted or "delivered" inferiorly by retracting the LLC downward with a small hook.

Osteotomies

Osteotomy is a surgical operation where bone is cut to shorten, lengthen, or change its alignment. Osteotomies are used in rhinoplasty surgery to narrow the nose, to correct the alignment of the nasal pyramid, and to close the open roof deformity that naturally results from a reduction of the bony dorsum. In the latter case, if it were not for the infracture of the nasal bones, the nasal dorsum would appear wide and flattened on frontal view. Complete osteotomies will mobilize and medialize the bony nasal sidewall together with the attached ULCs. Osteotomies progress from low to high (when indicated); medial and lateral osteotomies complete the infracture of the nasal bones.

Fig. 12.4 Basic anatomy of the nasal bones and cartilages



Medial Osteotomies

Several kinds of osteotomes are available, and the choice will vary with surgeon preference. These may range from 2 to 4 mm straight osteotomes to a 4–6 mm curved osteotome. Smaller osteotomes create less tissue trauma but require more skill to use. The osteotome is positioned at the cephalic edge of the bony hump removal (at the apex of the open roof), alongside the septum in a parasagittal plane. From this point, the mallet advances the osteotome obliquely, $15-20^{\circ}$ from the parasagittal plane, upward to the level of the nasof-rontal angle. A similar maneuver is performed on both sides of the septum.

If any kind of grafting is planned for the middle third of the nose it is best to perform the medial oblique osteotomy before graft placement so as not to disrupt the stable position of the cartilage graft in the process of performing the osteotomies.

Lateral Osteotomies

To help minimize the potential infraorbital swelling and bruising, lateral osteotomies are generally reserved for one of the final maneuvers in rhinoplasty, prior to any final adjustments and application of splints and dressings. Again, several kinds of osteotomes (straight, curved, guarded, or nonguarded 2–6 mm) are available, with the ultimate choice by surgeon preference. As with medial osteotomies, narrower osteotomes create less tissue trauma in creating lateral osteotomies but require more experience to use effectively.

Lateral osteotomies are performed through incisions at the lateral pyriform aperture. Just above the attachment of the inferior turbinate to the lateral nasal wall. It can be helpful to elevate the periosteum and soft tissue along the proposed path of the lateral bone cut prior to actually executing the osteotomy to help minimize bleeding. This is done with a narrow periosteal elevator through the incision at the pyriform.

The osteotome is firmly sited at the lateral pyriform aperture. Using a mallet, the osteotome is initially directed laterally (as though directed toward the lateral canthus of the eye) to engage the bone of the maxilla. From here it is advanced cephalically, taking care to stay lower in the nasofacial groove so as to avoid a step deformity along the lateral nasal wall. If a guarded osteotome is used, the surgeon can use his or her non-dominant hand to palpate the guard on the lateral edge of the osteotome through the overlying soft tissues, to precisely follow the position of the osteotome. At about the level of the infraorbital rim, the surgeon begins to curve the osteotome slightly up the lateral nasal wall to intersect with the medial oblique osteotomy at about the level of the medial canthus. At this point, the osteotome can be rotated medially to complete the fracture of the lateral nasal wall and medialize and narrow the nasal bones. Figure 12.5 demonstrates the pathway for the lateral osteotomy.

After completion of the procedure, the dressings are placed. A classic nasal packing is placed using vaselinated gauze. If complex septal or turbinate work was performed, then silastic splints may be placed and secured intranasally. Telfa gauze is used to create a cushion for the tape and casting material. The nose is then taped with 0.5 in wide surgical paper tape. Then followed by 0.5 in wide silk tape. The cast material is cut to the appropriate size and then placed in hot water. The cast is molded over the nose and after it sets, silk tape is then placed over the casting material.

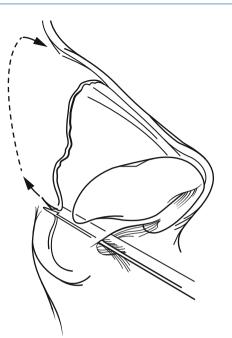


Fig. 12.5 The *dotted line* demonstrates the path of the osteotome during a lateral osteotomy

Communication between the surgeon and the anesthesiologist at the end point of the operation is critical to ensure a smooth emergence. The oropharynx is gently suctioned with a Yankauer suction catheter. As the anesthetic is titrated off and the patient becomes more aroused, the tube is removed. The anesthesiologist is cognizant of the importance of awakening the patient quietly and smoothly and the avoidance of traumatic disruption of the surgical site. The surgeon is present for the emergence and ensures that the patient is safe before moving to the recovery room. It can be reassuring for the patient to see the surgeon in this immediate postoperative period and hear words of encouragement regarding the surgery.

Anesthesiologist's Perspective

General Concepts and Patient Positioning

Although both regional anesthesia with sedation ^[16–18] and general anesthesia are acceptable anesthetic techniques, our group prefers general anesthesia for the majority of nasal cosmetic cases because of the benefit of airway protection, patient comfort, and the ability to use pharmacologic agents to improve intraoperative surgical conditions. Patients may be positioned with their head toward the anesthesiologist or at 90° away from the anesthesiologist. As with most otolaryngologic procedures, intravenous patency and appropriately functioning monitors must be confirmed after final positioning and draping, but prior to surgical start. At our institution



Fig. 12.6 Patient positioned on foam head support, endotracheal tube midline and taped to chin

the patient remains with their head near the anesthesiologist, but we move the entire operating room table toward the patient's feet. This gives additional room to both the anesthesiology and otolaryngologic team while adding the ability for the surgical team to move to the head of the bed to gain perspective on nasal symmetry. Figures 12.6, 12.7, and 12.8 depict patient positioning and room organization for a septorhinoplasty.

Optimizing Surgical Conditions

The goals and objectives to optimize operative surgical conditions by minimizing intranasal bleeding for nasal surgery including septorhinoplasty are identical to those used for Functional Endoscopic Sinus Surgery (FESS). See Chap. 13 for a detailed discussion of the theory behind and the intraoperative anesthetic management of sinus endoscopy surgery. Briefly, the objective for the anesthesiologist is to minimize nasal mucosal bleeding through a variety of anesthetic management options. The reduction of bleeding will facilitate surgical visualization and reduce the formation of nasal and subcutaneous septal hematomas. Hematomas can distort the anatomy and compromise the intraoperative surgical management. Postoperatively, septal hematomas can cause deep tissue fibrosis, leading to scarring and contour deformities, affecting the final nasal appearance ^[19].

Although the term "deliberate hypotension" is used it is inaccurate and in fact may encourage the use of agents shown to cause deterioration in the surgical field. In general, the anesthetic goal should be to achieve a reduction in cardiac output (CO) while preserving systemic vascular resistance (SVR). From here on we will use the term "deliberate cardiac reduction." Reducing the blood pressure via vasodilatation **Fig. 12.7** Operating room organization with the patient away from the anesthesia workstation and an open area cephalad for surgical staff



Fig. 12.8 The surgical team has access to the top of the operating room table when the room is properly arranged



with isoflurane ^[20], sevoflurane ^[21], desflurane, sodium nitroprusside ^[22], nitroglycerine, or hydralazine does not improve surgical conditions and may in fact create a worsened state ^[23]. This is in comparison with the reduction of blood pressure by cardiac output depression with beta-blockade therapy ^[23] or with the use of high-dose remifentanil ^[24, 25] and its resulting bradycardia from a sympathectomy and high parasympathetic tone.

Anesthetic Management

After the induction of general anesthesia using propofol (1–2 mg/kg) and high-dose remifentanil (5mcg/kg), we achieve optimal intraoperative conditions using a maintenance anesthetic with a nitrous oxide and remifentanil (0.05–0.5 mcg/ kg/min)^[26] technique supplemented with a low-dose infusion of propofol (25–40 mcg/kg/min). Generally we use the rule of



Fig. 12.9 The placement of a trans-nasal infraorbital nerve block and the resulting affect on nasal anatomy

100 to guide our maintenance anesthetic. We set the inspired percentage of nitrous oxide plus the propofol infusion in mcg/ kg/min to equal 100. For example if 70% nitrous oxide is used, then a propofol infusion of 30 mcg/kg/min would be administered. A nitrous oxide-narcotic technique is used to avoid the delayed emergence associated with the accumulation of propofol from a high-dose propofol technique during total intravenous anesthesia (TIVA). Interestingly, a recent study demonstrated that the administration of nitrous oxide during septorhinoplasty surgery decreased the incidence of opioid-induced hyperalgesia from remifentanil^[27]. Unless contraindicated, intraoperative administration of beta-blockade is utilized to achieve a target heart rate of 60 or lower. Airway management is achieved with either an appropriately sized RAE tube or a wire reinforced anode tube secured with tape to the chin. After securing the airway, our team also employs the use of bilateral sphenopalatine ganglion blocks using 1.5 ml of 1% lidocaine with epinephrine 1:100,000 for vasoconstriction as described in Chap. 13. Infraorbital nerve blocks (2 ml of 0.5% bupivacaine) are placed bilaterally at the end of the procedure for postoperative analgesia. When using these blocks for cosmetic procedures, we recommend placement at the end of the procedure because the deposit of local anesthesia between the nasal fold and the infraorbital notch could distort the nasal anatomy and may compromise the surgical outcome (Fig. 12.9a-c). When placing this block it is important to indentify the infraorbital notch with an index finger and maintain that finger in position during the block. The finger helps guide the needle tip while protecting the globe of the eye and blocking the needle tip from entering the notch itself. Ocular injury can occur during infraorbital nerve block presumed to be caused by the needle traversing the infraorbital notch and penetrating the globe of the eye [28].

All patients receive appropriate prophylactic antibiotics (first-generation cephalosporin or clindamycin for those with allergies to cephalosporins or penicillins) and at least double prophylactic antiemetic therapy with dexamethasone (also effective to reduce postoperative swelling ^[29]) and ondanse-tron. Despite the FDA black box warning, many will receive triple therapy with the addition of a small dose (0.625 mg) of droperidol soon after induction affording adequate time to monitor QT intervals intraoperatively and postoperatively. We generally omit neuromuscular blocking agents and

therefore eliminate the need for reversal and its concomitant effect on postoperative nausea and vomiting.

Intraoperative Events

It is critical that the anesthesiologist understands and anticipates the steps associated with rhinoplasty surgery in order to maintain homeostasis, hemodynamic stability, and idealized operative conditions. Soon after the anesthetic induction the nasal mucosa will be prepped with at least one vasoconstrictive agent. These agents include phenylephrine (0.25%), oxymetazoline or cocaine 4%. Each can be absorbed and has systemic effects of hypertension and tachycardia. Just before incision the surgeon will generally inject 10-15 ml of 1% lidocaine with epinephrine 1:100,000 intranasally. This solution is selected for intraoperative analgesia (lidocaine)^[30] and nasal mucosa vasoconstriction (epinephrine). The epinephrine concentration in this solution is 10 mcg/ml; therefore, the total dose of epinephrine can be quite significant (100-150 mcg). Unless there are no contraindications like asthma or COPD, a dose of labetalol (5-50 mg) is administered prior to injection to attempt to ameliorate the systemic effects of the injected/absorbed epinephrine. Labetalol is selected preferentially over esmolol or metoprolol due to its mixed alpha and beta-receptor blockade.

Intraoperative Bleeding

Intraoperatively it is important for the anesthesiologist to continually observe for frank bleeding from the nostrils and adjust the intraoperative management accordingly. It is important to appreciate that osteotomies create significant bleeding. At our institution the surgeons communicate to the anesthesiologists just prior to osteotomy. It has been our observation that deepening the anesthetic with the administration of an additional bolus of remifentanil (2–5 mcg/kg) 60–90 s prior to the osteotomy reduces bleeding. Occasionally, vasodilation may be beneficial when the nasal bone is fractured during osteotomies. Nitroglycerin 40–80 mcg will provide rapid predictable venodilation that is short in duration should the desired effect not be achieved.

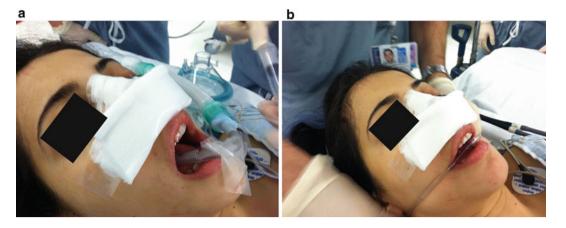


Fig. 12.10 (a) The patient emerges, awake, alert, and cooperative following instructions. (b) Nasal cannula are placed in the mouth for supplemental oxygen

Cartilage Graft Harvest

Occasionally a cartilage graft will be used for the reconstruction, especially during reoperations. The cartilage will be harvested locally from the nasal septum or from the auricular cartilage of the ear. In the latter situation it is critical to assure the intended surgical ear is identified and initialed preoperatively. Auricular graft harvest generally takes 45–60 min and precedes the nasal portion of the surgery. During the harvest aggressive hemodynamic control for bleeding reduction is unnecessary. The patient's head will be positioned on a donut and turned nearly 75–90° away from the operative ear. In this situation, because of the time lag, the placement of the infraorbital blocks immediately after intubation is acceptable given the fact that there will be adequate time for the local anesthesia to reabsorb and will therefore not interfere with the cosmetic portion of the surgery.

Emergence

In order to wake the patient up lucid, pain free, breathing spontaneously while not bucking on the endotracheal tube, we emerge the patient on a low-dose remifentanil infusion ^[31, 32]. Nasal cast placement signals an appropriate time to start reducing the remifentanil infusion to awake levels (0.05–0.1 mcg/kg/min). Generally the cast placement takes approximately 5–10 min allowing enough time (2–3 context sensitive half-lives) for remifentanil to be metabolized to plasma/receptor levels that imparts analgesia without significant respiratory depression (2.5 ng/ml plasma level or less) ^[33]. We normally maintain the nitrous oxide and propofol until the throat pack is removed and the oropharynx and stomach are suctioned. Both of these procedures are stimulating and might instigate bucking if the anesthetic is titrated prematurely. Once all stimulation is over, including wiping

the patient's face from residual blood or prep solutions, the nitrous oxide and the propofol infusion are discontinued. If the patient does not emerge spontaneously once the end-tidal nitrous oxide falls below 10%, the remifentanil infusion is stopped and the infusion line disconnected from the patient to avoid an inadvertent remifentanil bolus. Typically within 30–60 seconds of remifentanil termination the patient will spontaneously open their eyes, follow commands and breathe when asked (Fig. 12.10a, b). At this time the patient is extubated. After extubation the patient is reminded to take deep breaths. Mild hypoventilation from residual remifentanil will cause the rapid re-accumulation of alveolar nitrous oxide and the patient could become reanesthetized and apneic.

If osteotomies were created and a nasal cast was placed it is critical to avoid distorting the cast during the immediate extubation and postoperative period. To a large extent cast integrity determines the end result of the rhinoplasty. Care should be taken not to apply pressure or pinch the bridge of the nose with a non-rebreathing mask. In addition extubation should be timely, but should occur after the patient is awake, breathing spontaneously, and following commands. Although absolutely necessary, delivering positive pressure ventilation during a post extubation apneic episode will likely disrupt the cast. When possible we use a smaller child mask or an adult mask turned sideways to ventilate the patient only through the mouth (Fig. 12.11). Due to intraoral bleeding and secretions, post extubation laryngospasm with resulting negative pressure pulmonary edema is a risk after nasal surgery ^[34].

Monitored Anesthetic Care

Although our team prefers to employ general anesthesia for nasal cosmetic surgery, regional or local anesthesia with sedation or monitored anesthetic care (MAC) is entirely



Fig. 12.11 After extubation one can turn the mask sideways to delivery positive pressure ventilation without applying pressure or disrupting the nasal cast

acceptable for rhinoplasty surgery and may be the desired anesthetic technique for a variety of other facial plastic procedures including scar revisions, blepharoplasty surgery, and limited myocutaneous flaps. It is critical, however, that every anesthesiologist and otolaryngologist appreciate that head and neck surgeries performed under MAC are at an increased risk for initiating operating room fires. This will be discussed in detail later in the chapter.

Patient Education and Selection

As with all cases preformed under MAC, patient education, motivation and selection is critical when this anesthetic option is offered. This is especially important in facial plastics cases given the proximity of the surgical site to the face and airway. Preoperatively patients must be educated and have a clear understanding of the surgical procedure and a clear understanding of the anesthetic expectations. Patients presenting for and requesting MAC for their facial surgery must understand that due to the proximity of the surgery to the airway, that minimal sedation will be employed to assure the maintenance of their cooperation, airway, and oxygenation. With this knowledge many patients may opt for general anesthesia.

Patient selection is also paramount. They must be mature enough to cooperate, and they must be free of mental illness, anxiety disorders, or claustrophobia. The ability to communicate with the patient intraoperatively is also critical, therefore there can be no language barriers between providers and patients undergoing head and neck surgery under MAC.

Cooperation

When contemplating performing facial plastics cases under MAC, it is critical that the anesthetic and surgical teams also have superb communication and cooperation and that everyone has a clear understanding of surgical and anesthetic expectations. Because the patient must remain cooperative throughout the procedure while maintaining a patent airway requiring minimal supplemental oxygen, the surgical team must be informed that heavy sedation will be avoided. If the surgical team is not confident that they will be able to accomplish the planned surgery given these conditions, then general anesthesia should be employed.

Head and Neck Surgery and Fires

Due to the use of open oxygen delivery systems like the nasal cannula, head and neck surgery under MAC imparts a heightened risk for operating room fires. In a recent survey of otolaryngologists, 25% have witnessed an OR fire during their careers; 81% of the fires occurred while supplemental oxygen was in use ^[35]. Although lasers are frequently associated with OR fire, in this survey, the most common ignition source was an electrosurgical unit (59%). Lasers and light sources were the second and third most common ignition sources, 32% and 7%, respectfully. Before electrosurgical devices are used, it is critical that the otolaryngologic team communicate with the anesthesiology team, so that oxygen can be disrupted.

The key to preventing fires during head and neck surgery is to limit the oxygen concentration beneath the drapes, in the oropharynx, and in the vicinity of the surgical field. In order to accomplish this we attempt to perform these cases without supplemental oxygen, by using regional anesthesia, local anesthesia infiltration, and minimal sedation. If supplemental oxygen is necessary, then it must be assured that concentrated oxygen is not accumulating under the drapes. Preventing oxygen accumulation under the drapes should be adequate, but suction will be used when necessary ^[36, 37].

We recommend avoiding synergistic respiratory depression and only use one class of drug for sedation analgesia. Our group uses remifentanil at infusion doses of 0.05–0.1 mcg/kg/min. This dose imparts analgesia without profound respiratory depression (2.5 ng/ml plasma level or less). At this level, the patient is generally comfortable, cooperative, may need to be reminded to breath, but will do so with verbal reminding and require minimal supplemental oxygen to maintain saturation. Using concomitant midazo-lam will reduce the incidence of intraoperative pruritus and postoperative nausea and vomiting, but will increase respiratory depression and the need for supplemental oxygen [38, 39].

Antiemetic therapies are used including ondansetron and dexamethasone. During MAC cases care should be taken to administer dexamethasone after the administration of remifentanil ^[40] and to infuse it slowly, or the patient may experience a very unpleasant and potentially painful burning of the genitals, perineum, and peri-rectum ^[41]. When remifentanil is used as the sole anesthetic agent, we "fast track" our patients' discharge and by-pass the Post Anesthesia Care Unit (PACU).

Postoperative Considerations

Otolaryngologist's and Anesthesiologist's Perspectives

Immediate Postoperative Care

In the PACU attention should be on the patient's airway. Any mask placed on the patient for oxygenation should be done in such fashion as to not place pressure on the nose. The patient's head is elevated 30°, and cold compresses are gently placed over the eyes and nose. Care is taken to control postoperative pain, hypertension, and nausea. Vomiting and coughing causing the patient to Valsalva can result in venous drainage impediment and epistaxis or bleeding under the septal or nasal skin flaps. Epistaxis is one of the most common complications following rhinoplasty ^[42, 43]. Patients who experience some epistaxis may have a folded 4x4 sponge placed under their nose that is held in place by a surgical mask cut into a thin rectangular piece.

After the patient is more alert and has stable vitals, the patient is transferred to a secondary recovery unit and allowed to change into his or her clothes. Patients are encouraged to wear buttoned shirts so that they do not have to pull clothes over their head and risk displacing the nasal splint. Patients sit in a recliner at a 45° angle and are given clear liquids without carbonation if they are not nauseated. An intravenous line remains in place until discharge in the event that further antiemetic or pain medication is required.

Long-Term Postoperative Care

During the recovery process, the patient's caregivers are given postoperative instructions and contact numbers for the staff and the surgeon. The clinical nurse goes over every detail of the instructions and ensures that all questions are answered. It is most important to address postoperative pain because it is frequently the most common issue. For those who are most concerned, the authors recommend taking the pain medication as prescribed every 6 h for the first 24 h, and then taking it as needed after that. Patients are also instructed to avoid blowing their nose and to wipe the nose gently as needed. For minor postoperative bleeding, 0.05% oxymetazoline may be used sparingly for no more than 3 days. If the patient has continued bleeding, he or she should contact the surgeon immediately. The authors recommend that patients avoid nonsteroidal anti-inflammatory drugs because they affect platelet function and may lead to epistaxis. Patients are also told to avoid exerting themselves and to limit bending and lifting anything heaver than 10 lb. Restrictions are gradually lifted during the postoperative period as the patient recovers. Prior to discharge, the surgeon meets with the caregiver and the patient to go over the instructions and to address any concerns. Extra time spent in this critical period eases anxiety for the patient and the caregiver and assists in a quicker recovery. Patients are also given the time and date for their next postoperative visit.

It is made clear to the patient that should any issue arise, the surgeon or a member of the staff is available at all times. The first postoperative visit takes place between postoperative day 5 to day 7. The nasal splint is removed during this visit. Great care should be taken to gently remove the tape and splint over the nasal dorsum, which can be accomplished by first applying a generous amount of adhesive remover such as Detachol (Ferndale Laboratories, Ferndale, Michigan). After awaiting a few minutes, blunt dissection of the nasal skin from the overlying splint using a cotton-tipped applicator avoids any disruption between the skin-soft tissue envelope and the underlying framework. Patients will likely have a moderate amount of swelling and should be reminded that it can be almost a year before the final result is fully appreciated. Patients should avoid strenuous physical activity for two additional weeks and avoid heavy lifting (>10 lbs). Patients can expect to resume full activity with no restrictions at 6 weeks after surgery.

The smoothness of the postoperative course is directly proportional to the thoroughness of the preoperative preparation. The patient is instructed to begin pain medication and oral antibiotics. Head elevation and ice compresses over the eves for 36 h are recommended. The drip pad is changed as necessary. Meticulous cleaning of all suture lines two to three times a day with hydrogen peroxide and coverage with antibiotic ointment is stressed. The patient is seen 1 week later. On the morning that the cast is to be removed, the patient is instructed to take a shower and get the cast and nose wet. They are also told to take a pain medication 30 min before coming to the office. The cast is removed. When turbinates and complex septal surgery are done, the patient is encouraged to irrigate the nose with a generic salt water spray. The patient is seen 2 weeks later and then at regular intervals: 3, 6, and 12 months and then annually, thereafter. The usual

concerns include bruising, swelling, breathing, smiling, numbness, and initial appearance.

In the authors' practice, patients are seen in the postoperative period at 1 week, 1 month, 3 months, 6 months, and 1 year. Frequent postoperative visits provide positive reinforcement to recovering patients and help to identify those who may not be satisfied or who have an unexpected outcome.

Complications

Although serious acute life-threatening complications are extremely rare, the reported incidence of significant complications following rhinoplasty ranges from 1.7% to 18%. In 2006 an estimated 600,000 patients underwent ambulatory sinonasal procedures of these 134,000 were plastics cases. The unexpected admission rate was 2.65% and among the sample, there were no cases of cardiac arrest, malignant hyperthermia, or blood transfusion ^[44]. Common complications following rhinoplasty include bleeding, infection, nasal airway obstruction, and deformities.

Conclusion

The challenges of rhinoplasty are substantial because every patient is unique and achieving success is multifaceted. A surgeon must not only possess a comprehensive set of surgical skills but also be able to effectively communicate with the patient and understand his or her concerns and desires. Although the surgery itself poses considerable challenges and can be a major source of anxiety, the implementation of an efficient perioperative strategy that can be considered replicated can reduce trepidation and amplify success. This article describes the authors' approach to the preoperative, anesthetic, and postoperative care plan for patients undergoing rhinoplasty. There are many variations in perioperative planning, with no one correct strategy; however, all successful plans must foster open lines of communication and focus on the patient's overall well being.

Clinical Insights

For the Otolaryngologist (from the Anesthesiologist)

- The absolute value of the blood pressure is irrelevant with regard to intraoperative surgical conditions.
- Nasal mucosal bleeding is multifactorial and cannot be controlled or strictly under the influence of the anesthetic management.
- Regional anesthesia administered by the anesthesiologist can improve intraoperative conditions while facilitating postoperative analgesia.
- Head and neck surgeries under MAC carry a high risk for operating room fires.

For the Anesthesiologist (from the Otolaryngologist)

- The anesthesia should be maintained until the nasal cast is applied. Proper cast application alleviates postoperative edema and stabilizes osteotomy cuts.
- The endotracheal tube should be taped in a midline position to avoid facial distortion.
- A throat pack should be placed if general anesthesia is used to decrease amount of blood ingested and decrease risk of aspiration.
- After extubation the ability to mask ventilate the patient is not feasible in order to avoid pressure to the nose, risking disruption of the surgical result. This should be accounted for in the patient at risk of airway obstruction after extubation (i.e. large base of tongue)

Clinical Pearls

For the Otolaryngologist (from the Otolaryngologist)

- Preoperatively the patient should be medically optimized for elective surgery. The patient should be counseled on realistic expectations and outcomes.
- Don't be afraid to do closed rhinoplasties. Mentally visualize the anatomy through the skin.
- Do not distort the anatomy with excessive injection of anesthetic hemostatic solution.
- A carefully controlled medial osteotomy makes infracture and bone positioning easier.
- Recognize anatomical landmines (occult under projected tips, short nasal bones, weak middle vault, dorsal nasal deviation, malpositioned alar cartilages) and address them during surgery to avoid revisions.
- In addition to looking for nasal asymmetry, look for facial asymmetry, as you will not be correcting facial asymmetry during the rhinoplasty. Point out these issues preoperatively rather than trying to explain these limitations after surgery.

For the Anesthesiologist (from the Anesthesiologist)

- The absolute value of the blood pressure is irrelevant to improve surgical conditions.
- Strategies to reduce cardiac output are effective in improving surgical conditions.
- Avoid the use of inhaled anesthesia and vasodilators and use remiferitanil and beta blockers to improve intraoperative conditions.
- Head and neck surgeries under MAC carry a high risk for operating room fires.
- Use regional anesthesia to improve intraoperative conditions and post operative analgesia. Place ION blocks at the end of cosmetic nasal surgeries.
- The integrity of the nasal cast is critical to the surgical outcome, do not apply pressure to the cast after extubation.

References

- Cosmetic Plastic Surgery Research. http://www.cosmeticplasticsurgerystatistics.com/statistics.html. Accessed 12 Jan 2011.
- American Society for Aesthetic Plastic Surgery. http://www.surgery.org/. Accessed 12 Jan 2011.
- Adamson PA, Funk E. Nasal tip dynamics. Facial Plast Surg Clin North Am. 2009;17(1):29–40.
- 4. Becker DG, Becker SS. Reducing complications in rhinoplasty. Otolaryngol Clin North Am. 2006;39(3):475–92.
- Simons RL, Gallo JF. Rhinoplasty complications. Facial Plast Surg Clin N Am. 1994;2:521–9.
- Thomas JR, Tardy ME. Complications of rhinoplasty. Ear Nose Throat J. 1986;65:19–34.
- 7. Tardy ME. Rhinoplasty: the art and the science. Philadelphia: Saunders; 1997.
- McKinney P, Cook JQ. A critical evaluation of 200 rhinoiplasties. Ann Plast Surg. 1981;7:357.
- Monfriecola G, Riccio G, Savarese C. The acute effect of smoking on cutaneous microcirculation blood flow in habitual smokers and nonsmokers. Dermatology. 1998;197(2):115–8.
- Swamy RS, Most SP. Preoperative, anesthetic, and postoperative care for rhinoplasty patients. Facial Plast Surg Clin North Am. 2009;17:7–13.
- Economou PS, East CA. Perioperative settings in rhinoplasty. Facial Plast Surg. 2011;27(2):160–6.
- The American Society of Anesthesiologists Taskforce on Preparative Evaluation. Practice advisory for preanesthesia evaluation. Anesthesiology. 2002;96:485–96.
- Rohrich RJ, Hollier Jr LH, Janis JE, et al. Rhinoplasty with advancing age. Plast Reconstr Surg. 2004;114(7):1936–44.
- Quatela VC, Pearson JM. Management of the aging nose. Facial Plast Surg. 2009;25:215–21.
- 15. Shandilya M, Den Herder C, Dennis SC, et al. Pediatric rhinoplasty in an academic setting. Facial Plast Surg. 2007;23:245–57.
- Jourdy DN, Kacker A. Regional anesthesia for office-based procedures in otorhinolaryngology. Anesthesiol Clin. 2010;28:457–68.
- Rajapakse Y, Courtney M, Bialostocki A. Nasal fractures: a study comparing local and general anaesthesia techniques. ANZ J Surg. 2003;73:396–9.
- Molliex S, Navez M, Baylot D. Regional anaesthesia for outpatient nasal surgery. Br J Anaesth. 1996;76(1):151–3.
- Rohrich RJ. Ahmad rhinoplasty. J Plast Reconstr Surg. 2011; 128(2):49–73.
- Pavlin JD, Colley PS, Weymuller EA, van Norman G, Gunn HC, Koerschgen ME. Propofol versus isoflurane for endoscopic sinus surgery. Am J Otolaryngol. 1999;20:96–101.
- Ahn HJ, Chung SK, Dhong HJ, et al. Comparison of surgical conditions during propofol or sevoflurane anaesthesia for endoscopic sinus surgery. Br J Anaesth. 2008;100:50–4.
- 22. Jacobi KE, Boehm BE, Rickauer AJ, Jacobi C, Hemmerling TM. Moderate controlled hypotension with sodium nitroprusside does not improve surgical conditions or decrease blood loss in endoscopic sinus surgery. J Clin Anesth. 2000;12:202–7.
- Boezaart AP. Comparison of sodium nitroprusside-andesmololinduced controlled hypotension for functional endoscopic sinus surgery. Can J Anaesth. 1995;42:373–6.
- Manola M, De Luca E, Moscillo L, et al. Using remifentanil and sufentanil in functional endoscopic sinus surgery to improve surgical conditions. ORL J Otorhinolaryngol Relat Spec. 2005;67: 83–6.

- Eberhart LH, Folz BJ, Wulf H, et al. Intravenous anesthesia provides optimal surgical conditions during microscopic and endoscopic sinus surgery. Laryngoscope. 2003;113:1369–73.
- Coskun D, Celebi H, Karaca G, Karabiyik L. Remifentanil versus fentanyl compared in a target-controlled infusion of propofol anesthesia: quality of anesthesia and recovery profile. J Anesth. 2010;24:373–9.
- Echevarría G, Elgueta F, Fierro C, et al. Nitrous oxide (N₂O) reduces postoperative opioid-induced hyperalgesia after remifentanil-propofol anaesthesia in humans. Br J Anaesth. 2011;107: 959–65.
- Saeedi OJ, Wang H, Blomquist PH. Penetrating globe injury during infraorbital nerve block. Arch Otolaryngol Head Neck Surg. 2011;137:396–7.
- Kargi E, Hoşnuter M, Babucçu O, Altunkaya H, Altinyazar C. Effect of steroids on edema, ecchymosis, and intraoperative bleeding in rhinoplasty. Ann Plast Surg. 2003;51(6):570–4.
- Goktas U, Isik D, Kati I, et al. Effects of lidocaine infiltration on cost of rhinoplasty made under general anesthesia. J Craniofac Surg. 2011;22:2176–8.
- Lee JH, Koo BN, Jeong JJ, et al. Differential effects of lidocaine and remifentanil on response to the tracheal tube during emergence from general anaesthesia. Br J Anaesth. 2011;106:410–5.
- Nho JS, Lee SY, Kang JM, et al. Effects of maintaining a remifentanil infusion on the recovery profiles during emergence from anaesthesia and tracheal extubation. Br J Anaesth. 2009;103: 817–21.
- 33. Lee B, Lee JR, Na S. Targeting smooth emergence: the effect site concentration of remifentanil for preventing cough during emergence during propofol-remifentanil anaesthesia for thyroid surgery. Br J Anaesth. 2009;102:775–8.
- 34. Westreich R, Sampson I, Shaari CM, et al. Negative-pressure pulmonary edema after routine septorhinoplasty: discussion of

pathophysiology, treatment, and prevention. Arch Facial Plast Surg. 2006;8:8–15.

- 35. Smith LP, Roy S. Operating room fires in otolaryngology: risk factors and prevention. Am J Otolaryngol. 2011;32:109–14.
- ECRI Institute: Fires from Oxygen Use during Head and Neck Surgery. http://www.mdsr.ecri.org/summary/detail.aspx?doc_ id=8212. Accessed 12 Feb 2011.
- Anesthesia Patient Safety Foundation: Prevention and Management of Operating Room Fires. http://www.apsf.org/resources_video. php. Accessed 12 Feb 2011.
- Neff SP, Stapelberg F, Warmington A. Excruciating perineal pain after intravenous dexamethasone. Anaesth Intensive Care. 2002; 30:370–1.
- Avramov MN, Smith I, White PF. Interactions between midazolam and remifentanil during monitored anesthesia care. Anesthesiology. 1996;85:1283–9.
- 40. Rewari V, Garg R, Trikha A, Chandralekha. Fentanyl pretreatment for alleviation of perineal symptoms following preoperative administration of intravenous dexamethasone sodium phosphate – a prospective, randomized, double blind, placebo controlled study. Middle East J Anesthesiol. 2010;20(6):803–8.
- Funk W, Wollschläger H. Acute hypertension following dexamethasone. A critical incident during anesthesia]. Anaesthesist. 2006;55:769–72.
- 42. Rohrich RJ, Potter JK, Landecker A. Postoperative man- agement of the rhinoplasty patient. In: Gunter JP, Rohrich RJ, Adams Jr WP, editors. Dallas rhinoplasty: nasal surgery by the masters. 2nd ed. St. Louis: Quality Medical; 2007. p. 125–34.
- Cochrane CS, Landecker A. Prevention and management of rhinoplasty complications. Plast Reconstr Surg. 2008;122:60e–7.
- Bhattacharyya N. Ambulatory sinus and nasal surgery in the United States: demographics and perioperative outcomes. Laryngoscope. 2010;120:635–8.

Functional Nasal and Sinus Surgery

13

Alan J. Sim, Adam I. Levine, and Satish Govindaraj

Introduction

Functional endoscopic sinus surgery (FESS) was first described in the late 1970s and has become increasingly popular in the last 40 years. With its high success rate and low risk of major complications, it is one of the most commonly performed ambulatory surgical procedures in otolaryngology^[1]. Chronic sinusitis affects approximately 35 million Americans annually and accounts for 11.6 million visits to a physician^[2]. It is common in all ages and ethnic groups. Chronic sinusitis has been shown to have a dramatic effect on quality of life, comparable to that seen in conditions such as coronary artery disease and asthma^[3]. In addition to its use for the treatment of chronic sinusitis, the FESS approach has expanded to a variety of other surgical procedures including skull base surgery, transphenoidal pituitary tumor resection (Chap. 18), and the treatment of vascular malformations^[4].

Overview

The paranasal sinuses and nasal cavity are a complex labyrinth of hollow bony structures within the skull that function to provide warmth and humidification for the respiratory tract; allow the voice to resonate; decrease skull weight and

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serve as a mechanical and immunological defense against airborne pathogens and contaminants. The sinonasal mucosa is lined by pseudostratified ciliated columnar epithelium that contains a number of specialized cells whose collective function is to maintain a mucociliary transport system that functions to produce and clear a protective layer of mucous flowing from the sinuses into the nasal cavity.

When the paranasal sinus ostia become obstructed due to edema from allergy or infection, trauma, anatomic variation, foreign bodies, tumor, or ineffective mucociliary clearance, infection may ensue (Fig. 13.1). While medical management is almost always the initial treatment in all these cases, surgical intervention plays a definitive role by removing the obstruction and restoring sinus ventilation and mucociliary function.

Although FESS is reported to be safe, a number of serious complications, including death, have been reported. Major complications include massive hemorrhage, anosmia, blindness, cerebrospinal fluid leak, intracranial infection, or direct brain injury. These complications have been attributed to the close proximity of the paranasal sinuses to major blood vessels including the carotid artery, nerves, orbits, and the intracranial cavity (Fig. 13.2)^[5,6].

Intraoperative bleeding into the surgical field is thought to contribute significantly to these complications by impairing visualization of these important anatomical structures ^[7]. Poor surgical conditions can not only increase the complication risk, but can also prolong surgery and lead to conditions requiring reoperations like mucoceles from retained tissue. Poor surgical conditions can be due to a number of factors related to the patient, the surgery, and the anesthetic technique.

In addition to creating a "bloodless" intraoperative surgical field, the anesthetic goals for FESS include airway protection, patient immobility, a smooth emergence and extubation to limit postoperative bleeding from bucking, and a rapid recovery profile by avoiding postoperative nausea and providing sufficient postoperative analgesia. There are a variety of evidence-based anesthetic techniques that can be employed to reduce intraoperative bleeding, optimize surgical conditions, and limit complications from FESS.

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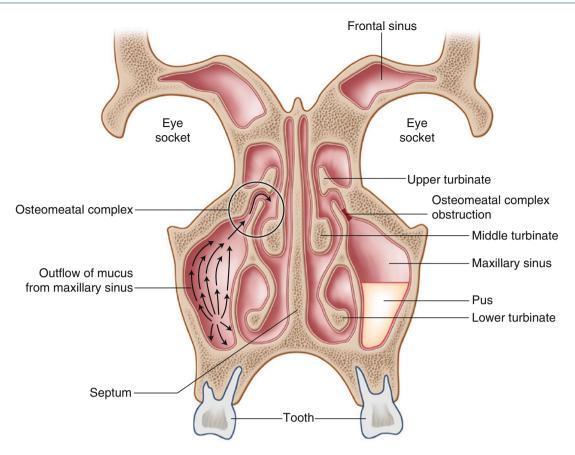


Fig. 13.1 The *right* maxillary sinus shows normal mucociliary clearance and *left* maxillary sinus shows ostium obstruction and secondary development of acute sinusitis

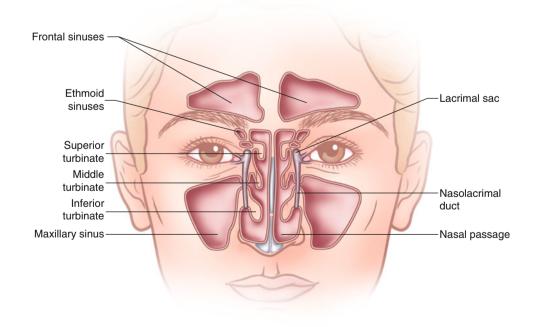


Fig. 13.2 Proximity of sinuses and surgical field to critical structures including the brain and orbit

The major challenge to the anesthesiologist caring for patients undergoing FESS lies in the negotiation between the patient's comorbidities and the risks incurred by creating surgical field optimization via induced physiological derangements (i.e., reduction in cardiac output and hypotension).

Preoperative Considerations

Otolaryngologist's Perspective

Patients undergo sinonasal surgery for a variety of reasons including nasal obstruction, control of epistaxis, and the treatment of inflammatory disease and neoplasia.

Nasal Obstruction Surgery

Surgery to address nasal obstruction consists of septoplasty, inferior turbinate reduction, and in certain cases correction of concha bullosa, a normal anatomic variant of the middle turbinate characterized as being air filled. In the vast majority of times medical therapy consisting of nasal sprays, allergy treatment, and environmental avoidance of allergens will alleviate the symptom of nasal obstruction. Patients are offered the option of surgery only if these medical modalities fail; however, this is a surgery to improve quality of life. The patients must decide whether their quality of life has deteriorated to a degree where they would entertain a surgical procedure.

The inferior turbinates are vascular structures that filter and humidify inspired air and may enlarge due to external stimuli such as allergens causing nasal obstruction. Patients undergo an endoscopic exam in the office and any areas of septal deviation or turbinate enlargement are noted. Most patients undergo allergy testing as well.

Epistaxis

Refractory epistaxis is seen in patients with anticoagulant and antiplatelet medication use, digital or external trauma to the nose, excessive nose blowing, and any condition leading to increased nasal vascularity such as allergies or infection. In addition, trauma and environmental factors such as air dryness from central heating and lack of humidity in the winter are common causes of epistaxis. Patients undergo a complete coagulation history including a focus on easy bruising or bleeding disorders, family history of easy bruising or bleeding disorders and medications. Lab tests including PT, PTT, INR, platelet count, and in certain cases bleeding time

Table 13.1 Diagnostic factors for chronic rhinosinusitis		
Major factors	Minor factors	
Facial pain/pressure	Headache	
Facial congestion/fullness	Fever	
Nasal obstruction	Halitosis	
Nasal discharge	Fatigue	
Hyposmia/anosmia	Dental pain	
Purulence in the nasal cavity	Cough	

will also be ordered. If lab work returns abnormal, a hematologist consultation is recommended to determine the precise coagulation defect and help guide perioperative coagulation management.

Prior to surgical intervention, patients will undergo nasal packing placement with either an anterior or anterior/posterior pack. This is kept in place for a minimum of 48 h and antibiotic prophylaxis for sinusitis is initiated. In addition, reversal of anticoagulation and blood pressure reduction are done if there is no medical contraindication to do so. When these measures fail, surgical exploration is indicated with either endoscopic direct cauterization or clipping of the sphenopalatine artery. In severe cases, clipping the anterior ethmoid artery is needed and performed through an external approach. Rarely these patients may require endovascular embolization.

Inflammatory Sinus Disease

The surgical management of inflammatory sinus disease has a success rate greater than 95% with proper patient selection ^[3]. If patients have stability of disease for greater than 18 months from the time of their surgery, their need for any revision surgery in the future significantly decreases [8]. Sinusitis has very strict criteria established by the American Academy of Otolaryngology. Acute bacterial sinusitis is a condition that lasts up to 4 weeks and has symptoms of fever, nasal congestion, purulent rhinorrhea, and is distinguished from a viral rhinosinusitis only by a duration of greater than 7-10 days. Chronic sinusitis is a separate entity with a duration of symptoms for greater than 3 months and established diagnostic criteria of major and minor factors (Table 13.1). In order to diagnose chronic sinusitis one needs at least two major or one major and two minor criteria. A computerized tomography (CT) scan of the paranasal sinuses is generally not necessary to make the diagnosis of chronic sinusitis. Surgical intervention for inflammatory sinus disease is recommended if medical management of acute or chronic sinusitis fails or if frequency exceeds four or more infections per year.

Ear pressure/fullness

A preoperative CT scan is performed prior to any surgical intervention as a map of the sinonasal anatomy used for dissection as well as to rule out the possibility of an underlying tumor causing secondary post obstructive sinusitis. Advancements in imaging have fostered the adaptation of CT image-guided surgery; however, this technology does not replace experience and a sound knowledge of the anatomy. In addition, the use of this technology has not resulted in decreasing complication rates associated with endoscopic sinus and skull base surgery ^[9]. The key areas to examine on a preoperative CT scan are the skull base with any asymmetry, dehiscence, or short height; location of the anterior ethmoid neurovascular bundle; the presence of anatomic variants such as an Onodi (ethmoid cell lateral or superior to sphenoid sinus) or Haller (ethmoid cell along floor of orbit) cell; the attachment site and presence of atelectasis of the uncinate process; the integrity of the lamina papyracea (medial orbital wall); and any dehiscence of the carotid artery or optic nerve in the sphenoid sinus.

Neoplasia of the Sinonasal Cavity

Endoscopic management of neoplasia of the sinonasal cavity has evolved and will continue to evolve over the next several decades as the indications for treatment are growing to include malignant tumors. In order to avoid areas of controversy, our discussion will pertain to the management of benign neoplasms. Other than nasal polyps, inverted papilloma is the most common neoplasm of the sinonasal cavity which carries a risk of malignant conversion to squamous cell carcinoma in 8-10% of cases [10]. The management of this tumor has evolved over the last three decades from a purely open treatment to mainly endoscopic treatment with open approaches used as an adjunct [11]. Inverted papilloma can have unicentric or multicentric attachment and the areas of attachment must be addressed to avoid the risk of recurrence ^[12]. This is done by surgical resection or drilling the underlying bone at the attachment site in critical areas such as the skull base.

Another tumor that is addressed endoscopically is juvenile nasopharyngeal angiofibroma (JNA)^[13]. JNAs are benign tumors that are locally aggressive and originate predominantly from the sphenopalatine foramen with extension to the nasopharynx, pterygopalatine and infratemporal fossae, sphenoid sinus, and at times have intracranial extension ^[13, 14]. It occurs in adolescent males and is known to be an androgen-dependent tumor with receptors for testosterone, dihydrotestosterone, and androgen ^[13, 15]. This is a tumor that possesses significant vascularity due to the presence of large venous channels that lack an elastic lamina thus topical vasoconstrictive agents may not be as effective in controlling blood loss and anesthetic control of hemodynamics is critical to maintain a surgical field that permits safe dissection. There are adjunctive measures such as preoperative embolization and intraoperative technology (i.e., coblation) ^[14] that help control intraoperative blood loss as well. In the management of benign tumors, clinical judgment and balancing the risks and benefits to the patient play a significant role that is unlike the treatment of malignant tumors where complete resection with clean margins is paramount. A bloodless field many times is not feasible; however, maximizing surgical conditions to improve visualization will allow a safer dissection and avoidance of unnecessary complications.

Anesthesiologist's Perspective

The preoperative evaluation of patients undergoing FESS should be, as always, a thorough, yet directed evaluation performed in accordance with the current American Society of Anesthesiologists (ASA) guidelines on preanesthestic evaluation. Such an evaluation includes a pertinent medical history with a focus on prior anesthetics, cardiopulmonary function, and medications taken. Physical examination should include an airway exam, a pulmonary exam with auscultation of the lungs, and a pertinent cardiovascular examination. Additional testing and laboratory studies should be obtained as indicated by the history and physical exam. Patients undergoing FESS may, in general, have comorbidities that may be of particular concern to the anesthesiologist. A systems-based approach should be employed with specific attention to the cardiovascular and respiratory systems.

Respiratory

Patients presenting for FESS vary in age from children to adults with roughly a 1:1 ratio of male to female ^[16]. Preoperative diagnoses include nasal polyps, chronic sinusitis, recurrent sinus infections, benign and malignant tumors, or management of previous surgical procedures (e.g., CSF leak, scar tissue formation) ^[16]. Over 35 million Americans suffer from chronic sinus disease and associated conditions may include obstructive sleep apnea (Chap. 9), gastroesophageal reflux disease (GERD), and cystic fibrosis. Roughly 20-30% of patients with nasal polyposis have reactive airway disease or asthma. Many of these patients have aspirin sensitivity (Samter's Triad), which can precipitate bronchospasm, urticaria, or anaphylaxis ^[16]. Nonsteroidal antiinflammatory drugs (NSAIDs) including ketorolac should be avoided in these patients, although acetaminophen is generally considered safe.

OSA patients may be problematic as they are more likely to be difficult to bag mask ventilate and are exquisitely sensitive to narcotic-induced respiratory depression. They should receive minimal to no sedation prior to induction and analgesia should be limited to short-acting opioids, acetaminophen, and local anesthetics. Postoperatively they may require admission to a monitored setting. Use of continuous positive airway pressure (CPAP) devices at home would ideally be used during recovery or immediately upon emergence, but in sinonasal surgery CPAP is rarely feasible and may be associated with postoperative bleeding or aspiration of blood.

Although not routine, when indicated, patients with a history of poorly controlled asthma or reactive airway disease should be seen and medically optimized by their pulmonologist or primary care physician prior to surgery. Preoperative use of oral or inhaled beta 2 agonists (albuterol), anticholinergics (ipratropium bromide), antileukotrienes (montelukast), corticosteroids, and antibiotics may be necessary. The use of intraoperative beta-blockers, even beta 1 selective agents metoprolol or esmolol, for controlled hypotension should be approached with caution because of increased likelihood of bronchospasm. A chest X-ray should be ordered if the history and physical exam are non-reassuring. Pulmonary function tests are rarely needed or useful.

Cardiovascular

Cardiovascular assessment can be obtained with a thorough history to determine the patient's functional exercise tolerance and the presence of symptoms with physical exertion. Any complaints of dyspnea, chest pain, palpitations, or dizziness with climbing 2 or more flights of stairs (>4 metabolic equivalents or METS) or occasionally during rest may indicate the need for further cardiovascular testing including an ECG, echocardiogram, stress test, or coronary angiography. Patients with a history of OSA may have associated pulmonary hypertension and right ventricular hypertrophy that can be diagnosed on a preoperative 12 lead ECG or echocardiography.

Patients with a history of significant coronary artery disease (CAD) or cardiac arrhythmias may not tolerate locally applied vasoconstrictors such as cocaine or epinephrine, which should be used with caution. Antiplatelet or anticoagulation therapy in patients should be discontinued prior to surgery, however, particular concern exists in the setting of coronary stent placement and cessation and resumption of these medications should be coordinated carefully with the patient's cardiologist ^[17]. Helpful preoperative studies would include a coagulation profile including prothrombin time and activated thromboplastin time, and complete blood count. In addition, controlled hypotension (discussed below) is not without risk and patients with significant cardiac or vascular disease including valvular pathology, congestive heart failure, and atherosclerotic disease may not tolerate significant decreases in mean arterial pressure and can suffer significant end-organ damage as a result.

Endocrine

Although chronic steroid use is common among patients undergoing FESS and its use for treatment of chronic sinusitis is controversial, preoperative steroids have been shown to improve surgical conditions in patients with rhinosinusitis and polyposis due to their anti-inflammatory and anti-edema effects ^[18, 19]. In addition, their role as an antiemetic is advantageous. Generally, adrenal pituitary insufficiency is not a concern so long as patients receive their usual daily maintenance dose preoperatively or are administered equivalent intravenous doses during the intraoperative period ^[20].

Intraoperative Considerations

Otolaryngologist's Perspective

Septoplasty and Turbinate Reduction Procedures

A complete description of the surgical procedures performed is beyond the scope of this chapter; however, the key points will be discussed. The need for a bloodless field is not critical during septoplasty or a turbinate reduction procedure. In general, when performed in the correct manner, a septoplasty will have minimal blood loss regardless of intraoperative hemodynamics (Fig. 13.3). A septoplasty can be performed with a headlight or endoscope. The endoscopic septoplasty is ideal for a posterior deviation or an isolated septal spur since it allows a more direct approach to the affected area. Turbinate reduction procedures can be performed in multiple ways ranging from radiofrequency ablation to partial turbinate resection. Packing is not placed in these procedures and occasionally silastic splints are placed over the septal flaps to help prevent septal hematoma.

Surgery for Epistaxis

In the surgical management of epistaxis, patients are usually brought to the operating room with nasal packing in place. It is important that the packing not be removed until the patient is prepped and draped and there is a functional suction and cautery device available. Although we usually recommend hypotensive anesthesia, in the management of epistaxis we prefer to begin the surgery at a normotensive or slightly hypertensive level in order to localize the site of bleeding. Hypotension, in fact, may give the surgical team a false impression as to the quality of hemostasis and therefore, is avoided. If there is active bleeding, immediate reduction in cardiac output may be necessary for better visualization. The use of short-acting agents is recommended since an acute elevation of blood pressure may subsequently be necessary

Fig. 13.3 An endoscopic view demonstrating elevation of the right mucoperichondrial flap during a septoplasty. On the left side note the vasculature of the septal mucoperichondrium with the cartilage on the right. When in the right plane, this is an avascular dissections and blood loss is minimal regardless of hemodynamics

after cauterization or ligation. Once this is established a standard hypotensive technique is initiated if there is no medical contraindication. The surgical plan calls for localizing the site of bleeding and performing either bipolar or unipolar cauterization of the site. A sphenopalatine artery ligation is performed when there is diffuse bleeding with a no discrete site of origin, or if no bleeding site was identified after packing removal and the history was suggestive of posterior arterial bleed with no septal source.

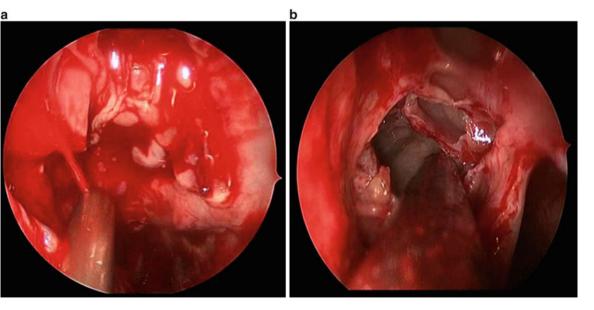
Inflammatory Sinus Surgery

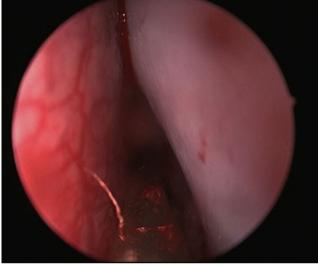
A hypotensive anesthetic technique is critical for endoscopic sinus surgery in both the management of inflammatory and neoplastic disease. Only those sinuses that demonstrate pathology are addressed during surgery. In general normal sinuses on preoperative CT scan are not opened to avoid exposing a patient to unnecessary risk. Intraoperative control of blood loss is managed by anesthetic technique; however, topical vasoconstriction is critical to maintain a "dry" field and permit safe dissection along the skull base and medial orbit. Another factor that must be considered is the degree of inflammation in the cavity. In patients with inflammatory polyp disease and vascular tumors, topical vasocontrictive agents and hypotensive anesthetic technique may not provided a clear surgical field and only after removal of the disease tissue does the surgical field improve (Fig. 13.4).

A typical sinus surgery begins with a maxillary anstrostomy. The maxillary sinuses are cleared of disease and the

Fig. 13.4 Despite an idealized (as described below) anesthetic technique and intraoperative vasoconstriction, there is a poor surgical field due to advanced inflammatory polyp disease. (a) Once the inflamed

polyp tissue is removed, the mucosa of the ethmoid skull base can be seen and the surgical field shows marked improvement and the ethmoidectomy can be safely completed (b)





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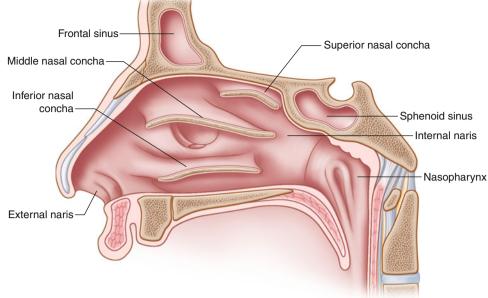


Fig. 13.5 Schematic showing the inferior slope of the skull base as one moves from an anterior to posterior direction

roof of the sinus is identified as it serves as a surgical landmark to avoid intracranial entry during dissection of the ethmoid cavity. If a surgeon dissects at the level of the maxillary sinus roof through the ethmoid cavity, a cerebrospinal fluid (CSF) leak will be less likely. Once the maxillary sinus is opened, the ethmoid sinuses are dissected next in an anterior to posterior direction up to the sphenoid sinus. The skull base is usually identified in the posterior ethmoid cavity or sphenoid sinus and then is skeletonized now in a posterior to anterior direction along the roof of the ethmoid cavity. The reason for this direction of dissection is that the skull base is at its lowest point posteriorly in the ethmoid cavity or sphenoid sinus, and identification of it at its lowest point reduces the risk of a CSF leak (Fig. 13.5).

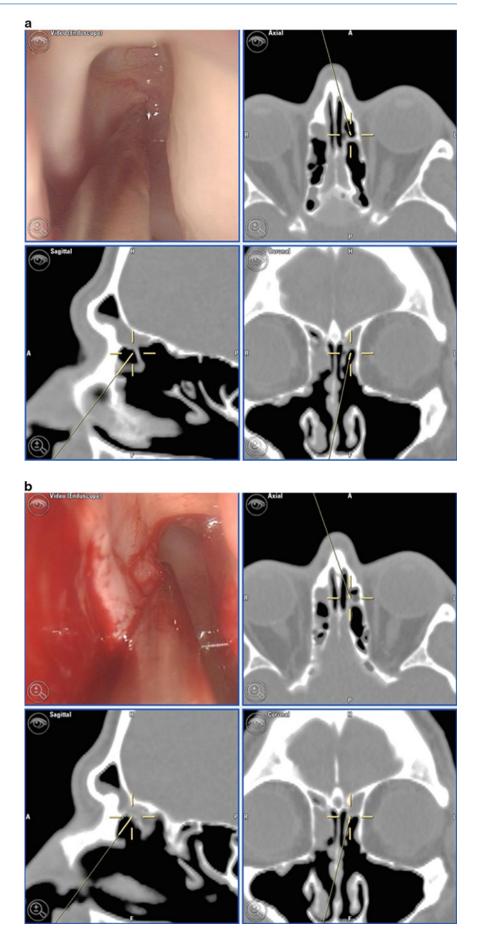
It is critical to remove all bony septations within the ethmoid cavity to maximize the surgical results and minimize the risk of delayed mucocele formation ^[21, 22]. This requires patience and meticulous dissection especially in revision cases where the bone has become osteitic (thicker) and more difficult to dissect. The sphenoid sinus is opened if there is disease present, and as mentioned the carotid artery or optic nerve may be dehiscent in the lateral wall of the sinus. We do not recommend the use of powered instrumentation within the sphenoid sinus unless the sinus is being transgressed for an endoscopic skull base procedure. The frontal sinus is usually the last sinus dissected and most difficult since identification of the medial orbital wall and skull base is critical for frontal ostium identification. One does not want to rely on image guidance to identify the frontal sinus drainage pathway, but rather use this technology as a means to confirm surgical location (Fig. 13.6).

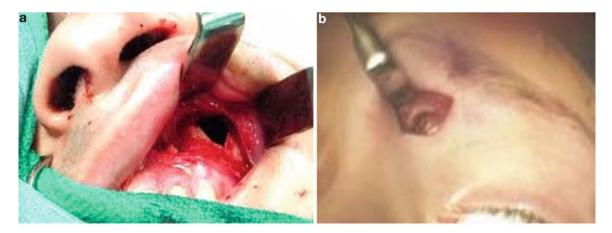
The majority of inflammatory and benign neoplastic procedures of the nasal cavity and paranasal sinuses are accomplished through an endoscopic approach. There are certain exceptions where external access to the maxillary sinus via a Caldwell-Luc approach or the frontal sinus through either trephination or an osteoplastic recessor flap (Fig. 13.7).

The Caldwell-Luc requires an upper gingivobuccal incision and dissection through the periosteum and onto the anterior wall of the maxilla. The canine fossa, a bony depression just superior and posterior to the upper bicuspid tooth, is the usual point of entry. Image guidance can also be utilized as an adjunct in order to permit entry into the sinus away from an area involved with tumor.

A frontal sinus trephination is the creation of an anterior window in the medial floor of the frontal sinus. This additional port allows an above and below dissection of the frontal sinus as well as access to the lateral and superior regions of the sinus that may be inaccessible through an endoscopic approach. The incision, once healed, is imperceptible. An osteoplastic flap is utilized in inflammatory frontal sinus disease for those cases that have failed an endoscopic approach. The most common reasons are neo-osteogenesis of the frontal recess with failure to maintain endoscopic patency ^[23]. In the treatment of bony or soft tissue tumors with areas of attachment that are not accessible through an endoscopic or trephination approach, an osteoplastic flap and obliteration of the frontal sinus with abdominal fat is the procedure of choice. In general hypotensive anesthetic techniques do not provide added benefit during open sinus procedures, and this is important information for the anesthesiologist so as to avoid any of the potential risks associated with this method of anesthesia.

Fig. 13.6 (a) The figure demonstrates the utility of the image guidance system in assisting in frontal sinus drainage identification. The probe in the top left figure is within an agger nasi cell in the anterior frontal recess. The frontal sinus drainage pathway will be posterior to this cell. (b) The probe now demonstrates entry into frontal sinus after partial removal of the agger nasi cell obstructing the frontal recess. This cell should be completely removed when performing a frontal sinusotomy





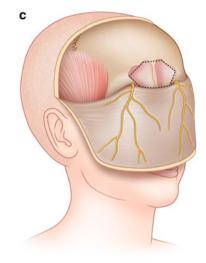


Fig. 13.7 (a) A Caldwell-Luc window is shown to gain access to the maxillary sinus. There is no visible incision in this approach. (b) Left Frontal Sinus Trephination with creation of a surgical window in the anterior floor of the frontal sinus that provides an additional access port

Balloon Sinuplasty

Balloon sinuplasty is a novel technique in the management of inflammatory sinus disease and has utility in the mild chronic sinusitis patient without hypertrophic mucosal changes. A guidewire is used to cannulate the obstructed sinus, and a balloon is passed over the guidewire and inflated to dilate the sinus opening. This technology cannot be used for the ethmoid sinuses. The true indications for this procedure, however, have not yet been determined ^[24], and they will evolve over the next several years. This procedure causes less mucosal trauma and may be utilized in the patient with inflammatory sinus disease who is a high surgical risk, since hypotensive anesthesia and unnecessary blood loss would be considered too risky. Another advantage is that this procedure may be performed in the office (Chap. 21) using the regional blocks we describe (later in this chapter) together with topical anesthetics (Fig. 13.8).

to the frontal sinus. (c) Drawing of the osteoplastic flap for complete access to the frontal sinus. The scalp is reflected inferiorly and a template of the anterior wall of the frontal sinus is created and reflected inferiorly giving access to the entire frontal sinus

Anesthesiologist's Perspective

Anesthetic Technique

If sinonasal disease is minimal and the patient is highly motivated, simple sinus surgery can be performed with the infiltration of local anesthetic, nerve blocks, and vasoconstrictors alone or with monitored anesthesia care (MAC)^[25]. In addition, good communication between all parties involved (surgeon, anesthesiologist, and patient) will be necessary to create optimal surgical conditions. However, patients who request general anesthesia or those with claustrophobia or conditions that limit their ability to cooperate (e.g., mental retardation) are poor candidates for these procedures under MAC. MAC should be used with caution in patients with significant risk of aspiration (GERD), airway compromise (OSA, obesity, airway pathology) or those with extensive sinus disease, active infections, or large tumor excisions that

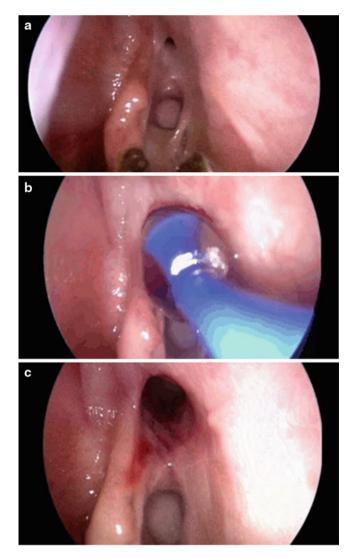


Fig. 13.8 (a) Preoperative image of stenotic left frontal sinus ostium. (b) In office placement and inflation of balloon under local anesthesia. (c) Post dilatation appearance of frontal ostium with minimal mucosal trauma and bleeding

will likely require longer operation times and can be associated with increased blood loss. In addition, as FESS grows in popularity and its applications broaden as a surgical technique, it is anticipated that general anesthesia will be preferred for more complex and aggressive procedures. For these reasons, general anesthesia is often the anesthetic technique of choice for FESS as it provides a secure airway and a motionless surgical field. Regardless of the anesthetic technique, the majority of FESS procedures are still done on an outpatient basis, and a rapid recovery profile remains a primary goal of anesthetic management.

General Considerations

As previously stated, the primary anesthetic goal for FESS, regardless of technique, is to minimize intraoperative bleeding. Due to the limited space of the surgical environment of the nose and sinus, even small amounts of bleeding can significantly compromise surgical visibility. Optimal surgical conditions will improve surgical visibility, minimize surgical time, and reduce surgical risk. Although the cause of significant intraoperative bleeding is usually multifactorial, there are a variety of effective techniques that can be implemented during the perioperative period that reduce bleeding and inflammation.

Barring the presence of a frank coagulopathy from clotting factor deficiencies or acquired or congenital platelet dysfunction, the vascularity of the nasal and sinus mucus plays a significant role in intraoperative bleeding. Mucosal bleeding is a function of the intensity of blood flow through the major vessels and capillaries within the nasal mucosa. This intensity is influenced by several factors including the relationship between mean arterial pressure (MAP) and regional or central venous pressures. Controlled or deliberate hypotension has long been advocated as an anesthetic technique to aid in the reduction of blood loss and improve visualization in sinus surgery ^[26, 27].

Although it is generally accepted that a MAP of 50 mmHg or a 30% drop from baseline MAP is safe in healthy ASA 1 patients, the definition of deliberate hypotension is not an exact number or blood pressure measurement. Deliberate or controlled hypotension must be individualized to each patient and the level of hypotension should not be determined prior to induction, but rather provided until the desired effect is obtained (e.g. dry or bloodless surgical field). The risk to achieve a "bloodless field" must be counterbalanced by the mandate to maintain acceptable cerebral and coronary blood flow ^[27].

Mean arterial pressure is a product of systemic vascular resistance (SVR) and cardiac output (CO):

$MAP = SVR \times CO$

Although a reduction in either SVR, CO, or both can be implemented to achieve a lower MAP, interventions that reduce CO have been demonstrated to be more effective in improving surgical conditions when compared to interventions that reduce SVR. Reduction of SVR is achieved primarily through the use of vasodilating agents such as calcium channel blockers, nitrates, or potent inhaled anesthetics. Blockade of alpha-adrenergic receptors can also be employed via drugs such as labetalol, hydralazine, or phentolamine. Reduction of SVR, however, has not been shown to improve operating conditions unless profound hypotension is achieved (MAP<50 mmHg), and there is evidence to suggest that vasodilatation may worsen bleeding and promote increased capillary blood flow ^[7].

Cardiac output is dependent upon preload, afterload, contractility, heart rate, and rhythm. Beta-blockers decrease heart rate and contractility, and there is a growing body of evidence that shows a significant correlation between bradycardia and improved operating conditions. In addition, optimal surgical conditions were achieved at higher mean arterial pressures (MAP>65 mmHg) in studies where betablockade was implemented as the primary technique in decreasing cardiac output ^[7]. Short-acting beta-blockade via esmolol was found to have superior operating conditions to that of sodium nitroprusside, which required a much lower MAP of 50–54 mmHg to achieve optimal conditions ^[7]. This suggests that decreased cardiac output via a reduction in heart rate and contractility may be more important than simply a reduction in systemic blood pressure.

Perioperative beta-blockade continues to be a controversial topic within anesthesiology and current studies show an increased risk in morbidity and mortality when beta-blockade is implemented in patients not otherwise "needing" these agents perioperatively. These results were observed in patients who were at a high risk of cardiovascular complications who were not previously on beta-blockers before surgery ^[28]. The effect of short-term use of beta-blockade intraoperatively in low-risk patients on morbidity and mortality has yet to be studied. Regardless, deliberate reduction of cardiac output is not without risk. Ischemic organ failure as a result of controlled hypotension is estimated at 0.6% ^[29]. As previously stated, the surgeon's need for achieving a bloodless surgical field may be at odds with the anesthesiologist's goal of maintaining adequate oxygen delivery to tissues. This technique may be challenging to implement in specific patients and should be tailored to their needs and limitations.

Preoperative Medications

Administration of steroids reduces inflammation and edema and is generally advocated preoperatively ^[19]. Preoperative antibiotic administration, however, has not been shown to have an effect on surgical conditions. Discontinuation of antiplatelet medications and anticoagulants should be considered after discussion with the patient's cardiologist and proper evaluation of the patient's cardiovascular risk, particularly with a history of significant coronary artery disease ^[17].

Local vasoconstriction is commonly infiltrated into the nasal sinuses to decrease mucosal congestion, decrease blood loss, and help achieve hemostasis (technique is described in more detail in the regional anesthesia section of this chapter). The combination of a local anesthetic is also frequently employed to provide intraoperative and postoperative analgesia^[25]. Topical agents are also used in combination with infiltration techniques. Common agents used are oxymetazoline, phenylephrine, cocaine, and epinephrine^[25]. The major risk attributed to use of vasoconstrictors in the nasal mucosa lies in the systemic absorption of these drugs and their related side effects. Serious adverse consequences that have been reported include hypertensive crisis, bradycardia, unstable tachyarrhythmias, myocardial infarction, stroke, and cardiogenic shock. These medications should

be used with caution or avoided entirely in patients with significant cardiac disease. Excessive use of phenylephrine or epinephrine results in a primarily alpha-agonist-induced hypertension and use of beta-blockade as treatment can be particularly dangerous by exacerbating increased afterload on the heart. Use of direct vasodilators or alpha-antagonists is the appropriate medical therapy in this instance ^[30].

Positioning

Head elevation or the reverse Trendelenberg position has been advocated as a technique to reduce blood loss and improve surgical conditions in FESS. Elevation of the head at least 15° creates venous pooling in the lower extremities and reduces venous sinus congestion and edema^[31]. It should be noted that blood pressure measurements at the level of the heart would not be indicative of pressures at the level of the brain and Circle of Willis, which will be lower. In addition, this decrease in blood pressures at the level of the head will be further exacerbated if a deliberate hypotensive technique is employed. Finally, this positioning also poses a minimal but not insignificant risk of venous air embolism, and such cases have been reported in FESS [32]. FESS can be performed with the head of the bed towards the anesthesiologist, or turned 90° away, depending on surgeon preference. If the head of the bed is turned 90°, care should be taken to secure the endotracheal tube and the use of circuit extensions (e.g., goose neck) is helpful. The arms may be crossed over the patients lap with the arm slightly flexed, and in this case placement of an antecubital intravenous line may not be reliable, especially if using total intravenous anestheia (TIVA). Care should be taken to assure intravenous flow is adequate and patent post-positioning. In addition to the anesthesia equipment, endoscopic sinus surgery requires bulky surgical equipment: an endoscope tower with screen and camera, image guidance system, and surgical instruments for the procedure. Proper setup and positioning is critical to allow access to the airway in the case of an emergency (Fig. 13.9).

Induction

Prior to induction, standard ASA monitors (ECG, NIBP, pulse oximetry, capnography) should be implemented. Use of an arterial catheter may be considered based on the patient's comorbidities, length of surgery or anticipated blood loss from neoplastic resections. A peripheral 18 g intravenous catheter is usually sufficient venous access, as estimated blood loss for FESS ranges from 150 ml (low-risk) to 300 ml or more (high-risk) and fluid requirements are minimal, roughly 3–4 ml/kg/hr ^[16]. Most of these patients will not require a urinary catheter.

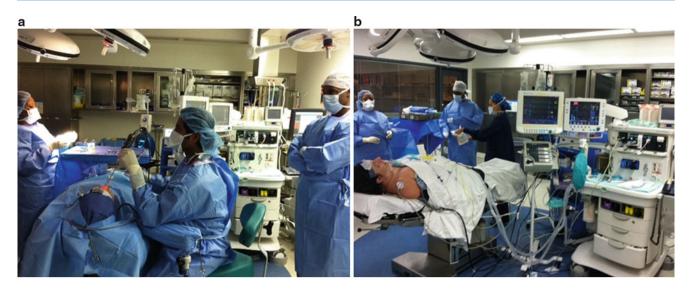


Fig. 13.9 Intraoperative set up for FESS showing a. surgeon position and b. final patient position (reverse Trendelenberg at 30°)

Induction of general anesthesia is usually achieved with the administration of propofol (1.5-2.5 mg/kg), as it is an ideal induction agent due to its rapid redistribution and antiemetic effects. A fentanyl (2–3 mcg/kg) or shorter-acting remifentanil (1–2 mcg/kg) bolus can be administered to blunt the hemodynamic response to intubation ^[16]. Remifentanil boluses greater than 2 mcg/kg have been associated with significant bradycardia, hypotension, or both ^[33]. A dose of glycopyrrolate 0.2 mg can be given prior to or concurrently with administration of remifentanil to reduce the incidence of significant bradycardia, without the risk of an increased heart rate during laryngoscopy ^[33]. Alternatively, lidocaine (1–2 mg/kg) administered intravenously as an adjunct to smaller doses of opioids and propofol can also be used to reduce the hemodynamic response to intubation.

Neuromuscular blocking agents (NMBA) may be used to facilitate endotracheal intubation and mechanical ventilation in FESS. There is always a concern when using neuromuscular blockade of residual paralysis and an increased risk of postoperative complications and re-intubation. Monitoring of neuromuscular blockade is often subjective in nature and several studies have indicated that recognition of fade with a train-of-four stimulus is unreliable among anesthesiologists ^[34–36]. As a result, objective train-of-four monitoring is advocated if neuromuscular blockade is used ^[33].

FESS does not require significant muscle paralysis and avoiding such agents will eliminate any risk of residual blockade. In addition, in most situations neuromuscular blockade must be reversed prior to emergence. Although there is currently conflicting evidence as to the use of neostigmine and the incidence of significant postoperative nausea and vomiting, one may argue similarly that avoiding a drug that may increase the risk of nausea and vomiting eliminates that risk entirely ^[37]. Facilitation of endotracheal intubation is not a requisite for muscle relaxation and a number of methods and techniques have been described forgoing the use of NMBAs in airway management. In our institution a high dose (\geq 5 mcg/kg) of remifentanil is used to facilitate tracheal intubation and minimize tachycardia and hypertension. A single dose of glycopyrrolate 0.2 mg minimizes severe bradycardia and hypotension and does not promote tachycardia, which might disrupt the planned hypotensive technique.

Airway Management

General anesthesia with an endotracheal tube (ETT) is the predominant airway management option for FESS. Topical local anesthetic applied to the vocal cords on direct laryngoscopy reduces the risk of laryngospasm and coughing on emergence. The use of an oral RAE (named after the inventors Ring, Adair and Elwyn) tube versus a standard ETT allows for easy surgical access and results in less kinking at the mouth ^[16]. The disadvantage of the RAE tube is the depth of the preconfigured bend in the tube is determined by the internal diameter (ID) of the tube. On average an orally placed ETT would be secured at 21 cm at the lips for females and 23 cm for males. Corresponding RAE tubes would require the use of potentially larger tubes than desired (7.5 ID for females and 8.5 ID tubes for males) (Table 13.2). The use of an appropriate ID RAE tube may, in fact, be placed too shallow and therefore increase the risk of intraoperative extubation. Extending the patient's head (which causes the ETT to be withdrawn) for surgical access further exacerbates this risk. Using larger ID RAE tubes places the patient at increased risk of sore throat and delayed vocal cord dysfunction. A reinforced anode tube can be used successfully with the advantage of increased length and smaller ID for taller patients, who may require insertion of the ETT at a greater

 Table 13.2
 Oral
 RAE
 internal
 diameters
 and
 corresponding

 preconfigured bend depth

 </td

Internal diameter (mm)	Depth at bend (cm)
6.0	18
6.5	19
7.0	20
7.5	21
8.0	22
8.5	23
9.0	24



Fig. 13.10 Post induction photo showing proper tube position and tape as well as insertion of nasal preparation and throat pack

depth. The ETT should be positioned at the midline and secured to the chin with tape. A throat pack is often inserted to reduce gastric filling with blood and secretions (Fig. 13.10).

The laryngeal mask airway (LMA) is also a viable option for airway management in FESS. Since its inception, the LMA has grown in popularity and has replaced endotracheal intubation for general anesthesia in select cases. Its use in head and neck surgery has increased significantly since its introduction and has been implemented in FESS with relative success ^[38]. In such cases a flexible, reinforced LMA may be used. Similar to that of the reinforced anode ETT, it can be positioned away from the surgical field to facilitate access to the nasal passages. Comparisons of airway protection between LMA and ETT have shown similar rates of contamination using direct fiberoptic examination postnasal surgery ^[39]. In regards to surgical conditions and intraoperative bleeding, use of an LMA versus an ETT with propofol and remifentanil infusions was found to have significantly better visualization and lower blood loss in the first 15 min of operating time ^[40]. Proposed theories for this observation are decreased catecholamine release during insertion of an LMA versus that of direct laryngoscopy and intubation and reduced

venous congestion due to spontaneous ventilation or limited positive pressure ventilation via the LMA. However, after 15 min similar surgical conditions were achieved, albeit the group with the ETT required higher infusion rates of remifentanil^[40]. In addition, another advantage attributed to the use of the LMA was its ability to offer a smoother emergence; however, techniques described using remifentanil and propofol with an ETT can result in a similarly smooth emergence without coughing or straining. Our group prefers the use of an ETT and utilizes high doses of remifentanil during the anesthetic induction to decrease blood pressure and heart rate from endotracheal intubation and continues a high-dose infusion throughout the procedure. Approximately 10 min before surgical completion, the remifentanil infusion is reduced to 0.05 mcg/kg/min and maintained until extubation to avoid bucking during emergence.

Ultimately, the choice to use an ETT or LMA for FESS will depend on the anesthesiologist's and surgeon's preferences and experience with a particular device, the duration and complexity of the procedure, and the patient's comorbidities and risk of aspiration. The LMA does not protect the airway against regurgitation, and in patients with a history of significant GERD, obesity, hiatal hernia or prior gastric surgery use of an LMA should be discouraged. Using the LMA for FESS or head and neck surgery is an advanced use of the device, and the anesthesiologist should be familiar with its use in such situations. Inadequate seating of the device may result in partial obstruction requiring high inspiratory pressures, which may result in gastric insufflation and a greatly increased risk for aspiration. Large blood losses also increase the likelihood of passive aspiration of blood and debris during the procedure.

Maintenance of Anesthesia

TIVA is the preferred maintenance technique for FESS. Specifically, a remifentanil and propofol infusion-based anesthetic has been shown to provide superior surgical conditions and significant decreases in intraoperative blood loss than that of a combination of volatile anesthetic and standard narcotic ^[41, 42]. All techniques were effective in achieving hypotension or mean arterial pressures less than 60 mmHg, however, only TIVA with propofol and remifentanil was successful in significant reductions in blood loss, likely due to the effects on contractility as opposed to SVR described earlier in this chapter ^[43].

Remifentanil may exclusively be the agent accountable for such an improvement in the quality of the surgical field. Compared to fentanyl, alfentanil, or sufentanil, remifentanil alone is associated with improved visualization in FESS^[43, 44]. In addition, remifentanil in combination with sevoflurane did show similar operating conditions when compared to a propofol and remifentanil technique ^[45]. This seemingly unique advantage of remifentanil is not yet fully understood. Remifentanil, like other narcotics, reduces sympathetic tone and shows a dose-dependent decrease in heart rate and cardiac output. These properties are essential in promoting controlled hypotension for FESS. However, remifentanil's major advantage lies in its pharmacokinetics and ability to be rapidly eliminated from the plasma. This allows for a high narcotic anesthetic to be administered without concern of any residual effects once the infusion is discontinued.

A modified nitrous-narcotic technique can be implemented using 50-75% N₂O, remifentanil (0.1-0.5 mcg/kg/ min), and low-dose propofol (25-50 mcg/kg/min) (our group's preference). There are several unique advantages for this technique in FESS. As described previously, the combination of remifentanil and propofol achieves excellent conditions via controlled hypotension and decreased cardiac output from relative bradycardia. This level of hypotension and heart rate control is easily titrated by adjusting the infusions of both drugs. Nitrous oxide has several effects on the cardiovascular system. While it is a mild myocardial depressant at high doses, its sympathomimetic effects on vascular tone often counteract this effect [33]. This combination of decreased contractility and vasoconstriction may be beneficial in a reduction of blood flow to the nasal sinuses and an improved surgical field. Another major advantage to this technique is a reliable and smooth emergence via the rapid elimination of all three drugs with little or no residual effects. The use of TIVA with remifentanil and propofol has the potential to reduce coughing and straining on emergence and reduce postoperative nausea and vomiting in the postoperative period, due to propofol's antiemetic effects and remifentanil's rapid elimination ^[46]. In addition, the risk of nausea and vomiting with nitrous oxide is often attenuated by the use of antiemetic agents such as ondansetron, droperidol, and dexamethasone. Table 13.3 shows commonly manipulated variables and methods for obtaining ideal surgical conditions.

Choice of Ventilation

Tachycardia and vasodilatation can result from hypercapnia and hypoventilation and may promote bleeding and worsen surgical conditions during FESS. It has long been advocated that a mild degree of hypocapnia or hyperventilation be implemented to induce vasoconstriction in the nasal sinuses and minimize surgical bleeding ^[7,41,46]. However, this principle has been studied and has not been shown to provide any particularly significant benefit versus normocapnia or hypercapnia in surgical conditions or bleeding in patients under a nitrousnarcotic technique with propofol and remifentanil ^[47].

One could argue that positive pressure ventilation (PPV) increases central venous pressure that can increase venous

 Table 13.3
 Shows commonly manipulated variables and methods for deliberate hypotension

Parameter	Variabl	e
↓ MAP	\downarrow HR	\downarrow SVR
	++++	+
↓ HR	Remifentanil	Beta-blockade
	++++	+++
Technique	TIVA	Volatile anesthetics
	++++	+
Airway	Endotracheal intubation	LMA
	+++	++
Ventilation	Normocapnia	Hypocapnia
	++	++
Post operative	Regional	Intravenous/oral
analgesia	++	++

Absolute	Relative
Neoplasms, AVMs, and other pathology involving anterior nasal cavity—ION	History of nasal cellulitis—ION
Neoplasms, AVMs, and other pathology involving pterygopala- tine fossa or infraorbital fissure—SPG	Preoperative diplopia due to underlying condition i.e. mucocele, thyroid ophthalmopathy
Patient refusal	Need to assess sensation after surgery, i.e. orbital floor repair
Documented hypersensitivity to local anesthesia	When combined with cosmetic surgery since the infraorbital block can distort anatomy

ION infraorbital nerve block contraindicated, SPG sphenopalatine ganglion block contraindicated

bleeding. Since remifentanil strongly suppresses ventilation at infusions of >0.05mcg/kg/min, positive pressure ventilation may be unavoidable if this agent is used. It is likely that the overwhelming evidence regarding the superior benefit of a high-dose remifentanil infusion outweighs the theoretical benefit of spontaneous ventilation. Similarly the use of positive end-expiratory pressure (PEEP) is generally avoided since it too leads to a decrease in venous return and increases venous congestion, but its effect has not been formally studied.

Regional Anesthesia for FESS

Regional anesthesia for head and neck surgery is a relatively new concept and can be used in conjunction with general anesthesia or as a primary anesthetic with sedation (MAC) in select and motivated patients. Communication with the surgeon is necessary before performing regional anesthesia as there are certain contraindications based on the location of the patient's pathology (Table 13.4). The proposed advantages of using specific nerve blocks in endoscopic sinus surgery include a decrease in intraoperative and post-operative pain and a reduction in the amount of general anes-thetic or sedation required for the procedure ^[48]. When these agents are combined with vasoconstrictors, they also provide an improved operative field for the surgeon.

The maxillary division of the trigeminal nerve (V2) provides the major sensory innervation to the midface, including the lower eyelid, upper lip, maxillary sinus, nasal cavity, and the soft and hard palate. Specifically, branches of the maxillary nerve, the infraorbital (IO) nerve and sphenopalatine (SP) ganglion can be blocked with local anesthesia for FESS.

The infraorbital nerve provides sensation to the cheek, upper lip, eyelid, and lateral aspect of the nose. The infraorbital nerve exits the infraorbital foramen approximately 1 cm below the inferior orbital ridge, palpable with a finger, along a vertical line from the medial limbus of the eye. The infraorbital notch can be palpated with a fingertip rolled over the inferior orbital rim. This block can be performed intraorally or transnasally; the transnasal approach is commonly employed at our institution. For the transnasal technique, the index finger of the nondominant hand is placed over the infraorbital foramen (Fig. 13.11a, b). A 25 gauge 11/2 inch long needle is positioned through the ipsilateral nares. While aspirating, the needle is advanced towards the finger marking the foramen, and 2 ml of 0.5–0.75% bupivicaine is injected [49]. As previously mentioned, this technique should not be performed in patients with pathology such as neoplasms or AVMs involving the anterior nasal cavity such as the septum and nasal vestibule.

The sphenopalantine ganglion or pterygopalatine ganglion supplies the lacrimal gland, paranasal sinuses, the mucosa of the nasal cavity and pharynx, the gingiva, and the mucous membrane of the hard palate. The ganglion may be blocked via the intraoral or transpalatal approach. With the patient supine and neck extended, the greater palatine foramen is identified medially to the gum line of the second or third molar on the posterior portion of the hard palate. Exposure may be aided by use of a curved blade laryngoscope. While aspirating, a 25 gauge 11/2 inch long needle is bent at 1.5 cm and advanced through the foramen at an angle of 45° at a superior and slightly posterior trajectory ^[49, 50]. Upon negative aspiration, 1.5-2.0 ml of lidocaine with 1:100,000 epinephrine mixture is injected. This region is highly vascular and significant systemic absorption is minimized by the use of epinephrine. In addition, the added vasoconstriction will be helpful in creating a bloodless surgical field. It should be noted that this technique has been associated with complications such as intravascular injection, infraorbital nerve injury, or transient diplopia ^[51]. It should also be avoided in cases where pathology may involve the pterygopalatine fossa since inadvertent trauma to the tumor may occur. An alternative option is to infiltrate the region overlying the foramen and not inject into the foramen itself. Figure 13.12 shows the authors' preferred method for SPG blockade.

With increasing frequency otolaryngologists are using intraoperative computerized tomography image (CT) guidance during FESS (Fig. 13.13). The deposition of local anesthesia during the use of the infraorbital nerve block can interfere with the guidance system registration since the process relies upon facial skin depth and facial topography for proper calibration and patient identification (Fig. 13.14).

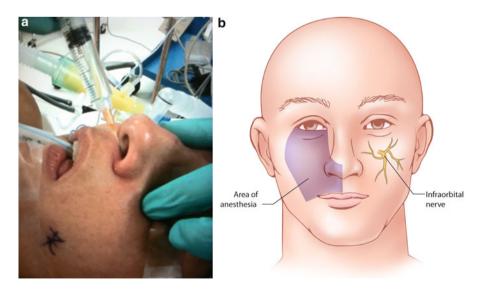


Fig. 13.11 Transnasal approach to infraorbital nerve block and relevant anatomy showing the distribution of anesthesia from the block



Fig. 13.12 Sphenopalatine ganglion block showing use of MAC 3 blade and curved needle and positioning of anesthesiologist during block placement b. Sphenopalatine ganglion blockade showing blanching of palate upon injection



Fig. 13.13 Surgeon demonstrating proper CT guidance system registration process and screen shot of registration confirmation



Fig. 13.14 Infraorbital nerve block placement and subsequent derangement of facial topography from local injection

In addition, placing the sphenopalatine block requires the ability to extend the patient's head and visualize their palate. This maneuver can disrupt the imaging band placed on the patient's forehead. Therefore, we recommend placing the SP block before the guidance system registration and the placement of the IO block after registration. In a yet to be published double blind randomized study, our group has determined that patients who receive both blocks require less opioids in the PACU and are discharged home 40 min early on average compared to patients who did not receive the blocks.

Emergence

Upon completion of surgery, and removal of the throat pack, a careful inspection of the oropharnynx and postnasal space should be performed, suctioning and removing any clots that may have formed. The goal for emergence in FESS should be as smooth as possible with minimal to no coughing or straining, as this will increase venous pressures and promote bleeding. The use of an LMA may result in a smoother emergence from general anesthesia compared to an endotracheal tube, however for reasons stated prior, the use of an LMA for FESS may not be desirable in many, if not most cases.

Extubation may be performed with the patient awake or still under anesthesia (deep) and the anesthesiologist must weigh the risks and benefits of either technique. The primary advantage of a deep extubation is a smoother recovery profile and a more expeditious transfer to the postoperative care unit (PACU). However, deep extubation results in an unprotected airway and significant risk of obstruction or aspiration. Nasal packing leaves oropharyngeal airflow the only viable option for an open airway, and this may be difficult for the anesthesiologist to maintain while the patient is still anesthetized. Frequent and thorough suctioning should be performed to decrease the risk of aspiration of blood and secretions. The advantage of an awake extubation is the return of protective airway reflexes and a conscious patient. The major disadvantage is the risk of bucking and coughing, with the possibility of laryngospasm, negative pressure pulmonary edema, and further bleeding. As stated earlier, a modified nitrous narcotic technique with a careful titration of propofol and remifentanil can achieve an awake patient reliably with minimal to no coughing. Low-dose propofol at 25 mcg/kg/min results in little accumulation of the drug. A quick and reliable emergence will be seen with an end-tidal nitrous oxide of <10%, and low-dose remifentanil infusions (0.05-0.1 mcg/ kg/min) will allow for an awake patient with adequate blunting of the cough reflex. Once appropriate extubation criteria are met, these agents will be eliminated with no residual by the time the patient is in the recovery room.

Postoperative Considerations

Otolaryngologist's and Anesthesiologist's Perspectives

One of the primary goals for the anesthesiologist in FESS is to limit the patient's stay in the postoperative care unit (PACU). As many of these cases are ambulatory procedures, it is important that these patients be discharged in a timely fashion with little to no anesthetic residual. However, as with many head and neck procedures, vigilance and close observation will be required to look for postoperative airway obstruction and bleeding, which can result in airway compromise and respiratory distress. The overall incidence of postoperative bleeding, or epistaxis, is small, roughly 1–1.6%, with major hemorrhage requiring either blood transfusion or re-exploration much rarer at about 0.19%. It is not surprising that the incidence of bronchospasm, 1.8%, is similar to that of epistaxis and indeed both conditions may be concurrent ^[52]. In addition, postoperatively many of these patients will have nasal packing resulting in a partially or completely obstructed nasal airway, with complete reliance on an unobstructed oropharyngeal airway. This may be of particular concern in patients with OSA or asthma.

Postoperative nausea and vomiting (PONV) is of concern in FESS as retching and vomiting can worsen postoperative bleeding. In addition, it leads to increased recovery time and use of additional resources. It is often multifactorial in origin, and may be attributed to upper airway inflammation, blood in the stomach, and use of opioids. Decompression of the stomach prior to extubation may be helpful. Avoidance of volatile anesthetics and use of propofol may reduce the risk of PONV and regional nerve blocks will reduce the need for longer acting opioids. Remifentanil may not play a significant role in PONV due to its rapid elimination. Highdose steroids such as dexamethasone (10-12 mg) intraoperatively as well as standard antiemetics, such as ondansetron (4 mg) or droperidol (0.625–1.25 mg) should be administered prophylactically. A scopolamine patch may be used in high-risk patients; however, it should be used with caution as it may delay emergence.

Postoperative pain after FESS ranges from mild to moderate and a multimodal approach is often implemented. Remifentanil is rapidly metabolized and will offer no sustained postoperative analgesia. Nonselective NSAIDS such as ketorolac are commonly avoided in FESS due to their inhibition of thromboxane A2 and a resultant decrease in platelet aggregation. Current evidence shows local anesthetics, selective COX-2 or COX-3 NSAIDs, and opioids have similar pain control in FESS when used separately [48, ^{53, 54]}. Therefore, the general approach is to utilize local anesthesia and nerve blocks with long acting local anesthetics such as bupivicaine prior to surgery, administer nonopioid analgesics such as acetaminophen or selective COX-2 inhibitors intraoperatively or immediately postoperatively and use longer acting opioids such as fentanyl for rescue treatments [48].

Conclusion

FESS is extremely effective in the treatment of sinonasal disease and continues to grow in scope and popularity. Intraoperative bleeding and poor visualization within the surgical field contributes significantly to the risk of surgical complications, prolongs surgical time, and may contribute to worse long tern surgical outcome. It is clear that proper anesthetic management is necessary for good outcomes, and requires an anesthesiologist well versed in the various anesthetic techniques described in this chapter. Proper utilization of these techniques and application of key physiologic principles with clear anesthetic goals can significantly make a positive impact on patients undergoing FESS.

Clinical Insights

For the Otolaryngologist (from the Anesthesiologist)

- The use of regional anesthesia, specifically, the infraorbital nerve block, may obscure or interfere with the ability to "register" and therefore the use of the frameless stereotactic surgical systems. It is important to communicate with the anesthesiologist when calibration has taken place such that these blocks may be performed.
- Patients with significant cardiopulmonary disease may not tolerate a reduction in cardiac output (deliberate hypotension), these patients should be identified early and the risks and benefits of the proposed anesthetic and procedure should be discussed with the patient, anesthesiologist, and surgeon present.
- Patients with significant cardiovascular disease may not tolerate the use of intranasal vasoconstrictive agents. Their use must be discussed with the Anesthesiology team beforehand.
- Intraoperative bleeding is multifactorial and may not respond to pharmacologic interventions.

For the Anesthesiologist (from the Otolaryngologist)

- In patients with nasal or pterygopalatine fossa tumors, an infraorbital or sphenopalatine block, respectively may result in tumor spread or violation. Prior to the placement of the infraorbital and sphenopalatine blocks, check with the surgeon to make sure there is no surgical contraindication.
- During open sinus procedures there is less of a need for significant cardiac reduction or hypotensive anesthesia.

- Endoscopic procedures for revision sinus surgery or tumor resection may require as much operative time as open procedures, however, with less morbidity.
- Secure intravenous line when using a total intravenous technique as infiltration of propofol into the subcutaneous tissue may lead to overlying skin necrosis and significant morbidity.
- Palpation of the eye during the procedure is performed at times to rule out violation of the medial orbital wall. If the orbital wall has been violated, we will see ocular movement upon external palpation.
- The eyes are not taped but rather covered with steri strips so that inspection of the eye can be performed during the surgery to rule out proptosis or chemosis that are suggestive of an orbital hematoma.

Clinical Pearls

For the Otolaryngologist (from the Otolaryngologist)

- Open communication with the anesthesiologist is important to avoid inadvertent trauma to sinonasal tumors with regional blocks or unnecessary hypotensive anesthesia.
- Inform the anesthesiologists if there is any orbital wall dehiscence to avoid orbital emphysema during preoxygenation and anesthetic induction.
- Do not underestimate the importance of cardiac reduction (hypotensive anesthesia) and topical vaso-constriction in creating a safe surgical field.
- After sinus surgery, patients undergo meticulous debridement in the office to avoid scarring and to insure a well-healed sinus cavity with normal function. Absence of postoperative care will lead to potential long-term sinus surgery failure.
- If using topical epinephrine during the procedure, one should avoid the use of oxymetazoline because it binds irreversibly to alpha receptors and decreases the efficacy of epinephrine by occupying a percentage of alpha 1 receptors.
- The use of regional blocks for postoperative analgesia has resulted in earlier discharge from post anesthesia care units (*accepted and pending publication*). This is important for surgeons operating in ambulatory surgery centers.

For the Anesthesiologist (from the Anesthesiologist)

- Reducing the cardiac output via a reduction in heart rate (remifentanil, beta-blockers) or contractility (beta-blockers) provides a superior surgical field then reducing the systemic vascular resistance with vasodilators or inhaled anesthetics.
- Merely reducing the blood pressure does not assure a superior surgical field and may only increase anesthetic risk.
- Cerebral perfusion is lower than the MAP, measured at the arm, if a head up position is utilized intraoperatively.
- The use of regional anesthesia (SP and ION blocks) may shorten PACU stays by a third and reduce postoperative analgesic requirements.
- It is imperative that the anesthesiologist, in addition to monitoring vital signs, assess and evaluate the adequacy of the anesthetic's impact on the surgical field. This will help identify possible avenues in which to maximally optimize care, such as reduction of heart rate, blood pressure, or preload.
- Intraoperative bleeding is multifactorial and may not respond to pharmacologic interventions.

References

- Senior BA, Kennedy DW, Tanabodee J, Kroger H, Hassab M, Lanza DC. Long-term impact of functional endoscopic sinus surgery on asthma. Otolaryngol Head Neck Surg. 1999;121: 66–8.
- Benninger MS, Ferguson BJ, Hadley JA, et al. Adult chronic rhinosinusitis: definitions, diagnosis, epidemiology, and pathophysiology. Otolaryngol Head Neck Surg. 2003;129:S1–32.
- Khalid AN, Quraishi SA, Kennedy DW. Long-term quality of life measures after functional endoscopic sinus surgery. Am J Rhinol. 2004;18:131–6.
- Stankiewicz JA. Advanced endoscopic sinus surgery. 1st ed. St. Louis: Mosby; 1995.
- Cumberworth VL, Sudderick RM, Mackay IS. Major complications of functional endoscopic sinus surgery. Clin Otolaryngol Allied Sci. 1994;19:248–53.
- Maniglia AJ. Fatal and other major complications of endoscopic sinus surgery. Laryngoscope. 1991;101:349–54.
- Boezaart AP, van der Merwe J, Coetzee A. Comparison of sodium nitroprusside- and esmolol-induced controlled hypotension for functional endoscopic sinus surgery. Can J Anaesth. 1995;42: 373–6.
- Senior BA, Kennedy DW, Tanabodee J, Kroger H, Hassab M, Lanza D. Long-term results of functional endoscopic sinus surgery. Laryngoscope. 1998;108:151–7.
- Stankiewicz JA, Lal D, Connor M, Welch K. Complications in endoscopic sinus surgery for chronic rhinosinusitis: a 25-year experience. Laryngoscope. 2011;121:2684–701.

- Lawson W, Kaufman MR, Biller HF. Treatment outcomes in the management of inverted papilloma: an analysis of 160 cases. Laryngoscope. 2003;113:1548–56.
- Lawson W, Patel ZM. The evolution of management for inverted papilloma: an analysis of 200 cases. Otolaryngol Head Neck Surg. 2009;140:330–5.
- Chiu AG, Jackman AH, Antunes MB, Feldman MD, Palmer JN. Radiographic and histologic analysis of the bone underlying inverted papillomas. Laryngoscope. 2006;116:1617–20.
- Blount A, Riley KO, Woodworth BA. Juvenile nasopharyngeal angiofibroma. Otolaryngol Clin North Am 2011;44:989–1004, ix.
- Ruiz JW, Saint-Victor S, Tessema B, Eloy JA, Anstead A. Coblation assisted endoscopic juvenile nasopharyngeal angiofibroma resection. Int J Pediatr Otorhinolaryngol. 2012;76:439–42.
- Hwang HC, Mills SE, Patterson K, Gown AM. Expression of androgen receptors in nasopharyngeal angiofibroma: an immunohistochemical study of 24 cases. Mod Pathol. 1998;11:1122–6.
- Jaffe RA, Samuels SI. Anesthesiologist's manual of surgical procedures. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2004.
- Douketis JD, Berger PB, Dunn AS, et al. The perioperative management of antithrombotic therapy: American College of Chest Physicians Evidence-Based Clinical Practice Guidelines (8th Edition). Chest. 2008;133:2998–339.
- Wright ED, Agrawal S. Impact of perioperative systemic steroids on surgical outcomes in patients with chronic rhinosinusitis with polyposis: evaluation with the novel Perioperative Sinus Endoscopy (POSE) scoring system. Laryngoscope. 2007;117:1–28.
- Sieskiewicz A, Olszewska E, Rogowski M, Grycz E. Preoperative corticosteroid oral therapy and intraoperative bleeding during functional endoscopic sinus surgery in patients with severe nasal polyposis: a preliminary investigation. Ann Otol Rhinol Laryngol. 2006;115:490–4.
- Marik PE, Varon J. Requirement of perioperative stress doses of corticosteroids: a systematic review of the literature. Arch Surg. 2008;143:1222–6.
- Lee JT, Kennedy DW, Palmer JN, Feldman M, Chiu AG. The incidence of concurrent osteitis in patients with chronic rhinosinusitis: a clinicopathological study. Am J Rhinol. 2006;20:278–82.
- Kennedy DW, Senior BA, Gannon FH, Montone KT, Hwang P, Lanza DC. Histology and histomorphometry of ethmoid bone in chronic rhinosinusitis. Laryngoscope. 1998;108:502–7.
- Hahn S, Palmer JN, Purkey MT, Kennedy DW, Chiu AG. Indications for external frontal sinus procedures for inflammatory sinus disease. Am J Rhinol Allergy. 2009;23:342–7.
- Stewart AE, Vaughan WC. Balloon sinuplasty versus surgical management of chronic rhinosinusitis. Curr Allergy Asthma Rep. 2010;10:181–7.
- 25. Wormald PJ. Endoscopic sinus surgery: anatomy, three-dimensional reconstruction, and surgical technique. 2nd ed. New York: Thieme; 2008.
- 26. Adams AP. Techniques of vascular control for deliberate hypotension during anaesthesia. Br J Anaesth. 1975;47:777–92.
- 27. Petrozza PH. Induced hypotension. Int Anesthesiol Clin. 1990;28:223–9.
- Group PS, Devereaux PJ, Yang H, et al. Effects of extended-release metoprolol succinate in patients undergoing non-cardiac surgery (POISE trial): a randomised controlled trial. Lancet. 2008;371: 1839–47.
- Lindop MJ. Complications and morbidity of controlled hypotension. Br J Anaesth. 1975;47:799–803.
- Groudine SB, Hollinger I, Jones J, DeBouno BA. New York State guidelines on the topical use of phenylephrine in the operating room. The Phenylephrine Advisory Committee. Anesthesiology. 2000;92:859–64.
- 31. Ko MT, Chuang KC, Su CY. Multiple analyses of factors related to intraoperative blood loss and the role of reverse Trendelenburg

position in endoscopic sinus surgery. Laryngoscope. 2008;118: 1687–91.

- Celebi N, Artukoglu F, Celiker V, Aypar U. Repeated attacks of venous air embolism during endoscopic sinus tumor surgery: a case report. Int J Pediatr Otorhinolaryngol. 2005;69:1437–40.
- Barash PG. Clinical anesthesia. 6th ed. Philadelphia: Wolters Kluwer/Lippincott Williams & Wilkins; 2009.
- Brull SJ, Murphy GS. Residual neuromuscular block: lessons unlearned. Part II: methods to reduce the risk of residual weakness. Anesth Analg. 2010;111:129–40.
- 35. Murphy GS, Brull SJ. Residual neuromuscular block: lessons unlearned. Part I: definitions, incidence, and adverse physiologic effects of residual neuromuscular block. Anesth Analg. 2010;111:120–8.
- Murphy GS, Szokol JW, Marymont JH, Greenberg SB, Avram MJ, Vender JS. Residual neuromuscular blockade and critical respiratory events in the postanesthesia care unit. Anesth Analg. 2008;107:130–7.
- Cheng CR, Sessler DI, Apfel CC. Does neostigmine administration produce a clinically important increase in postoperative nausea and vomiting? Anesth Analg. 2005;101:1349–55.
- Ahmed MZ, Vohra A. The reinforced laryngeal mask airway (RLMA) protects the airway in patients undergoing nasal surgery–an observational study of 200 patients. Can J Anaesth. 2002;49:863–6.
- Webster AC, Morley-Forster PK, Janzen V, et al. Anesthesia for intranasal surgery: a comparison between tracheal intubation and the flexible reinforced laryngeal mask airway. Anesth Analg. 1999;88:421–5.
- Atef A, Fawaz A. Comparison of laryngeal mask with endotracheal tube for anesthesia in endoscopic sinus surgery. Am J Rhinol. 2008;22:653–7.
- Pavlin JD, Colley PS, Weymuller Jr EA, Van Norman G, Gunn HC, Koerschgen ME. Propofol versus isoflurane for endoscopic sinus surgery. Am J Otolaryngol. 1999;20:96–101.
- Blackwell KE, Ross DA, Kapur P, Calcaterra TC. Propofol for maintenance of general anesthesia: a technique to limit blood loss during endoscopic sinus surgery. Am J Otolaryngol. 1993;14: 262–6.

- Eberhart LH, Folz BJ, Wulf H, Geldner G. Intravenous anesthesia provides optimal surgical conditions during microscopic and endoscopic sinus surgery. Laryngoscope. 2003;113:1369–73.
- Manola M, De Luca E, Moscillo L, Mastella A. Using remifentanil and sufentanil in functional endoscopic sinus surgery to improve surgical conditions. ORL J Otorhinolaryngol Relat Spec. 2005;67:83–6.
- Cafiero T, Cavallo LM, Frangiosa A, et al. Clinical comparison of remifentanil-sevoflurane vs. remifentanil-propofol for endoscopic endonasal transphenoidal surgery. Eur J Anaesthesiol. 2007;24: 441–6.
- 46. Lee B, Lee JR, Na S. Targeting smooth emergence: the effect site concentration of remifentanil for preventing cough during emergence during propofol-remifentanil anaesthesia for thyroid surgery. Br J Anaesth. 2009;102:775–8.
- 47. Nekhendzy V, Lemmens HJ, Vaughan WC, et al. The effect of deliberate hypercapnia and hypocapnia on intraoperative blood loss and quality of surgical field during functional endoscopic sinus surgery. Anesth Analg. 2007;105:1404–9 [table of contents].
- Friedman M, Venkatesan TK, Lang D, Caldarelli DD. Bupivacaine for postoperative analgesia following endoscopic sinus surgery. Laryngoscope. 1996;106:1382–5.
- 49. Waldman SD. Atlas of interventional pain management. 2nd ed. Philadelphia: Saunders; 2004.
- 50. Douglas R, Wormald PJ. Pterygopalatine fossa infiltration through the greater palatine foramen: where to bend the needle. Laryngoscope. 2006;116:1255–7.
- Mercuri LG. Intraoral second division nerve block. Oral Surg Oral Med Oral Pathol. 1979;47:109–13.
- May M, Levine HL, Mester SJ, Schaitkin B. Complications of endoscopic sinus surgery: analysis of 2108 patients-incidence and prevention. Laryngoscope. 1994;104:1080–3.
- 53. Buchanan MA, Dunn GR, Macdougall GM. A prospective double-blind randomized controlled trial of the effect of topical bupivacaine on post-operative pain in bilateral nasal surgery with bilateral nasal packs inserted. J Laryngol Otol. 2005;119:284–8.
- Church CA, Stewart 4th C, O-Lee TJ, Wallace D. Rofecoxib versus hydrocodone/acetaminophen for postoperative analgesia in functional endoscopic sinus surgery. Laryngoscope. 2006;116:602–6.

Thyroid, Parathyroid, and Parotid Surgery

14

Steven Porter, Andrew Schwartz, Samuel DeMaria, Jr., and Eric M. Genden

Introduction

Surgery of the thyroid, parathyroid, and parotid glands requires a coordinated team of an experienced surgeon and anesthesiologist familiar with the challenges of each procedure. Surgery of the parathyroid, thyroid, and parotid is distinct with regard to the surgical concerns, anesthetic priorities, and postoperative management. Unlike parotid surgery, which is typically performed under general anesthesia, thyroidectomy and parathyroidectomy can be performed under local, regional, or general anesthesia.

Overview

Over the past decade there has been a national trend toward minimally invasive surgical techniques and limited surgical dissection ^[1]. This is particularly true in the case of thyroid and parathyroid surgery. Minimally invasive procedures have enjoyed increasing popularity because there is less surgical dissection, less postoperative pain, and in most cases, decreased surgical time. A successful outcome is dependent

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Department of Otolaryngology – Head and Neck Surgery, Icahn School of Medicine at Mount Sinai, One Gustave L. Levy Place, New York, NY 10029, USA e-mail: eric.genden@mountsinai.org upon strong communication between anesthesiologist and surgeon, efficient surgical technique, and adequate intraoperative control of patient anxiety and discomfort.

In contrast to thyroid and parathyroid surgery, minimally invasive surgical techniques are not typically applied to parotid gland surgery; however, there are equally important sets of concepts that need to be addressed during parotid surgery. The safe identification of the facial nerve and complete excision of the tumor without tumor spill are typically considered the most important concepts ^[2]. The former requires that the patient remain unparalyzed during surgery and the surgical field remain meticulously hemostatic throughout the procedure. Cooperation between the surgeon and the anesthesiologist is essential to achieve both the appropriate levels of anesthesia and blood pressure to maintain a still patient without the use of paralytic. While clinical experience is important for thyroid, parathyroid, and parotid surgery, cooperation and communication between the surgeon and anesthesiologist are essential for optimal outcomes, particularly in complex cases.

Thyroid Surgery

Introduction

Surgery on the thyroid gland is a common operation performed on a daily basis in many hospitals. A thorough discussion of preoperative thyroid-specific labs and radiologic assessment of the thyroid gland should be completed prior to the development of an anesthetic plan. This section will describe an overview of the thyroid gland and its function, preoperative assessment of thyroid disorders, intraoperative management of the patient with thyroid disease, a discussion of thyrotoxicosis and myxedema coma, and postoperative complications for which practitioners should be vigilant.

Overview

The Thyroid Gland

Basic Anatomy

The thyroid gland is a butterfly-shaped gland with two lateral lobes connected inferiorly by a central isthmus (Fig. 14.1). The blood supply of the thyroid gland is derived from the superior and inferior thyroid arteries, which are branches of the external carotid artery and thyrocervical trunk, respectively. The inferior thyroid artery as it approaches the gland is in close proximity to the recurrent laryngeal nerve. The recurrent laryngeal nerve is a branch of the vagus nerve and serves as the main source of motor function to the larynx. It courses in the trache-oesophageal groove and enters the larynx at the cricothyroid joint. It is in this area where care must be taken during thyroidectomy to avoid inadvertent injury to the nerve. The superior laryngeal nerve on the other hand provides sensory innervation to the larynx and tenses the vocal cords to allow alteration in pitch through its motor supply to the cricothyroid muscle.

Basic Physiology

The thyroid gland is a vascular, ductless gland located anterior to the trachea. The major function of the gland is the synthesis, storage, and secretion of thyroid hormones. The hypothalamic-pituitary-thyroid axis is responsible for negative feedback control of thyroid hormone synthesis and secretion (Fig. 14.2). Thyrotropin-releasing hormone (TRH) is produced and released by the hypothalamus. TRH is transported via the blood to the anterior pituitary gland where it leads to the release of thyroid-stimulating hormone (TSH). Activation of the thyroid gland's TSH receptors stimulates the production and secretion of triiodothyronine (T3) and tetraiodothyronine (T4). TSH also stimulates thyroid gland growth and development. Through negative feedback, T3 and T4 inhibit the release of both TRH and TSH. Somatostatin, glucocorticoids, and dopamine also suppress TSH synthesis and secretion.

Synthesis of thyroid hormones is a complex process. Thyroglobulin (Tg), a glycoprotein contained in the thyroid gland, is used as a scaffold for thyroid hormone production. Tg contains multiple tyrosine residues that are iodinated in

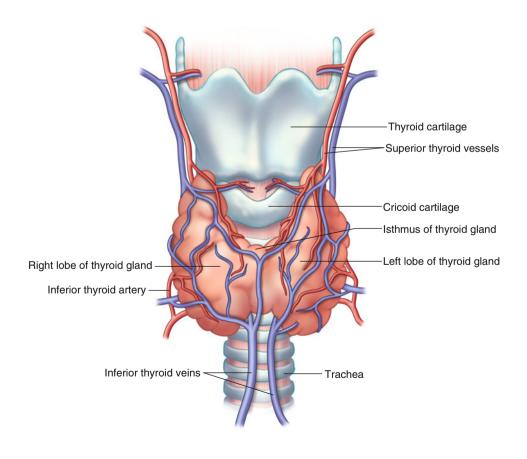


Fig. 14.1 Thyroid gland anatomy and blood supply

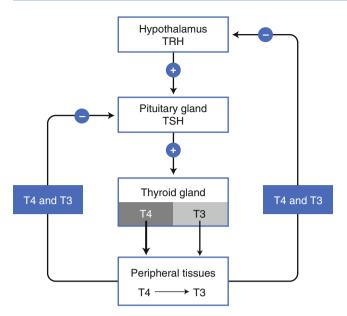


Fig. 14.2 Hypothalamic-pituitary-thyroid axis

the process of thyroid hormone synthesis. These residues are coupled to form T3 and T4. The process of thyroid hormone synthesis requires iodide, which is obtained from dietary sources. The thyroid gland secretes T4 and T3 in a 10:1 ratio. However, T3 is the biologically active hormone, with receptor binding affinity 100 times that of T4. Most of the T3 in circulation is from peripheral deiodination of T4 by the liver, kidney, and skeletal muscle. The majority of circulating thyroid hormone is bound to thyroid binding globulin.

Thyroid hormone is involved in growth, development, and metabolism. It regulates biochemical reaction speed and metabolic rate and therefore oxygen consumption. Thyroid hormone receptors are ubiquitous, located in virtually every organ in the body. Their effects are primarily mediated via transcriptional regulation of target genes. Thyroid hormone acts as a chronotrope and an inotrope. It leads to increased cardiac output and decreased systemic vascular resistance. It also helps regulate triglyceride and cholesterol metabolism in the liver.

Hypothyroidism

Hypothyroidism occurs when there is a decrease in circulating thyroid hormone levels. This can result from a reduction in thyroid hormone synthesis at the level of the thyroid gland or anywhere along the hypothalamic–pituitary–thyroid axis. Congenital hypothyroidism that occurs *in utero* results in severe mental retardation and developmental delay, termed cretinism. Adult-onset hypothyroidism may be primary (disease of thyroid gland) or secondary (decreased TSH secretion). Primary hypothyroidism is the most common form and is more prevalent in women ^[3]. The most common cause of primary hypothyroidism is Hashimoto's thyroiditis (chronic

General	Fatigue, weight gain, anemia, cold intolerance
Dermatologic	Dry coarse skin, brittle hair, hair loss, nonpitting peripheral edema
Ears, eyes, throat	Hearing loss, hoarse voice, periorbital edema, facial puffiness, goiter
Pulmonary	Dyspnea, pleural effusions, hypoventilation, sleep apnea
Cardiac	Bradycardia, congestive heart failure, pericardial effusions
Gastrointestinal	Anorexia, constipation
Genitourinary	Menstrual disorders, decreased libido, impotence, infertility
Neuromuscular	Muscle weakness, delayed ankle jerk relaxation phase
Psychiatric	Depression, psychomotor retardation, coma

 Table 14.1
 Manifestations of hypothyroidism

 Table 14.2
 Cardiovascular risks associated with hypothyroidism ^[6]

Impaired cardiac contractility and diastolic function
Increased systemic vascular resistance
Decreased endothelial-derived relaxation factor
Increased serum cholesterol
Increased C-reactive protein
Increased homocysteine

autoimmune thyroiditis). Typical signs and symptoms result from a global decrease in metabolic activity and are presented in Table 14.1.

Patients can be lethargic and intolerant of cold. Significant cardiovascular changes can be the earliest clinical manifestation and include decreased stroke volume, decreased heart rate, decreased cardiac output, and decreased contractility ^[4]. Cardiovascular risks associated with hypothyroidism are presented in Table 14.2.

Paradoxically, patients are often hypertensive secondary to increased systemic vascular resistance from increased levels of circulating catecholamines. Patients may also present with pleural and/or pericardial effusions. Severely hypothyroid patients can manifest classic signs such as: coarse, scaly skin; sparse hair; nonpitting edema of the hands, feet, and periorbital areas; hoarseness; and delayed deep-tendon reflexes.

Hyperthyroidism

Hyperthyroidism results from increased activity of the thyroid gland resulting in excess circulating levels of thyroid hormone. Patients have disturbances in metabolism and oxygen consumption leading to a variety of signs and symptoms (Table 14.3).

Anxiety, diaphoresis, muscle weakness, and heat intolerance are common symptoms. Goiter and ophthalmopathy

Ta	ble	14	.3	Signs and	l symptoms c	of hype	rthyroidism ^[41]
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Signs	Symptoms
Hyperactivity	Nervousness
Tachycardia	Fatigue
Systolic hypertension	Weakness
Warm, moist, smooth skin	Perspiration
Eyelid retraction	Heat intolerance
Tremor	Palpitations
Hyperreflexia	Increased appetite
Muscle weakness	Weight loss

	Table 14.4	Cardiovascular	signs and	symptoms	of hyperthyroidism
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Palpitations	Anginal chest pain
Exercise intolerance	Atrial fibrillation
Exertional dyspnea	Cardiac hypertrophy
Systolic hypertension	Peripheral edema
Hyperdynamic precordium	Congestive heart failure

(particularly in Graves' disease) are common signs. As with hypothyroidism, a myriad of cardiovascular problems occur (Table 14.4).

Patients can present with tachycardia, atrial fibrillation, palpitations, and angina. Thyroid hormones have a direct effect on vascular smooth muscle leading to decreased SVR and blood pressure ^[5]. The renin–angiotensin–aldosterone system is activated leading to a marked increase in circulating blood volume and cardiac output ^[6]. Volume overload can lead to congestive heart failure (CHF). Hyperthyroidism, like hypothyroidism, is more common in women than men. Hyperthyroidism may be secondary to benign or malignant processes. The majority of cases of hyperthyroidism are caused by Graves' disease, followed by multinodular goiter, or toxic adenoma.

Although rare, it is important to note that medullary thyroid cancer is associated with multiple endocrine neoplasia (MEN) types 2a and 2b. In such cases the practitioner must be aware of the possibility of an associated pheochromocytoma. Anesthetizing a patient with an unrecognized pheochromocytoma can be associated with significant morbidity and mortality and such a diagnosis should be entertained in the presence of unexpected or uncontrolled hypertension and tachycardia.

Preoperative Considerations

Otolaryngologist's Perspective

Thyroid surgery is performed in a number of circumstances including management of thyroid cancer, a symptomatic thyroid goiter, Graves' disease, or hyperthyroidism, or for Table 14.5 Risk factors for thyroid malignancy

Age <20 and >60 years	Family history of thyroid carcinoma
Male	Fixation of nodule to adjacent tissue
Rapid growth, pain	Vocal cord paralysis
History of radiation therapy	Size >4 cm
Associated Lymphadenopathy	Aerodigestive tract extension

diagnostic purposes in the event that a thyroid nodule may be suspicious for carcinoma. The risk factors for malignancy in the thyroid nodule are shown in Table 14.5.

The diagnostic evaluation for the thyroid nodule consists of three main areas: thyroid function tests, imaging, and fine needle aspiration of the nodule. It is important to determine, through history, physical and laboratory examinations (TSH level) that the patient is euthyroid *prior* to surgery. Any irregularity in thyroid function may result in intraoperative and postoperative complications and dramatically increases morbidity and mortality.

Ultrasound is the best initial imaging study for the thyroid. It provides information on nodule size, content, and associated cervical lymphadenopathy. A limitation of ultrasound is in patients with substernal extension of the thyroid where a CT scan of the neck and chest will provide more detailed information on extent. In patients with hyperthyroidism, iodine-based contrast should be avoided since it will exacerbate their condition and possible even precipitate a thyroid storm, thus a non-contrast study or magnetic resonance imagining (MRI) with contrast is preferred.

In general, the rate of thyroid surgery has been rising dramatically over the past decade. The most common indication for thyroid surgery is management of the thyroid nodule. After serum evaluation and imaging, fine needle aspiration of the nodule is indicated. The American Thyroid Association has standard guidelines for the management of thyroid nodules. Fine needle aspiration is the procedure of choice for thyroid nodule evaluation. The possible results obtained from nodule aspiration are: benign, follicular neoplasm, Hurthle cell neoplasm, diagnostic or suspicious for malignancy, and nondiagnostic. An ultrasound-guided aspiration is associated with a lower false negative rate and nondiagnostic result, thus it is recommended in those nodules that are cystic, nonpalpable, or located posteriorly in the gland, and in any case where the initial aspiration result was nondiagnostic. In cases where persistent nondiagnostic results are obtained, close observation for nodule change or surgery is recommended as up to 7% of these patients may be harboring an occult malignancy.

Depending on the indication for surgery, there are several preoperative considerations that should be taken into account prior to thyroidectomy. In the event that a thyroidectomy is being performed for malignancy, it is important to define the extent of disease. Large thyroid cancers can encroach on the tracheal airway leading to tracheal deviation and possibly, airway obstruction ^[7]. Aggressive forms of the disease can erode into the larynx, trachea, or esophagus. If these conditions are not appreciated prior to surgery, induction of anesthesia can result in a sudden airway obstruction or uncontrollable hemorrhage.

When thyroidectomy is performed for hyperthyroidism, thyroid goiter is not uncommon. A thyroid goiter can distort the tracheal airway making intubation challenging. In longstanding goiter, extubation following surgery can be complicated by tracheomalacia and airway obstruction. In the case of active Grave's disease, a thyroid storm may ensue during the course of surgery leading to tachycardia, hypertension and in severe cases, cardiovascular decompensation. Preoperative communication between the surgeon and the anesthesiologist is important in recognizing these conditions and the potential risks associated with surgery. In these cases, planning for an awake fiberoptic intubation is important as is preparation for emergent tracheostomy if airway loss or obstruction occurs.

Anesthesiologist's Perspective

Surgical removal of the thyroid gland (partial or total thyroiddectomy) can be the definitive treatment for hyperthyroidism, especially in patients who have failed medical therapy or who are intolerant to medical management, or for malignant disease. Preoperative assessment for the patient presenting for thyroid surgery should begin with a focused history and physical exam. Particular attention should be given to the airway and neck examination. Patients may present with large goiters. These can lead to tracheal deviation, tracheal compression, and stridor. Radiologic assessments (ultrasound, computed tomography) are useful in determining the extent of airway disturbance by the mass of the goiter (Chap. 2). A discussion with the surgeon is important as they may have the most recent radiologic examinations and shed light on any worsening in the airway.

The laboratory evaluation of thyroid disease focuses on the evaluation of TSH and thyroid hormone levels. Physiologic changes (e.g., hepatic disease, pregnancy) can alter the amount of thyroxine-binding globulin, thus altering the amount of total hormone in the blood. As most labs can now assess the level of free T4 directly, older tests such as the resin T3 uptake have been made unnecessary.

Typical surgical positioning will involve neck extension and shoulder roll placement, therefore reviewing studies and/ or querying patients regarding cervical pathology is important. Neck range of motion should be assessed. Any abnormalities should be documented.

Rarely, one or both thyroid lobes enlarge and grow through the thoracic inlet, resulting in a substernal goiter. These goiters, in particular, can lead to severe airway obstruction that is often worse with supine positioning. Such obstructive goiters may cause other symptoms such as recurrent laryngeal nerve compression. This may lead to hoarseness that should be documented preoperatively. Flow-volume loops may be helpful in quantifying the extent of airway obstruction. Flow-volume loops may be abnormal even when the patient is asymptomatic or no evidence of tracheal compression is noted radiologically ^[8]. Also, entrance into the mediastinum, if planned, needs to be communicated to the anesthesiology team so preparations can be made (e.g., arterial line, larger IV access for a thoracic case).

As mentioned above, an euthryoid state should be achieved if surgery is to be performed. It is therefore incumbent upon the anesthesiologist to review thyroid function tests in the preoperative period. Many medications can be used to render the patient euthyroid. The antithyroid medications, methimazole and propylthiouracil, inhibit thyroperoxidase. Thyroperoxidase normally facilitates the addition of iodine to tyrosine residues on thyroglobulin, a key step in the formation of both T3 and T4. Thus, these medications block the synthesis of thyroid hormone. Propylthiouracil also blocks the conversion of T4 to T3 outside of the thyroid gland. β -Blockade helps to attenuate the sympathomimetic state and, like propylthiouracil, has the additional benefit of decreasing the peripheral conversion of T4 to T3. Although seemingly counterintuitive, iodide in high concentration transiently inhibits thyroid hormone secretion, known as the Wolff-Chaikoff effect. It can be administered in the form of a saturated solution of potassium iodide or as the contrast dye iopanoic acid.

All current patient medications should be continued in the perioperative period. Elective surgery should be postponed in the patient with uncontrolled hyperthyroidism until a therapeutic medical regimen is established to reduce the risk of intraoperative thyroid storm ^[9]. Many medications can alter thyroid hormone synthesis and warrant a review of pertinent thyroid lab studies (Table 14.6).

Table 14.6 Drugs affecting thyroid function ^[7]

Drug name	Target organ effect site
PTU	Thyroid
Methimazole	Thyroid
Radiographic contrast agents	Thyroid
Amiodarone	Thyroid, liver, brain
Lithium	Thyroid
Salicylates	Blood (transthyretin)
Furosemide	Blood (transthyretin)
Propranolol	Liver, peripheral β-blockade
Ferrous sulfate	Small intestine
Phenobarbital	Liver
Dexamethasone	Liver

Preoperative assessment of the cardiac conduction system via an electrocardiogram is important for patients with hyperthyroidism. Sinus tachycardia is the most common cardiac rhythm disturbance, but atrial fibrillation is present in up to 20% of patients with hyperthyrodism ^[6]. Treatment of atrial fibrillation is usually accomplished via rate control with β -blockade. A normal resting heart rate is helpful in determining if the patient has been medically optimized for the surgical procedure.

Hyperthyroid patients may have congestive heart failure. This may seem counterintuitive given the fact that these patients have an increased cardiac output. Chronic hyperthyroidism with sinus tachycardia or atrial fibrillation can produce rate-related left ventricular dysfunction and heart failure ^[10]. The history and physical exam should focus attention to the signs and symptoms of CHF (dyspnea, orthopnea, rales). Elective thyroid surgery should be deferred in the patient with symptomatic CHF until successful medical optimization. An echocardiographic examination often proves useful in examining left ventricular function. More invasive monitoring (such as arterial and/or central venous catheterization) may be warranted in the patient with active, symptomatic CHF.

Intraoperative Considerations

Otolaryngologist's Perspective

Thyroidectomy may be performed under local, regional, or general anesthesia ^[11]. Determining the most appropriate anesthetic is predicated on the medical condition of the patient, the patient's anatomy, and patient preference. Irrespective of the choice for anesthesia, the patient is typically placed in the supine position, arms tucked against the body, and the neck gently extended usually with a small shoulder roll (Fig. 14.3a, b). Figure 14.3c–e shows the sequence of prepping and draping prior to thyroid and para-thyroid surgery.

This position allows for palpation of the thyroid and cricoid cartilages that can be marked out so a transverse incision can be planned on the anterior neck. To minimize soft tissue swelling, we prefer that a dose of intravenous steroids be administered at least 15 min before the incision is made. The ether drape is placed so that the anatomy below the mandible is accessible. To secure this position, we commonly use an adhesive drape along the chin to maintain the drape position. Regardless of the anesthetic technique, we prefer an injection of subcutaneous lidocaine with 1% epinephrine at a dilution of 1:100,000 to control incision bleeding.

Thyroidectomy for unilateral nodules, moderately sized goiters, and in most cases of early stage thyroid malignancy

can be performed utilizing a minimally invasive approach. The size of the incision should be tailored to the size of the gland, the need for an adjunctive neck dissection, or the presence of substernal extension. In general, the platysma layer is divided with subplatysmal skin flaps raised superiorly to the hyoid bone and inferiorly to the sternal notch. The strap musculature over the thyroid gland is retracted laterally exposing the gland itself (Fig. 14.4).

In cases of malignancy, tumor may extend beyond the gland capsule and these muscles will need to be resected. Once the gland is exposed the middle thyroid vein may be divided in order to medialize the gland. The superior pole of the gland is then released by clamping the superior pole vessels with a stitch or surgical clips. Dissection is then performed intimately along the gland itself thus reflecting the parathyroid glands and their blood supply laterally. The recurrent laryngeal is identified and traced up to the cricothyroid joint where it enters the larynx (Fig. 14.5).

If this nerve is involved with tumor and the patient has normal preoperative vocal cord movement, all attempts are made to preserve the nerve. In unilateral nodules, the affected side is removed and the isthmus ligated. A harmonic scalpel is used in our institution to aid in hemostasis and to help expedite the surgical procedure.

The excised gland is evaluated by pathology for the presence of malignancy. If the nodule is consistent with carcinoma, the remaining contralateral gland is excised and removal of paratracheal lymph nodes is performed. This does not require a reversal of paralysis, if used. This is important information for the anesthesiologist. In cases of known malignancy and positive lymph nodes located in the lateral neck, a neck dissection is necessary and reversal of paralysis will be necessary. This information will need to be communicated to the anesthesiologist.

Depending on the indication for surgery, the surgeon is often able to determine if the laryngeal or tracheal airway will be affected during the course of surgery or on extubation. In the event of malignancy, tracheal airway invasion may require a tracheal resection and reconstruction. In some cases, the airway reconstruction can be accomplished with minimal risk however in the event of extensive disease or tracheomalacia, careful attention to detail during extubation is necessary to avoid airway compromise. In certain cases of extensive tracheal involvement, a tracheostomy may be necessary with delayed reconstruction when feasible.

The tracheal airway may not be invaded, yet vocal cord paralysis may affect the airway in some patients ^[12]. In some cases, the surgeon may be aware of a vocal paralysis, however in other cases a paralysis may not be identified until the patient becomes symptomatic following extubation. In the case of a preoperative unilateral paralysis, patients may compensate quite well and remain asymptomatic. In the case of acute paralysis during surgery, however, the patient may become Fig. 14.3 (a) Proper surgical positioning with shoulder roll and foam "donut" for thyroid and/or parathyroid exposure.
(b) Proper anesthesia positioning with wire reinforced endotracheal tube and anesthesia circuit positioned to track above and over the patient's face. The monitors have been placed on the patient's back. (c–e) Patient for thyroidectomy, sterile prep and drape in place



stridorous and decompensate rapidly upon extubation. In addition, aspiration may develop due to the inability of the contralateral vocal cord to compensate and protect the larynx. This is particularly common in elderly patients or patients with compromised pulmonary function. In the case of bilateral vocal paralysis, nearly all patients become stridorous and symptomatic soon after extubation. This situation may require an emergent tracheostomy and at the very least close postoperative monitoring for airway compromise. If there is any question as to vocal cord function, a bedside flexible laryngoscopy may be performed to confirm whether a true vocal cord paralysis or possible obstructive airway edema are present.

In the event of an extensive longstanding goiter or substernal goiter, the tracheal wall may become weak or thinned resulting in tracheomalacia. This can lead to tracheal airway collapse and obstruction during extubation. In extreme cases, a tracheotomy may be required. This can often be determined at the time of surgery so that the surgical and anesthesia teams can be prepared for management of the airway in the event of an obstruction. Substernal goiters may require a larger incision; however, it is rare for a sternal split to be necessary in these cases. A cardiothoracic surgeon can be on standby and communication with the anesthesiologist is critical if there will be a possibility of a sternal split since this may require an adjustment in anesthetic technique and monitoring.

Anesthesiologist's Perspective

Most patients presenting for thyroid surgery will tolerate an intravenous induction of general anesthesia without event. Patients with significant compression of their trachea by thyroid masses should be considered for an awake fiberoptic intubation (Chap. 8). If there is a concern that airway collapse may occur even in the presence of a secured endotracheal tube, then a rigid bronchoscope should be available for use by the surgeon. There are rarely concerns for significant blood loss during this operation, unless there is a substernal thyroid requiring a sternal split hence, large bore venous access is generally not necessary. Intra-arterial catheterization is also generally not indicated for this surgery, assuming no patient-

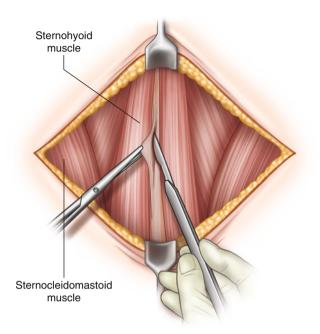


Fig. 14.4 Initial surgical approach to the thyroid gland

specific comorbidities necessitate invasive monitoring. However, intra-arterial catheterization is warranted in the case of surgery for the substernal thyroid, especially if there is a high likelihood of sternal split. Although the majority of these tumors can be excised via the traditional cervical approach, occasionally a sternotomy is required.

In most situations the patient's head will stay toward the anesthesiologist, however, in cases scheduled for unilateral neck dissections, a 90° turn placing the operative side out toward the room away from the anesthesiologist may be used. A 180° turn should be anticipated and planned for in cases scheduled for bilateral neck dissections. As with all cases, it is critical to discuss patient positioning preoperatively in order to plan airway management, intravenous, non-invasive, and invasive monitor placement.

Although the vast majority of thyroid surgeries are performed under general anesthesia, regional anesthesia has been used as well. Deep and superficial cervical plexus blockades have been effectively utilized as the sole anesthetic for thyroid surgery, but are more commonly employed as adjuncts to general anesthesia. Prior to the administration of cervical blockade, communication with the surgeon is necessary to confirm that the extent of thyroid disease does not involve the region of block placement. This is critical in cases with a large goiter or thyroid malignancy so as not to violate the tumor capsule or clinically suspicious cervical lymph nodes. Figure 14.6 depicts the anatomy of the cervical plexus.

Bilateral superficial cervical plexus (SCP) blockade has been shown to significantly reduce postoperative pain as compared to placebo ^[13]. SCP blockade is performed in the

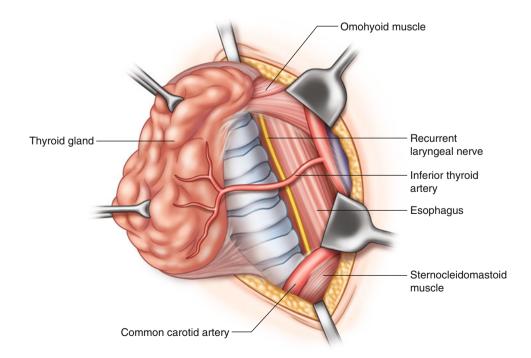
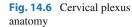
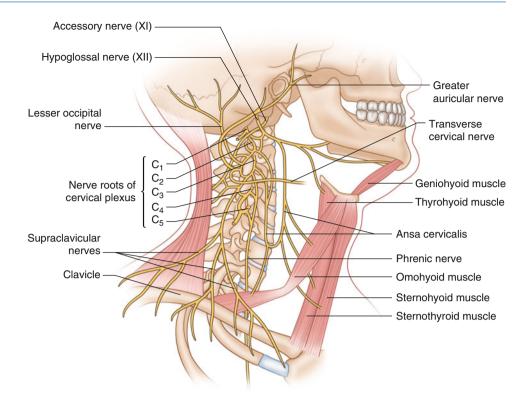


Fig. 14.5 Exposure of left thyroid and major neurovascular structures





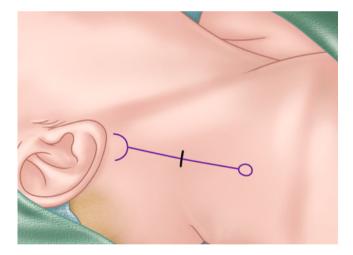
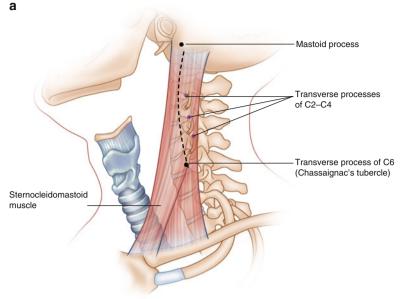


Fig. 14.7 Surface landmarks for the superficial cervical block

supine position with the patient's head turned away from the side being blocked. The midpoint of the posterior border of the sternocleidomastoid muscle is identified and local anesthetic is administered 1-2 cm at an angle perpendicular to the skin (Fig. 14.7).

For deep cervical plexus (DCP) block, a line is drawn from the mastoid process to the prominent turbercle of C6 (Fig. 14.8a and b). The transverse processes of C2, 3, and 4 lie at or near this line at 1.5 cm intervals below the mastoid process. The needle is advanced posteriorly until contact is made with the transverse process, withdrawn 1-2 mm, and then local anesthetic deposited at each site ^[14] Since DCP blocks will anesthetize the phrenic nerve, it should be used with caution in patients with significant pulmonary disease and bilateral DCB should never be done. We recommend placing bilateral SCP blocks along with a unilateral DCP block on the side of the enlarged thyroid if that exists.

Standard premedications can be administered. Midazolam (0.01–0.03 mg/kg) and/or fentanyl (0.5–2 mcg/kg) and/or remifentanil (0.02-0.05 µg/kg/min) are typical for our institution. After application of standard American Society of Anesthesiologists monitors, general anesthesia is typically induced with propofol (1.5-2.5 mg/kg). At the time of writing this text, thiopental is commercially unavailable in the USA; however an induction with thiopental offers a theoretic advantage as its thiourea structure has some antithyroid activity, although a significant effect is unlikely with a single induction dose^[15]. Ketamine is best avoided in hyperthyroid patients secondary to its sympathomimetic activity. Muscle relaxants frequently aid in tracheal intubation, but specific surgical conditions may preclude their use (see below). Pancuronium is avoided in hyperthyroid patients secondary to its sympathetic nervous system stimulating effects as well as a duration of action that may extend beyond the end of surgery. When we are not employing intraoperative recurrent laryngeal nerve monitoring, we recommend the use of a reinforced anode endotracheal tube for intubation. This type of tube is unlikely to kink or be compressed during surgical manipulation or patient positioning, and also can be placed



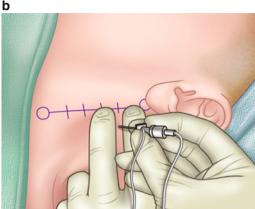


Fig. 14.8 Anatomical (a) and Surface (b) landmarks for the deep cervical plexus block

over the head to avoid interfering with the surgical field. Eye protection is essential in all patients but special care must be given to patients with proptosis or other ophthalmopathy.

The use of persistent neuromuscular blocking agents should be avoided in cases that include peripheral neck dissections in patients with known malignancy or during cases with planned intraoperative recurrent laryngeal nerve monitoring. Recurrent laryngeal nerve (RLN) injury is a known complication of thyroid surgery (see postoperative complications). As a potential monitor for RLN damage, intraoperative neural monitoring (IONM) during thyroid surgery is being increasingly utilized. This may be accomplished by the surgeon palpating the cricoarytenoid joint while electrically stimulating the nerve, by electrophysiological nerve monitoring, with direct monitoring of vocal cord movement with direct laryngoscopy while stimulating the nerve, or by the visualization of vocal cord movement via a fiberoptic bronchoscope placed through a larvngeal mask airway. It is critical for the anesthesiologist and surgeon to discuss monitoring prior to the procedure.

Many nerve-monitoring systems are available. The most commonly used electrophysiological equipment relies on endotracheal tube-based surface electrodes ["nerve integrity monitor" (NIM[®]) endotracheal tube (NIM[®] EMG Endotracheal Tube, Medtronic Xomed Surgical Products, Jacksonville, FL)] ^[16]. These tubes contain embedded electrodes that are placed in contact with the bilateral vocal cords (Figs 14.9 and 14.10).

These are available in typical endotracheal tube sizes, but have external diameters that are significantly larger than standard tubes (e.g., if inner diameter=6.0 mm, then outer

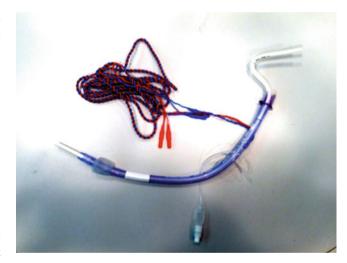


Fig. 14.9 Surface-embedded electrode endotracheal tube with an intubating stylette to improve intubation success

diameter = 8.8 mm). Although it is recommended that patients should be intubated using the largest tube that can be safely placed in order to optimize electrode and vocal cord contact, given the size of the O.D. diameter we routinely use a 6.0 ID tube for most patients. Tube lubrication is avoided as it may alter the signal. Preoperative glycopyrrolate may also be advantageous to decrease saliva and improve monitoring signals. Care should be taken to ensure the tube is positioned without rotation to ensure proper bilateral electrode-vocal cord contact. Post-positioning preincision tube position verification has been recommended.



Fig. 14.10 Patient intubated with a surface-embedded endotracheal tube

Due to the size and potential difficulty in placing tubes with embedded electrodes, several companies have developed adhesive electrodes that can be placed on standard endotracheal tubes (Fig. 14.11). Our group has recently switched to these devices with a high degree of satisfaction and decreased intubation difficulties.

IONM involves electromyographic recordings. As such, the anesthesiologist needs to be aware of the various impacts that medications have on these recordings. EMG is very sensitive to the effect of neuromuscular blocking agents and these medications are ideally avoided. If these drugs are deemed necessary for intubation, it is essential to utilize succinylcholine or a small dose of a non-depolarizing drug in order to have prompt return of muscle activity ^[17]. In the case of an unknown pseudocholinesterase deficiency, succinylcholine use will invalidate the EMG recordings, although the lack of reappearance of twitches with a nerve stimulator will clue the anesthesiologist in on this problem ^[18].

The majority of thyroid surgeries are performed with an endotracheal tube. However, the laryngeal mask airway (LMA) has also been used. The use of the LMA offers some advantages over the use of an endotracheal tube. It likely obviates the need for muscle relaxant and reversal medications. Its use decreases the sympathetic nervous system stimulation caused by laryngoscopy, intubation, and extubation. The LMA, being a supraglottic device, also allows direct visualization of the vocal cords using a fiberoptic bronchoscope. This technique allows for real-time monitoring of vocal cord mobility in response to stimulation of the RLN ^[19]. The major disadvantage is an unprotected airway with increased risk of aspiration. The LMA may become dislodged and malpositioning is more common as compared to a well-secured endotracheal tube. LMA rotation leading to stridor has also been reported using this technique^[13].

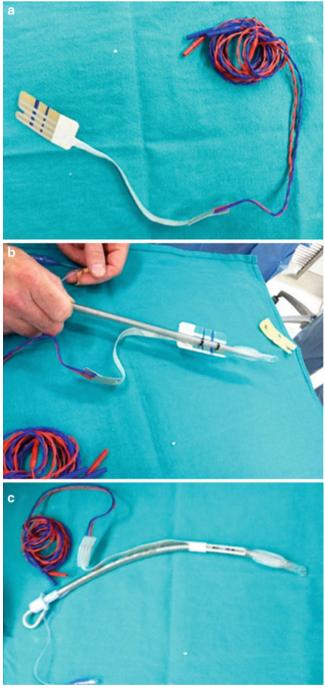


Fig. 14.11 (a) Adhesive electrode system for use on standard endotracheal tubes. (b and c) Adhesive electrodes being placed on a standard anode tube

Intraoperatively, general anesthesia can be maintained with a variety of agents (volatile anesthetics, nitrous oxide, hypnotics, opioids, etc.). The minimum alveolar concentration (MAC) is not affected by hyperthyroidism. Muscle relaxants, when used, should be titrated to effect using a peripheral nerve stimulator. Co-existing muscle disease (myasthenia gravis) and skeletal muscle weakness secondary to hyperthyroidism itself are not uncommon and patients may exhibit reduced non-depolarizing muscle relaxant requirements^[20]. Although a balanced anesthetic with inhaled anesthetic is acceptable, our group prefers to use a remifentanil nitrous narcotic technique with a low infusion dose of supplemental propofol as described in Chaps. 12 and 13 for its reliable smooth emergence.

Regardless of the anesthetic, avoiding "light" anesthesia is essential. Although surgical stimulation tends to be minimal, manipulation of tissue planes on or close to the trachea can cause marked sympathetic outflow. Intraoperative avoidance of drugs that stimulate the sympathetic nervous system is a prudent strategy. Therefore, a direct-acting vasopressor (phenylephrine) is the first-line drug therapy for intraoperative hypotension. All patients should receive intraoperative dexamethasone (10 mg), which has been shown to reduce postoperative nausea and vomiting (PONV), decrease surgical site inflammation, decrease postoperative pain, and improve early postoperative voice function following thyroidectomy ^[21]. It is standard to administer ondansetron to further decrease PONV. Droperidol can be used as a rescue antiemetic in the recovery unit or as prophylaxis in patients with a history of severe PONV. Reversal of muscle relaxation is prudent if administering paralytics.

Prior to emergence the surgeons will typically request that a Valsalva maneuver be performed to confirm hemostasis. If the anesthetic depth was prematurely reduced, the sustained high airway pressures may induce bucking, in addition the maneuver itself may cause severe bradycardia, while subjecting the patient to risk of barotrauma. Besides watching for cardiovascular and pulmonary compromise, we recommending maintaining an adequate depth of anesthesia and only titrate the depth after the Valsalva maneuver has been accomplished and surgical closure has commenced.

A smooth emergence and extubation is critical as coughing and "bucking" can lead to suture line tearing and hematoma formation with accompanying morbidity and mortality. This can be accomplished by deep extubation or, in our experience, a nitrous oxide/propofol/remifentanil technique. The low-dose propofol infusion and nitrous oxide can be maintained and shut off upon dressing placement and after the surgical prep has been wiped from the patient. Wiping the anterior neck and trachea can be quite stimulating and could initiate bucking. The remifentanil infusion is kept on at a dose of 0.05–0.1 mcg/kg/min until eye opening or when the end-tidal nitrous oxide is less than 10%, whichever comes first. We frequently encounter awake patients who are following commands and have no reaction to the endotracheal tube under these wake-up conditions. The use of laryngeal tracheal anesthesia (LTA) at intubation also helps to decrease coughing on emergence.

Thyrotoxicosis and Thyroid Storm

Although exceedingly rare, every anesthesiologist needs to be capable of managing a patient presenting with perioperative thyrotoxicosis (1.4 per 1,000 in women between the ages of 35-60). In the case of emergent surgery in the setting of partially treated or untreated hyperthyroidism, the patient should be treated with intravenous β -blockers titrated to a heart rate less than 90. Esmolol (0.5 mg/kg bolus followed by an infusion at 100–300 µg/kg/min) is a common approach. Oral, rectal, or nasogastric antithyroid therapy (methimazole or PTU) should be administered, although the gradual effect of the drug usually requires weeks of therapy. Sodium ioponate (500 mg) can be given, but must be preceded by antithyroid therapy for 2-3 h [22]. Glucocorticoids (dexamethasone 0.2-1 mg/kg up to 8-10 mg IV every 6-8 h) help to reduce peripheral conversion of T4 to T3. Invasive monitoring of hemodynamics with an arterial line is recommended.

Thyroid storm is the most serious perioperative complication involving the thyroid gland. This is a potentially lifethreatening hypermetabolic state most common in Graves' disease. Triggers include infection or the stress response to surgery. Not all thyrotoxicosis is Graves' disease, however, and the vigilant physician must also keep in mind other clinical scenarios that may engender this state such as toxic adenoma, toxic multinodular goiter, subacute thyroiditis, and silent thyroiditis. Thyroid storm usually occurs intra- or postoperatively up to two days. The clinical symptoms include hyperthermia, tachycardia, and cardiovascular collapse. The differential diagnosis includes malignant hyperthermia, neuroleptic malignant syndrome, inadequate anesthesia (intraoperatively), and pheochromocytoma. The diagnosis is clinical and, if suspected, empiric treatment should begin promptly. The patient should be actively cooled and dehydration managed with glucose-containing intravenous fluids. β -Blocker therapy should be instituted to reduce the heart rate. Propranolol is the β -blocker of choice since it also prevents conversion of T4 to T3. Antithyroid medications (PTU 200-400 mg) via nasogastric tube should be considered. Iodide preparations are also indicated. Dexamethasone (0.2-1 mg/kg up to 8-10 mg IV every 6-8 h)helps to decrease peripheral conversion of T4 to T3. The mortality rate for thyroid storm can be as high as 75%. As such, these patients should be taken to the intensive care unit for further treatment and monitoring ^[3].

Myxedema Coma

Myxedema coma, also exceedingly rare, deserves to be mentioned as well. A severe form of decompensated hypothyroidism, this disease state tends to occur in symptomatic

collia		
	Hypothermia	Infections
	Stroke	Congestive heart failure
	Drugs (narcotics, tranquilizers, amiodarone)	Trauma
	GI bleeding	Metabolic disorders (hypoglycemia, hyponatremia, hypercalcemia)

 Table 14.7 Precipitating and exacerbating factors for myxedema coma [7]

hypothyroid patients who have been exposed to cold weather, infection, systemic disease, or drugs (see Table 14.7).

Most common in older women, the clinical course is one of fatigue progressing through stupor to coma with concomitant respiratory failure. These patients tend to display the classic signs of hypothyroidism noted above. Treatment should be initiated in an intensive care unit and usually includes: ventilatory support to correct hypoventilation and respiratory acidosis; correction of metabolic abnormalities (hyponatremia, hypoglycemia); slow re-warming to normothermia; intravenous glucose-containing fluid therapy to correct hypotension; and administration of exogenous thyroid therapy (intravenous levothyroxine 250–500 mg) with careful monitoring of the cardiovascular response. Vigilance for concomitant adrenal insufficiency is also important.

Postoperative Considerations

Otolaryngologist's and Anesthesiologist's Perspectives

Airway compromise represents the most significant, lifethreatening complication in the immediate postoperative period after thyroidectomy. However, due to the proximity of the parathyroid glands, signs and symptoms of hypocalcemia must also be considered.

Airway Emergencies and RLN Damage

Airway compromise can result from a variety of reasons including airway invasion, vocal cord paralysis, and tracheomalacia; however, postoperative bleeding can occur suddenly and without warning in the immediate postoperative period and can lead to a dramatic and life-threatening loss of airway from direct tracheal compressive from the hematoma along with and venous and lymphatic compression resulting in impressive airway edema. Due to the proximity of the recurrent laryngeal nerve during dissection, transient and rarely permanent paralyses are also a potential complication. Rates as high as 6–7% have been reported in the literature.

Dissections in and around the thyroid gland, including surgery on the parathyroid glands, require extreme vigilance for damage to the RLN. Although IONM, as discussed above, can help surgeons to confirm identification of the RLN, only through careful surgical dissection the RLN can be preserved. Unilateral RLN damage can result in hoarseness and voice change secondary to unilateral vocal cord paresis in the adducted position, although over adduction by the contralateral vocal cord can often mask these symptoms. Bilateral RLN damage can result in airway obstruction, aphonia, and, potentially, respiratory arrest without immediate re-intubation and establishment of a surgical airway. This situation, however, is extremely rare, with one quoted incidence of 1 in over 30,000 thyroid operations ^[23].

Risk factors for both transient and permanent RLN palsy include: the type of underlying thyroid disease, the extent of resection, and whether or not the RLN was exposed in total lobectomy cases ^[24]. Most RLN paresis tends to be transient ^[25].

Hematoma

Any hematoma in the anterior neck has the potential to affect the trachea and, thus, cause airway compromise. Wound dressings should be checked immediately after extubation, especially in the event of coughing on emergence, upon arrival in the recovery unit, as well as prior to discharge. The incidence of hematoma is probably less than 2% ^[26]. However rare, it remains a major life-threatening complication. Early recognition with immediate intervention is the key to the management of this complication. The possibly long interval between the initial operation and the hematoma development (after 24 h) makes ambulatory thyroid surgery inadvisable.

Hypoparathyroid/Hypocalcemia

Surrounding the thyroid gland are the parathyroid glands. Damage to these glands during thyroid surgery can range from stunning of their functionality to inadvertent excision along with thyroid tissue. As one of the primary regulators of calcium homeostasis in the body (see "Parathyroid Surgery" section), perioperative monitoring of ionized (free) calcium is recommended and a single PTH measurement at 4–12 h postoperatively allows for accurate prediction of patients at risk of hypocalcaemia. Patients with a normal postoperative PTH level can be safely discharged on the first postoperative day.

Conclusion

Successful anesthetic management of the patient for thyroidectomy requires a high level of vigilance by the anesthesiologist and surgeon. Preoperative management focuses on signs and symptoms of hyper or hypothyroidism, correction of thyroid abnormalities, and airway control. Smooth extubation is paramount and should be managed with attention to life-threatening postoperative airway compromise of various causes.

Clinical Insights

For the Otolaryngologist (from the Anesthesiologist)

- The use of intraoperative nerve monitoring and the need for paralysis or avoidance of paralysis needs to be communicated effectively.
- Airway derangements and extend of the thyroid mass are crucial information for the anesthesiologist.
- The surgeon must be aware that a safely performed awake fiberoptic intubation takes little added time and avoids airway catastrophes in the "questionable" airway.

For the Anesthesiologist (from the Otolaryngologist)

- To be best prepared, the anesthesiologist should be aware of the indication for thyroidectomy. This may help the anesthesiologist to prepare for an airway complication, thyroid storm, hematoma, or postoperative hypocalcemic crisis.
- It is ideal if the surgeon and anesthesiologist communicate throughout the surgical case to understand and anticipate potential intraoperative and postoperative complications.
- When operating on a patient where there is a potential unilateral or bilateral vocal cord paralysis, the anesthesiologist should be informed of a potential airway obstruction so the anesthesiologist can be prepared for re-intubation if required.

Clinical Pearls

For the Otolaryngologist (from the Otolaryngologist)

- Preoperative planning is essential to predict postoperative complications such as vocal paralysis, airway obstruction, or hypocalcemia. Communicating these considerations to your anesthesiologist will help to anticipate and prepare for postoperative complications.
- If the airway may be obstructed by a goiter or airway invasion, a fiberoptic intubation may be indicated.
- During the course of surgery, if a vocal cord paralysis is anticipated, it should be communicated to the anesthesiologist.

For the Anesthesiologist (from the Anesthesiologist)

- Ask about the lesion: is it substernal, is it encroaching or deviating the airway, is there recent imaging?
- Decide whether traditional intubation will be safe or effective.
- Be alert for signs and symptoms of the non-euthryoid patient and do not do an elective case if the patient is not euthryoid.
- Smooth emergence is key: have a plan for smooth emergence from the beginning.

Parathyroid Surgery

Introduction

Much like surgery on the thyroid gland, surgery on the parathyroid glands is a common operation performed on a daily basis in many hospitals. Most patients presenting for surgery for primary hyperparathyroidism in the USA do not have severe or life-threatening symptoms of hypercalcemia. It is still of utmost important for the anesthesiologist to review the pertinent preoperative laboratory values. A backup plan for the treatment of life-threatening hypercalcemia is paramount. This section will describe an overview of the parathyroid glands and their function, preoperative assessment of parathyroid disorders,

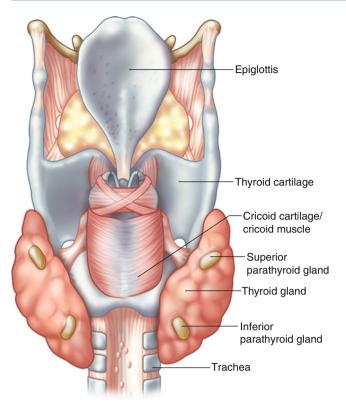


Fig. 14.12 The parathyroid gland and surrounding structures (posterior view

management of the patient with hypercalcemia, and postoperative complications of which to be vigilant.

Overview

The Parathyroid Glands

Four parathyroid glands located in the neck, posterior to the thyroid gland, are responsible for the production and release of parathyroid hormone (PTH) (Fig. 14.12).

PTH regulates serum calcium and phosphate concentrations. PTH causes a net increase in serum calcium via direct and indirect effects. PTH directly acts on the skeleton to increase calcium release and on the kidney to increase calcium reabsorption. PTH acts indirectly on the intestinal tract to increase calcium absorption via 1,25-dihydroxyvitamin D (calcitriol) which it activates in the kidneys. Glandular function is controlled by a negative feedback mechanism via plasma calcium concentration. Very small decreases in ionized calcium-sensing receptor resulting in a rapid release of hormone. A rise in serum calcium leads to decreased PTH synthesis and release.

Primary hyperparathyroidism (PHPT) is characterized by excessive PTH release secondary to benign parathyroid

adenoma, parathyroid gland carcinoma, or parathyroid hyperplasia. PHPT is present in approximately 1% of the adult population. It can occur at any age, however most cases present in patients over 45 years of age ^[27]. PHPT is two to three times more common in woman than men. PHPT is caused by a single parathyroid adenoma in about 80-85% of cases. The remaining cases can be ascribed to multiple gland hyperplasia affecting all parathyroid glands in 10%, double adenomas in 4%, and parathyroid carcinoma in 1% [28]. Other uncommon sources of parathyroid tumors include excessive stimulation due to chronic renal failure or lithium therapy. PHPT may be associated with multiple endocrine neoplasia (MEN) types 1 and 2. MEN-1 (Werner's syndrome) consists of pancreatic and pituitary tumors with concomitant hyperparathyroidism. MEN-2 is divided into MEN-2a which consists of pheochromocytoma, medullary cancer of the thyroid, and hyperparathyroidism; MEN-2b (Sipple syndrome) consists of pheochromocytoma, medullary cancer of the thyroid, and multiple neuromas. Long-term medical management and/or observation is not efficacious in treating PHPT. The definitive treatment for PHPT is surgical removal of the diseased portions of the glands [29]. Operative cure rates of 95–98% with complication rates of 1-2% are possible when parathyroidectomy is performed by an experienced team^[2]. The goal of the surgery is normalization of PTH and calcium/phosphate levels.

Preoperative Considerations

Otolaryngologist's Perspective

The parathyroid glands produce parathyroid hormone (PTH) which regulates serum calcium levels through its end-organ effects on the gut, bone, and kidney. The end result of an increase in PTH is to elevate serum calcium levels. Hyperparathyroidism, a pathologic elevation in serum PTH, can be primary, secondary, or tertiary. Primary hyperparathyroidism results from a single gland adenoma (85% cases), four-gland hyperplasia (5-15% cases), double adenoma (5% cases), or parathyroid carcinoma (1% cases). When serum calcium and PTH levels are extremely high, parathyroid carcinoma must be considered in the differential diagnosis. Secondary hyperparathyroidism is seen in those patients with renal failure and loss of calcium in the urine. In these patients, the chronic stimulus to PTH production results in four-gland hyperplasia. If persistent, autonomous PTH production occurs after the renal failure and serum calcium are corrected as with a renal transplant, this condition is termed tertiary hyperparathyroidism.

In North America, the most common presentation of hyperparathyroidism is an elevated serum calcium level identified on routine screening. A key point to remember is Table 14.8 Indications for parathyroidectomy

Elevation of serum calcium above 1.6 mg/dl for lab	Decrease in bone density above two standard deviations of normal
Life-threatening episode of hypercalcemia	24 h urine calcium excretion >400 mg
Decrease in creatinine clearance >30% expected for age	Renal calculi

that those patients with a fluctuation in protein content, such as those who are malnourished or in liver failure, there will be falsely decreased total calcium. In these patients, ionized calcium is more accurate as it does not vary with protein levels. Common presentations of the disease include a patient who presents with a calcium oxalate kidney stone, osteopenia, muscular weakness, fatigue, depression, peptic ulcer disease, and/or mental confusion. Many patients who appear asymptomatic often suffer from subtle symptoms, such as nonspecific fatigue, weakness, musculoskeletal complaints, constipation, depression, or a history of peptic ulcers, hypertension, cholelithiasis, and/or pancreatitis.

Preoperatively, an elevated serum calcium level and intact parathyroid hormone level should be confirmed. Although rare, familial hypocalciuric hypercalcemia may present like a parathyroid adenoma, therefore a 24-h calcium collection for calculation of the calcium-to-creatinine clearance ratio should be considered to rule out familial hypocalciuric hypercalcemia.

In general surgery is indicated in those patients who have a significantly elevated serum calcium (greater 1.0– 1.6 mg/dl above upper limit of normal) or evidence of renal or bone involvement. Of paramount importance in surgical preparation is localization studies that facilitate the possibility of a minimally invasive approach ^[30]. Table 14.8 lists common indications for parathyroid surgery.

Localization studies prior to surgery assist in adenoma localization. A sestamibi scan is a nuclear study that is the most common study to determine adenoma location. Delayed images will highlight a parathyroid adenoma. In up to 5% of cases a double adenoma can be seen. An ultrasound is commonly done with a sestamibi resulting in a 97% success rate in localization if the etiology is primary hyperparathyroidism secondary to an adenoma. A sestamibi study has the ability to locate mediastinal adenomas unlike ultrasound. In cases where ultrasound and sestamibi fail to localize the site of an adenoma, MRI, CT scan of the neck, or venous sampling may be performed. Venous sampling only provides information as to whether the adenoma may be on the left or right. False negative results may occur with sestamibi in the settting of four-gland hyperplasia, cystic degeneration of the adenoma, or Hashimoto's thyroiditis. The ability to localize an adenoma permits a minimally invasive surgical approach to tumor removal. As a single adenoma is the most common cause of primary hyperparathyroidism, a minimally invasive approach is feasible in the majority of cases.

Anesthesiologist's Perspective

The preoperative assessment for the patient presenting for parathyroidectomy begins with a focused history and physical examination. Surgical patient positioning is similar to thyroid surgery, with neck hyperextension and shoulder roll placement. Therefore neck range of motion should be assessed and documentation of cervical disc disease should be performed.

PHPT leads to a diverse spectrum of signs and symptoms, affecting many organ systems. Up to 80% of patients in Western countries are asymptomatic at presentation and the diagnosis begins with routine calcium screening tests ^[31]. PHPT is usually confirmed following an immunoreactive PTH assay. The classic signs and symptoms of PHPT, including arthralgias, myalgias, abdominal pain, kidney stones, and psychiatric changes are rare at presentation in developed countries, but may occur in milder forms.

Osteitis fibrosa cystica is a distinctive bone disease associated with hyperparathyroidism. This disorder was more common in the past when patients were diagnosed with more advanced disease. More common today are osteopenia and osteoporosis from ongoing calcium release in the bones in patients with PHPT. Other symptoms, if present, of proximal skeletal muscle weakness, hypotonia, and easy fatigability are noted for their striking resolution after surgical correction of PHPT. Care must be taken during positioning and laryngoscopy to avoid the potential for pathologic fracture.

Nephrolithiasis is among the most common renal manifestations of PHPT. It occurs in approximately 15–20% of patients with PHPT ^[32]. The majority of renal calculi are calcium oxalate or calcium phosphate. Nephrocalcinosis, renal insufficiency, and a variety of tubular disorders also occur. Patients may have polydypsia and polyuria at presentation. Hypophosphatemia and hypomagnesemia are also encountered.

PHPT has been associated with a number of cardiovascular manifestations ^[33]. Systemic hypertension may be prominent and should be controlled preoperatively. The electrocardiograph (ECG) must be evaluated in the preoperative period. Disturbances include a prolonged PR interval and a short QT interval with subsequent shortening of the ST segment. Increased mean carotid intima-media thickness and increased vascular stiffness have been described ^[34]. In cases of severe refractory hypertension, pheochromocytoma should be ruled out secondary to the association between PHPT and MEN.

Hypercalcemia

The goal of preoperative treatment is normalization of serum calcium and euvolemia. Hypercalcemia is treated primarily with aggressive hydration with normal saline to both dilute the calcium in the blood and promote calciuresis in the kidneys. This calciuresis can be facilitated with the administration of loop diuretics such as furosemide with dosing titrated to effect. Thiazide diuretics are contraindicated because they can increase serum calcium. Other factors that may aggravate hypercalcemia should be avoided, such as lithium, volume depletion, inactivity, and a high calcium diet. Moderate calcium intake should be encouraged (1000 mg/day) as low calcium intake can lead to increased PTH secretion. Vitamin D intake (400–600 international units) should be maintained as low vitamin D levels stimulate PTH release. In life-threatening hypercalcemia, calcitonin 2–8 units/kg IV every 6 h, bisphosphonates (e.g., zolendronate 4–8 mg IV over 5 min), and dialysis may be necessary.

Intraoperative Considerations

Otolaryngologist's Perspective

Parathyroidectomy, like thyroidectomy, may be performed under local, regional, or general anesthesia ^[35]. Determining the most appropriate anesthetic technique is predicated on the medical condition of the patient, the patient's preference, the patient's anatomy, and adenoma localization. Irrespective of the choice for anesthesia, the patient positioning, draping, and preincision injection are similar to that for thyroidectomy.

Intraoperative parathyroid hormone assessment is commonly used in most major centers where a high volume of parathyroid surgery is performed. This technique has alleviated the need for an additional dissection and biopsy of a second parathyroid gland to rule out hyperplasia. The reason is that if there is a single adenoma, the serum PTH level will steadily decrease. In the setting of double adenoma or fourgland hyperplasia, there may be an additional drop in PTH levels; however, after multiple samples are drawn the PTH level would either level off or increase suggesting that there is additional pathologic PTH secretion present. Intraoperative parathyroid assessment requires that a baseline serum PTH be measured before intubation and serially every 5 min following excision of the adenoma. To effectively utilize intraoperative PTH assessment, blood samples must be drawn throughout the procedure. This requires the placement of vascular access that may include an arterial line or a large bore intravenous catheter that is reliable and readily accessible to the anesthesiologist for blood sampling. Parathyroid hormone has a half-life of five minutes, thus after parathyroid removal serial blood samples are obtained looking for at least a 50% decrease in parathyroid hormone level that does not trend upward for as long as 20 min after adenoma removal. If parathyroid hormone levels begin to trend upward, a search for a second adenoma or the possibility

of four-gland hyperplasia is considered. The use of intraoperative parathyroid hormone avoids the need for reoperation and surgical failure.

The length of surgery can vary considerably. In cases where the adenoma has been localized by preoperative ultrasound, sestamibi, and/or MRI, the surgical time can range from 15 to 40 min and these cases are ideal for a local and/or regional anesthetic. However, in cases where the adenoma has not been preoperatively localized or in reoperative cases, the duration of surgery can be several hours and a full general anesthetic is recommended. Communicating this information to the anesthesiologist is critically important so that the appropriate anesthetic measures can be taken. Because the anatomy is superficial and the strap muscles offer little resistance to retraction, paralysis is not necessary from the standpoint of access. Because parathyroid adenomas are soft and compressible, there is rarely any airway compression that requires special consideration.

Anesthesiologist's Perspective

No particular anesthetic technique is mandated for parathyroidectomy for PHPT. Total parathyroidectomy can be performed safely under regional anesthesia ^[36]. This is accomplished with deep and superficial cervical plexus blockade (see section on Thyroid Surgery for a description of block performance). This may be of benefit for the patient who is at high cardiovascular risk from general anesthesia.

Standard premedications are usually administered after application of standard ASA monitors. Midazolam (0.005-0.03 mg/kg IV) and/or fentanyl (0.5-2 mcg/kg IV) and/or remifentanil (0.02-0.05 mcg/kg/min) are typical for our institution. In the rare case of a patient with mental status changes, premedication is avoided. General anesthesia is typically induced with propofol (1-2.5 mg/kg), or, if indicated, etomidate (0.2 mg/kg) with or without a non-depolarizing muscle relaxant. Muscle relaxant dosages are titrated to effect with the use of a peripheral nerve stimulator. This is critical, as preoperative muscle weakness can decrease muscle relaxant requirements. The use of remifentanil can allow the anesthesiologist to avoid muscle relaxation for intubation. If being used for induction, 2-3 mcg/kg of remifentanil with or without 0.2 mg of glycopyrrolate to avert bradycardia can be administered. The use of lidocaine 1-1.5 mg/kg as an adjunct for induction and intubation can aid if one is avoiding muscle relaxants. We use a reinforced endotracheal tube to prevent kinking of the tube with surgical manipulation.

The patient is positioned with the neck hyperextended using a shoulder roll and a "doughnut" pillow. Care must be taken during patient positioning as PHPT can predispose the patient to pathologic fractures. Blood loss is minimal and a single peripheral intravenous usually suffices for intraoperative fluid replacement.

All patients should receive intraoperative dexamethasone for reduced postoperative nausea and vomiting as well as decreased surgical site inflammation (0.2–1 mg/kg up to 10 mg). It is standard to administer ondansetron (0.1–0.2 mg/kg IV up to 4–8 mg) to further decrease PONV. Droperidol (0.0625 mg IV) can be used as a rescue antiemetic in the recovery unit for severe PONV.

Anesthesia can be maintained with a variety of agents (volatile anesthetics, nitrous oxide, narcotics, and muscle relaxants). Our group typically utilizes a technique involving nitrous oxide inhalation with propofol and remifentanil infusions. The use of remifentanil intraoperatively, especially in a non-paralyzed patient, can allow for a comfortable and immobile patient.

Intraoperative PTH assay is being increasingly utilized. This technique predicts postoperative success in PHPT patients ^[37]. Multiple peripheral blood samples are required and can usually be obtained by backflow from an antecubital IV 18 gauge or greater, after administration of a tourniquet or use of the venous stasis feature of a noninvasive blood pressure cuff placed on the same arm as the intravenous line. Alternatively an arterial catheter can be inserted to facilitate blood sampling in a patient with difficult intravenous access.

Two studies addressed concerns that either the anesthetic agents or the act of laryngoscopy could affect intraoperative PTH assays. In the first study, sedation with propofol versus midazolam without laryngoscopy in patients with secondary hyperparathyroidism undergoing dialysis access surgery showed no difference in PTH levels ^[38]. In another study, after laryngoscopy, the PTH levels significantly increased in patients without hyperparathyroidism but did not increase to statistical significance in patients with primary or secondary hyperparathyroidism ^[39]. As the catecholamine surge associated with direct laryngoscopy likely influences PTH secretion to some extent, our group advocates for a baseline PTH level to be drawn after insertion of the intravenous but before induction of general anesthesia in these patients.

A smooth emergence and extubation are preferred as coughing and bucking can lead to suture line tearing. For recommendations on extubation, please refer to the previous section on emergence following thyroid surgery.

Postoperative Considerations

Otolaryngologist's and Anesthesiologist's Perspectives

Although minimally invasive parathyroid surgery may be performed through an incision as small as 2 cm, the risk of bleeding and/or a vocal cord paralysis resulting in an airway emergency must always be considered. Although complications occur in less than 2% of cases, when the rare complication occurs, if it is not recognized and addressed expeditiously, the consequences can be grave. Observation and gentle palpation of the wound in the postoperative period is a reliable way to assess for a wound hematoma. Similarly, talking with the patient to assess for hoarseness and a subjective feeling of tightness in the neck can also be reliable tools for assessment.

Although postoperative morbidity is rare, careful postoperative monitoring is essential. In addition, postoperative calcium monitoring is performed especially in those patients that have an elevated alkaline phosphatase and are at risk of hungry bone syndrome where the body aggressively absorbs calcium into the bones due to prolonged deprivation resulting in a serum hypocalcemia. In these patients, calcium levels are checked every 6 h and physical exam findings for hypocalcemia such as a Chovstek's sign (see below) and in severe cases, tetany. Patients generally stay in the hospital for 24 h.

Hypocalcemia can occur in the immediate postoperative period. This can lead to significant morbidity. Signs and symptoms of hypocalcemia include muscle spasm, tetany, bronchospasm, and laryngospasm. Treatment is airway control as necessary and calcium chloride administration (1 g IV over 10–15 min). Serial measurements of calcium are obtained in the post-anesthesia care unit. The patient should be monitored for signs and symptoms of hypocalcemia. Chvostek's sign (facial muscle contracture following tapping in the facial nerve distribution) and Trousseau's sign (carpopedal spasm with blood pressure cuff inflation) may be observed.

Life-threatening airway compromise can occur in the postoperative period. Postoperative bleeding can result in hematoma formation, which can compress the trachea. Intubation with surgical decompression is the treatment of choice for this complication. Laryngeal edema secondary to surgical trauma may also occur and require urgent re-intubation. As with thyroid surgery, bilateral recurrent laryngeal nerve injury can lead to aphonia and complete airway obstruction. This emergency requires immediate re-intubation and, possibly, tracheostomy.

Conclusion

Successful anesthetic management of the PHPT patient for parathyroidectomy requires a high level of vigilance by the anesthesiologist. PHPT leads to a variety of organ system disturbances that can alter management in all phases of anesthetic care (pre-, intra-, and post-operative). Preoperative management focuses on hydration, correction of hypercalcemia, and blood pressure control. During anesthetic maintenance careful ECG monitoring for signs of hypercalcemia is essential. Smooth extubation is paramount. Significant hypocalcemia can present complications in the postoperative period. The practitioner should be aware of and be prepared for potential airway compromise following parathyroidectomy.

Clinical Insights

For the Otolaryngologist (from the Anesthesiologist)

- Alerting the anesthesiologists to significant metabolic derangements beforehand is crucial to proper management.
- As with thyroid surgery, any new imaging or airway evaluation information is helpful for the anesthesiologist to best manage the patient.

For the Anesthesiologist (from the Otolaryngologist)

- Surgery of the parathyroid glands can be straightforward and uncomplicated or has the potential to require extensive dissection that can be time consuming and complex. Because identification of the parathyroid glands can be difficult, especially in revision surgical cases, we prefer that there is as little distortion of the anatomy as possible. Therefore, we prefer that no esophageal monitors or nasogastric tubes be placed during the surgical procedure. These can be palpable and misleading during the surgical dissection.
- Access to the neck should be unencumbered. Therefore the drapes at the head of the bed should allow for exposure of the neck from the mandible to the sternum.
- Access to the arterial or venous line for PTH assessment should be easily available for the anesthesiologist so there is minimal disruption during the course of surgery.
- Drawing a PTH prior to laryngoscopy and surgical stimuli provides a true baseline as these procedures will raise PTH levels.

Clinical Pearls

For the Otolaryngologist (from the Otolaryngologist)

- Surgery of the parathyroid glands can be simple or exceptionally complicated. The surgeon and the anesthesiologist should be prepared based on the preoperative workup.
- In revision cases or nonlocalizing adenomas a local or regional technique is not recommended.
- Esophageal monitors or nasogastric tubes can be misleading and complicate the dissection— the esophagus should be kept free of these instruments until after the dissection is complete.

For the Anesthesiologist (from the Anesthesiologist)

- Understand the implications of disordered calcium metabolism in the perioperative period, with a particular emphasis on laryngospasm from hypocalcemia.
- Because intraoperative PTH is commonly used, the anesthesiologist should have access to the vascular access and access should not disrupt the surgeon's field.
- The feasibility of a local and/or regional anesthetic technique for the procedure is often initiated by the surgeon but flexibility is important to all parties for a good outcome.
- Drawing a PTH prior to laryngoscopy and surgical stimuli provides a true baseline as these procedures will raise PTH levels.

Parotid Surgery

Introduction

The parotid gland is the largest of the paired major salivary glands. It is located lateral to the masseter muscle anteriorly and extends posteriorly over the sternocleidomastoid muscle behind the angle of the mandible. The gland is divided into a superficial lobe and a deep lobe by the facial nerve. The parotid duct courses anteriorly from the parotid gland over the masseter muscle and pierces the buccinator muscle to enter through the buccal mucosa adjacent to the second maxillary molar.

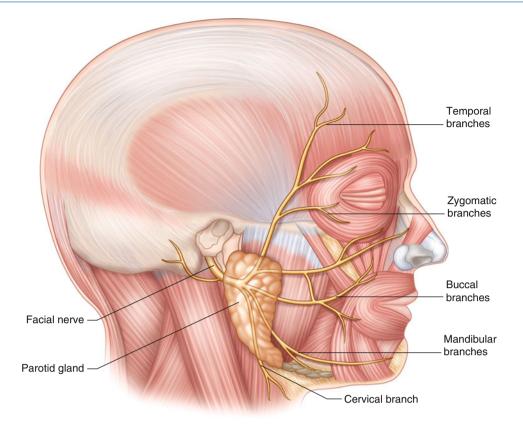


Fig. 14.13 Anatomy of parotid gland and relationship of facial nerve (CNVII)

Overview

The most common indications to perform surgery on the parotid gland include chronic infection and management of a benign or malignant neoplasm. Although the incidence varies from different series in the literature, approximately 80% of salivary gland neoplasms originate in the parotid gland, 10-15% develop in the submandibular gland, and the remaining tumors arise in the sublingual and minor salivary glands. Approximately 80% of parotid neoplasms are benign in contrast to the submandibular and sublingual glands where 50-60% of tumors, respectively, are malignant. The treatment for chronic infection and most salivary gland neoplasms is excision of the lesion with a margin of normal tissue. Because most parotid tumors occur in the region of the tail of the gland and are superficial to the facial nerve, parotidectomy with identification and preservation of the facial nerve is diagnostic and curative in most cases (Fig. 14.13). Complete excision by parotidectomy is usually curative for benign and superficial low-grade malignancies. In most cases, the facial nerve can be preserved

unless there is facial nerve involvement by the tumor. During parotidectomy for malignancy, the periparotid, upper jugular, and posterior submandibular triangle lymph nodes are inspected. Any suspicious nodes are biopsied or included in the dissection. Surgery of the parotid gland requires a great deal of expertise because the complication of facial nerve paralysis is devastating functionally and psychologically.

Surgery on the parotid gland is most often secondary to neoplastic disease. Although many surgical specimens turn out to be benign, the preoperative diagnosis for most patients is a parotid mass. These masses usually do not affect the patient's airway or breathing and do not affect the induction of general anesthesia. During surgery on the parotid gland, facial nerve monitoring is the norm. As such, maintaining general anesthesia without paralysis becomes the challenge for the anesthesiologist. This section will describe an overview of the parotid glands and their function, preoperative assessment of the patient with parotid disease, management of the unparalyzed patient under general anesthesia, and postoperative complications of which to be aware.

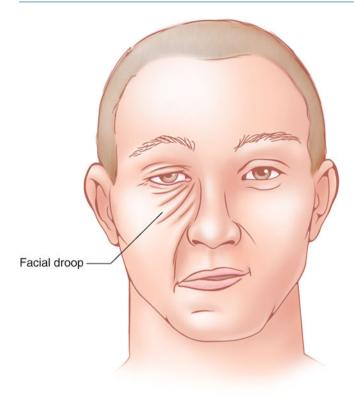


Fig. 14.14 Facial droop associated with facial nerve injury

Preoperative Considerations

Otolaryngologist's Perspective

Preoperative consideration is largely related to determining the nature of the disease and the extent of surgery. Because inflammatory, benign, and low-grade malignancies can be managed with facial nerve preservation, the patient can remain unparalyzed and facial monitoring should be performed. In such cases, the time of surgery can also be estimated. In contrast, if the preoperative workup suggests the presence of a high-grade invasive malignancy or there is a preoperative facial palsy, then facial nerve monitoring is not indicated and the patient can be paralyzed throughout the procedure ^[40] (Fig. 14.14). It also suggests that a more time intensive surgery may be required to complete the resection and the neck dissection. These issues should be discussed with the anesthesiologist to plan appropriately for the case.

Anesthesiologist's Perspective

The main question to be discussed with the surgeon is whether the parotid disease involves the facial nerve preoperatively. If the patient has preoperative facial nerve palsy on the side of the lesion, intraoperative monitoring of the facial nerve is usually unnecessary due to preexisting damage and intraoperative paralysis may occur. If the plan is for lack of paralysis, this should be discussed with the surgeon prior to induction of anesthesia.

As the majority of parotid disease is benign, patients usually do not have significant comorbidities related to the indication for surgery. Thus, a thorough history and physical is usually all that is required prior to proceeding to the operating room.

Intraoperative Considerations

Otolaryngologist's Perspective

Intraoperative considerations should be focused on facial nerve monitoring and blood pressure management. As discussed, preservation of the facial nerve requires that the patient remain unparalyzed yet still throughout the surgical procedure. Because the parotid gland is a vascular organ, careful blood pressure management can be helpful in maintaining hemostasis during the dissection of the gland and identification of the facial nerve. This is particularly important when operating on a chronically infected gland because of the increased inflammation and vascularity.

Anesthesiologist's Perspective

For a parotidectomy, general anesthesia is required as no particular nerve block will cover all of the involved operative field. General anesthesia may be induced in a variety of fashions (see intraoperative management in the above two sections). If muscle relaxation for intubation is mandatory, succinylcholine (1.5-2.0 mg/kg) can be administered. At our institution, as described above, the use of a combination of remifentanil and propofol for induction of general anesthesia can be easily utilized for an immobile, apneic patient for endotracheal intubation. We use a reinforced endotracheal tube to minimize kinking of the tube or bending of the tube during surgical manipulation. The patient will be turned 90° with the operative site away from the anesthesia machine. Should intraoperative facial nerve monitoring be required intraoperatively, an immobile patient is still imperative. Remifentanil infusions (0.05 mcg/kg-0.2 mcg/kg), usually on the higher end of the range, can be utilized for this purpose along with propofol (25-100 mcg/kg/min) and/or any volatile agent and/or nitrous oxide (40-70%).

Should the surgeon require deliberate hypotension for a clean surgical field or hemostasis during the dissection phase, an arterial line may be necessary based on the extent of patient comorbidity. The usual recommendation is to maintain a mean arterial pressure no less than 20% of baseline, in deliberate hypotension, 30% is a target. As there is no current monitor to be sure of adequate total body perfusion, in the absence of severe chronic hypertensive disease, a mean arterial pressure of 50–60 for the duration of the dissection phase is usually tolerated without postoperative sequelae.

It should be noted that if paralysis is required for intubation, per the anesthesiologist's assessment, and succinylcholine is contraindicated, an intubating dose of vecuronium or other non-depolarizing muscle relaxant can be administered with careful attention to peripheral nerve stimulator. Once a return of 1 or more twitches is observed, paralysis can be reversed with an appropriate dose of neostigmine and glycopyrrolate with continued monitoring of the train-of-four ratio to greater than 0.7–0.9. At this point, communicating to the surgeon that paralysis has been reversed is allowable.

Emergence from general anesthesia is conducted in a similar fashion to emergence following thyroid and parathyroid surgery, as described above. Less emphasis on "smoothness" of extubation is applied as any rebleeding (unlikely) does not generally compromise the airway (although this is not absolute).

Modified Radical Neck Dissection

Also of note is the need to avoid paralysis for the patient undergoing a neck dissection associated with malignant disease of the thyroid, parathyroid, or parotid gland. Motor nerves including the hypoglossal nerve and the spinal accessory nerve may be injured during the dissection and, as such, the surgeon may require that the use of NMB agents be avoided during this phase of the surgery. As discussed in detail above, the use of an ultra-short-acting potent narcotic such as remifentanil can facilitate such conditions.

Postoperative Considerations

Otolaryngologist's and Anesthesiologist's Perspectives

Postoperatively, the risk of hematoma is the most significant concern. Although rare, a parotid hematoma should be identified quickly to prevent late complications such as skin slough or dissemination of the hematoma into the neck, where the airway can be compromised. A parotid hematoma, although rare, can, in theory, track down the neck and cause similar airway compromise as described in the previous two sections above. In the case of a patient undergoing parotid surgery using facial nerve monitoring, postoperative assessment of the facial nerve by gross neurologic exam can be helpful in ruling out damage to the facial nerve.

Conclusion

Communication between the surgeon and the anesthesiologist as to the monitoring of the facial nerve during parotid surgery is of the utmost importance. Assessment of the facial nerve, documentation of both preoperative and postoperative function, and a smooth emergence without coughing are of highest priority.

Clinical Insights

For the Otolaryngologist (from the Anesthesiologist)

- Communication as to the involvement, if any, of the facial nerve is crucial to anesthetic planning.
- Any requirements for deliberate hypotension should be planned for commensurate with the patient's underlying medical condition.

For the Anesthesiologist (from the Otolaryngologist)

- It is essential that the surgeon and the anesthesiologist communicate prior to the start of the procedure to review the nature of the case, the preference regarding paralysis, and the anticipated extent of the procedure.
- The majority of the parotid surgery will require that the patient remain unparalyzed, but completely still throughout the procedure.
- Because of the vascular nature of the parotid gland, tight blood pressure control can be effective in maintaining hemostasis during the crucial facial nerve dissection.

Clinical Pearls

For the Otolaryngologist (from the Otolaryngologist)

- Successful parotid surgery is a result of communication between the surgeon and the anesthesiologist, careful preoperative planning, and meticulous surgical dissection.
- Facial nerve monitoring should be considered in revision cases, malignant tumors, and possibly in all cases in order to gain familiarity with the system and proper setup.

For the Anesthesiologist (from the Anesthesiologist)

- Plan to avoid paralysis (in general) and discuss this plan with your surgeon.
- Ask about nerve involvement as you may be able to use paralytics.
- Although less likely than in thyroid surgery, postoperative bleeding may track down to the airway and compromise the patient's ability to breathe.

References

- Greene AB, Butler RS, McIntyre S, et al. National trends in parathyroid surgery from 1998 to 2008: a decade of change. J Am Coll Surg. 2009;209(3):332–43.
- Wittekindt C, Streabel K, Arnold G, Stennert E, Guntinas O. Recurrent pleomorphic adenoma of the parotid gland: analysis of 108 consecutive patients. Head Neck. 2007;29(9):822–8.
- 3. Ringel MD. Management of hyothyroidism and hyperthyroidism in the intensive care unit. Crit Care Clin. 2001;17:59–74.
- Bennett-Guerrero E, Kramer DC, Schwinn DA. Effect of chronic and acute thyroid hormone reduction on perioperative outcome. Anesth Analg. 1997;85:30–6.
- 5. Kohl BA, Schwartz S. Surgery in the patient with endocrine dysfunction. Anesthesiology Clin. 2009;27:687–703.
- Klein I, Danzi S. Thyroid disease and the heart. Circulation. 2007;116:1725–35.
- Amathieu R, Combes X, Abdi W, et al. An algorithm for difficult airway management, modified for modern optical devices (Airtraq laryngoscope; LMA CTrach): a 2-year prospective validation in patients for elective abdominal, gynecologic, and thyroid surgery. Anesthesiology. 2011;114(1):25–33.
- Miller MR, Pincock AC, Oates GD. Upper airway obstruction due to goiter: detection, prevalence and the results of surgical management. Q J Med. 1990;74:177.

- Prescott PT. Disorders of the thyroid. In: Lubin MF, Smith RB, Dodson TF, et al., editors. Medical management of the surgical patient. 4th ed. New York: Cambridge University Press; 2006. p. 367–73.
- Klein I. Endocrine disorders and cardiovascular disease. In: Zipes DP, Libby P, Bonow R, Braunwald E, editors. Braunwald's heart disease: a textbook of cardiovascular medicine. 7th ed. Philadelphia: W.B. Saunders; 2005. p. 2051–65.
- Seybt MW, Terris DJ. Minimally invasive thyroid and parathyroid surgery: where are we now and where are we going? Otolaryngol Clin North Am. 2010;43(2):375–80.
- Honings J, et al. The management of thyroid carcinoma invading the larynx or trachea. Laryngoscope. 2010;120(4):682–9.
- Steffen T, Warschkow R, Brandle M, Tarantino I, Clerici T. Randomized controlled trial of bilateral cervical plexus block versus placebo in thyroid surgery. Br J Surg. 2010;97:1000–6.
- Mulroy MF. Peripheral nerve blockade. In: Barash PG, Cullen BF, Stoetling RK, editors. Clinical anesthesia. 5th ed. Philadelphia: Lipincott, Williams and Wilkins; 2006. p. 723–4.
- Stoetling RK, Dierdorf SF. Endocrine diseases. In: Stoetling RK, Dierdorf SF, editors. Anesthesia and co-existing disease. 4th ed. Philadelphia: Churchill Livingstone; 2002. p. 395–440.
- Randolph GW, et al. Electrophysiologic recurrent laryngeal nerve monitoring during thyroid and parathyroid surgery: international standards guideline statement. Laryngoscope. 2011; 121:S1–S16.
- Chu KS, Tsai CJ, Lu IC, et al. Influence of NDMR on intraoperative neuromonitoring during thyroid surgery. J Otolaryngol Head Neck Surg. 2010;39:397–402.
- 18. Snyder S, Hendricks J. Intraoperative neurophysiology testing of the RLN: plaudits and pitfalls. Surgery. 2005;138:1183–92.
- Pott L, Swick JT, Stack BC. Assessment of recurrent laryngeal nerve during thyroid surgery with laryngeal mask airway. Arch Otolayngol Head Neck Surg. 2007;133:266–69.
- Wall III RT. Endocrine disease. In: Hines RL, Marschall KE, editors. Stoetling's Anesthesia and co-existing disease. 5th ed. Philadelphia: Churchill Livingstone; 2008. p. 365–406.
- 21. Worni M, Schudel HH, Seifert E, et al. Randomized controlled trial on single dose steroid before thyroidectomy for benign disease to improve postoperative nausea, pain, and vocal function. Ann Surg. 2008;248:1060–6.
- Wall III RT. Endocrine disease. In: Hines RL, Marschall KE, editors. Stoetling's Anesthesia and co-existing disease. 5th ed. Philadelphia: Churchill Livingstone; 2008. p. 365–406.
- Miller RD, editor. Anesthesia. 7th ed. Elsevier; 2010 [Chapter 35: Diseases involving the endocrine system and disorders of nutrition].
- Wagner HE, Seiler C. Recurrent laryngeal nerve palsy after thyroid gland surgery. Br J Surg. 1994;81:226–8.
- Chiang FY, et al. RLN palsy after thyroidectomy with routine identification of the recurrent laryngeal nerve. Surgery. 2005;137: 342–7.
- Calò PG, Pisano G, Piga G, et al. Postoperative hematomas after thyroid surgery. Incidence and risk factors in our experience. Ann Ital Chir. 2010;81(5):343–7.
- Wermers RA, Khosla S, Atkinson EJ, et al. Incidence of primary hyperparathyroidism in Rochester, Minnesota, 1993–2001: an update on the changing epidemiology of the disease. J Bone Miner Res. 2006;21:171–7.
- American Association of Clinical Endocrinologists and American Association of Endocrine Surgeons. Position statement on the diagnosis and management of primary hyperparathyroidism. Endocr Pract. 2005;11:49–54.
- Stoetling RK, Dierdorf SF. Endocrine diseases. In: Stoetling RK, Dierdorf SF, editors. Anesthesia and co-existing disease. 4th ed. Philadelphia: Churchill Livingstone; 2002. p. 421–5.

- Silverberg SJ, Bilezikian JP. The diagnosis and management of asymptomatic primary hyperparathyroidism. Nat Clin Pract Endocrinol Metab. 2006;2(9):494–503.
- Bilezikian JP, Silverberg SJ. Clinical practice. Asymptomatic primary hyperparathyroidism. N Engl J Med. 2004;350:1746–51.
- Silverberg SJ, Bilezikian JP. Evaluation and management of primary hyperparathyroidism. J Clin Endocrinol Metab. 1996;81:2036–40.
- Andersson P, Rydberg E, Willenheimer R. Primary hyperparathyroidism and heart disease—a review. Eur Heart J. 2004;25: 1776–81.
- Walker MD, Fleischer J, Rundek T, et al. Carotid vascular abnormalities in primary hyperparathyroidism. J Cln Endocrinol Metab. 2009;94:3849–56.
- 35. Adler JT, Sippel RS, Chen H. New trends in parathyroid surgery. Curr Probl Surg. 2010;47(12):958–1017.
- Cheong YT, Taib NA, Normayah K, Hisham AN. Total parathryoidectomy under local anesthesia for renal hyperparathyroidism. Asian J Surg. 2009;32:51–4.

- 37. Mandell DL, Genden EM, Mechanick JI, Bergman DA, Diamond EJ, Urken ML. The influence of intraoperative parathyroid homone monitoring on the surgical management of hyperparathyroidism. Arch Otolaryngol Head Neck Surg. 2001;127: 821–27.
- Sippel RS, Becker YT, Odorico JS, Springman SR, Chen H. Does propofol anesthesia affect intraoperative parathyroid hormone levels? A randomized, prospective trial. Surgery. 2004;136:1138–42.
- Mahajna A, Barak M, Mekel M, Ish-Shalom S, Krausz MM. Parathyroid hormone response to tracheal intubation in hyperparathyroid patients and normal subjects. Endocr J. 2005;52: 715–9.
- Koch M, Zenk J, Iro H. Long-term results of morbidity after parotid gland surgery in benign disease. Laryngoscope. 2010;120(4): 724–30.
- 41. Braverman, Utiger, editors.Werner and Ingbar's the thyroid: a fundamental and clinical text. 8th ed. p. 516.

Head and Neck Cancer Surgery I: Resection

15

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Introduction

Head and neck cancers comprise about 3–5% of all adult cancers. Perioperative care of these patients can be challenging for both surgeon and anesthesiologist often for different reasons. The goal of the surgeon is to remove the primary cancer as well as perform a successful neck dissection to remove diseased lymph nodes, while leaving the patient with an appropriate cosmetic and functional outcome. The anesthesiologist seeks to secure a potentially compromised airway safely while maintaining anesthesia with surgical staff working directly around the airway. As emphasized throughout this text, communication between staff from both disciplines allows for a team approach to these cancers.

Overview

The sites considered to be "head and neck cancer" sites are the oral cavity, pharynx (nasopharynx, oropharynx, and hypopharynx), larynx, salivary glands, and nasal cavity/ paranasal sinuses ^[1]. Other cancers of the head and neck region include skin cancers and thyroid cancer, either of which may also require neck dissection as part of the surgical

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Department of Otolaryngology – Head and Neck Surgery, Icahn School of Medicine at Mount Sinai, One Gustave L. Levy Place, New York, NY 10549, USA e-mail: alfred-marc.iloreta@gmail.com therapy. In general, careful consideration of malignancy should be given to any neck mass presenting in an adult and men over the age 50 are especially affected.

Preoperative Considerations

Otolaryngologist's Perspective

The anesthetic management is a critical component to successful care of the surgical head and neck cancer patient. Perhaps the most useful operation on which to focus a discussion of this topic is the neck dissection, which is a common and representative procedure encompassing many of the issues facing both the surgeon and the anesthesiologist caring for these patients. Within the chapter, we will also mention a few additional special circumstances encountered in head and neck cancer surgery.

A thorough history and focused head and neck physical exam, including fiberoptic laryngoscopy, is an important first step by the otolaryngologist ^[2]. Biopsies should initially be minimally invasive, as fine needle aspiration (FNA) is quite sensitive for thyroid and epithelial malignancies. As such, performing an open biopsy of a lateral neck mass as an initial diagnostic step is not recommended ^[3]. Doing so risks tumor spillage into the neck and will prevent the patient from receiving a therapeutic neck dissection.

Appropriate diagnostic imaging should be ordered, often including a CT, MRI, and or PET-CT scan^[4]. These are useful to anatomically define the extent of disease, as well as to identify subclinical or non-palpable lesions. Once the appropriate workup has been completed, a discussion about treatment options can begin. Often, neck dissection is indicated for head and neck cancers for which primary surgical therapy has been recommended.

When surgery is being performed for a head and neck primary tumor, and there is demonstrable disease in the neck,

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clinically or radiographically, neck dissection is indicated. In patients with subclinical disease, also known as the N0 neck, the indications for surgery are as follows:

- The patient declines radiation therapy in the setting of a neck that requires treatment.
- The probability of occult neck disease is substantial, usually defined as greater than 20%.
- The need for a staging neck dissection to determine postoperative adjuvant therapy ^[5].

Patients may receive elective neck irradiation rather than neck dissection when the risk of occult nodal metastases is substantial. In general, however, there are some guidelines for elective treatment, surgical or nonsurgical, of the neck for squamous call carcinoma (SCC) of the upper aerodigestive tract (Table 15.1).

Management of the neck is paramount in the treatment of cancers of the upper aerodigestive tract, the skin in the head and neck, the thyroid and salivary glands. Because there is a complex network of lymphatics that drain through the neck, the lymph nodes are often referred to as "watchmen" of disease within this region ^[30]. Neoplastic cells from primary head and neck cancers travel through the lymphatic fluid and can become trapped in the cervical lymph nodes, where they tend to aggregate to evade the host immune system and eventually proliferate, causing significant morbidity and mortality [31, 32]. Lymph nodes containing disease may be treated nonsurgically or surgically, and this decision often rests on what treatment is being rendered for the primary tumor. When surgical therapy is performed on the primary tumor, the diseased neck(s) is/are often treated surgically as well. Removal of a group of cervical lymph nodes en bloc is referred to as a "neck dissection" [30].

Basic Anatomy

A *comprehensive* neck dissection includes all levels of the lateral neck (I–V) and if indicated the central compartment of the neck (level VI) (Table 15.2).

Radical neck dissection implies a comprehensive dissection that also sacrifices three structures: spinal accessory nerve, internal jugular vein, and sternocleidomastoid muscle. *Modified radical* neck dissection is a comprehensive dissection that spares one or all of those structures. Finally, *selective* neck dissection implies removal of fewer than five of the lateral neck compartments ^[34–36]. The risks of neck dissection are outlined in Table 15.3. Relative contraindications for neck dissection include unresectable disease, vertebral involvement, significant involvement of the great vessels or skull base (including intracranial disease) and distant metastases ^[37].

Table 15.1 Indications for elective treatment of the neck in head and neck cancer

Any site—primary tumor characteristics

- Perineural spread ^[6]
- Lymphovascular invasion ^[7]

Oral cavity [8, 9]

- Advanced T3,T4 tumors ^[10]
- Any T classification with depth of invasion >4 mm [11]
- Consider neck treatment for all buccal SCCA^[12, 13]
- Consider bilateral neck for lesions encroaching the midline, or tumors of the floor of the mouth

Oropharynx

• Almost all T stage classifications [14, 15]

 High risks of retropharyngeal nodal spread often necessitates radiation either in addition to or instead of surgical treatment, as access to the retropharyngeal lymph nodes is difficult ^[16]

Nasopharynx

 Salvage surgery for recurrent nasopharyngeal carcinoma following chemoradiation failure ^[17]

Hypopharynx

Almost all T stage classifications [14, 18]

Larynx [19-21]

- Supraglottic: all except superficial lesions
- Glottic: T3 and T4
- · Subglottic: all cases

Lip ^[22]

- Advanced T3/T4 tumors
- Any T classification involving oral commissure
- Recurrent lesions

Other indications for neck dissection

- Thyroid CA
 - Presence metastatic neck disease ^[23]
 Medullary thyroid cancer ^[23]
- Cutaneous malignancies
 Merkel cell ^[24, 25]
- Positive neck disease with dissection and RT – Cutaneous SCC
- Only if with positive neck disease ± XRT^[26]
- High-grade salivary malignancy ^[15, 27–29]
 - Consider neck dissection with patients with high-grade mucoepidermoid cancer, undifferentiated carcinoma, carcinoma ex-pleomorphic, adenocarcinoma, squamous cell carcinoma

Anesthesiologist's Perspective

Perioperative assessment is critical for all patients and is discussed in detail for the general patient population presenting for otolaryngology surgery in Chap. 7, however patients presenting for head and neck cancer surgery pose a significant challenge to the anesthesiologists. Although there is a growing population of young, healthy patients presenting with intraoral malignancies presumed to be associated with the Human Papilloma Virus (HPV) ^[38–40], the majority of patients will be elderly ^[41, 42] and have significant medical problems. Given the risk factors (tobacco exposure ^[6], ethanol abuse ^[43], gastroesophageal reflux ^[44]), anticipated comorbidities and

Table 15.2 Levels of the neck [33]

- 1. Level IA: submental triangle
 - (a) Boundaries: anterior belly of the digastric muscle and the hyoid bone
 - (b) Cancers from FOM, anterior tongue, anterior mandibular alveolar ridge, lower lip
- 2. Level IB: submandibular triangle
 - (a) Boundaries: body of the mandible and the anterior and posterior bellies of the digastric muscle
 - (b) Metastases from oral cavity, anterior nasal cavity, midface soft tissue, submandibular gland

3. Level II: upper jugular nodes

- (a) Boundaries
 - Anterior: lateral border of the sternohyoid muscle
 - *Posterior*: posterior border of the sternocleidomastoid muscle
 - *Superior*: skull base
 - *Inferior*: carotid bifurcation(surgical), hyoid bone (radiological)
 - Level II is divided by the *accessory nerve* into levels IIa (anteriorly) and IIB (posteriorly)
- (b) Metastases from the oral cavity, nasal cavity, nasopharynx, oropharynx, hypopharynx, larynx and parotid
- 4. *Level III*: middle jugular nodes
- (a) Boundaries
 - · Anterior: lateral border of the sternohyoid muscle
 - *Posterior*: posterior border of the sternocleidomastoid muscle
 - Superior: hyoid bone
 - Inferior: omohyoid (surgical), cricoid notch (radiological)
- (b) Metastases from oral cavity, NP, oropharynx, hypopharynx, larynx
- 5. Level IV: Lower jugular nodes
 - (a) Boundaries:
 - Anterior: lateral border of the sternohyoid muscle
 - Posterior: posterior border of the sternocleidomastoid
 muscle
 - *Superior*: omohyoid (surgical), cricoid notch (radiological)
 - Inferior: clavicle
 - (b) Metastases from hypopharynx, thyroid, cervical esophagus and larynx
- 6. Level V: Posterior triangle
- (a) Boundaries:
 - Anterior: posterior border of the sternocleidomastoid muscle
 - *Posterior*: anterior border of the trapezius muscle *Inferior*: clavicle
 - (b) Metastases from nasopharynx, oropharynx, cutaneous structures of posterior scalp and neck
- 7. *Level VI*: nodes of the anterior compartment—pretracheal, paratracheal, precricoid lymph nodes
 - (a) Boundaries:
 - Lateral: lateral border of the sternohyoid muscle
 - Superior: hyoid bone
 - Inferior: suprasternal notch
 - (b) Metastases from the thyroid, glottis and subglottic, apex of piriform, cervical esophagus

the impact of head and neck cancer on airway anatomy (e.g., primary involvement or effects from previous surgery, chemotherapy, or radiation therapy), it is critical that the anesthesiologist focus their preoperative attention on the

Table 15.3 Risks of neck dissection [1]

- 1. Bleeding, need for transfusion, hematoma
- 2. Wound infection
- 3. Shoulder weakness or pain (CN XI)
- 4. Prolonged facial or neck edema
- 5. Chyle leak
- 6. Neck scar, loss of SCM
- 7. Specific cranial nerve deficits: X (dysphagia, hoarseness, aspiration), IX(dysphagia), XII (dysarthria, dysphagia), VII marginal weakness
- 8. Paresthesias of the neck skin around the ear and possibly the tongue (greater auricular nerve)

 Table 15.4 Clinical risk factors for cardiac complications in the perioperative period [45]

Ischemic heart disease
History of congestive heart failure
History of cerebrovascular disease
Insulin therapy for diabetes mellitus
Preoperative creatinine level greater than 2.0 mg/dL

airway and those organ systems most at risk (e.g., cardiac and respiratory). This approach will afford the anesthesiologist the means to best stratify risk, initiate further work up or optimization, and plan the most prudent anesthetic. The authors refer the reader to Chaps. 7 and 8 for a detailed review of the preoperative assessment of the airway; however, there are a number of issues that pertain specifically to patients with head and neck cancer that will be reviewed here in detail.

Cardiac Evaluation and Surgical Risk

The risk factors for head and neck cancer (e.g., gender, advanced age, significant history of tobacco and ethanol ingestion) are also risk factors for cardiovascular disease. Therefore, a thorough cardiac evaluation is mandatory in order to best stratify risk and formulate an anesthetic plan. Table 15.4 outlines the significant clinical risk factors for patients undergoing noncardiac surgery (history of coronary artery disease, congestive heart failure, cerebrovascular disease, insulin-dependent diabetes mellitus, and renal insufficiency). The more risk factors a patient has, the greater the risk of perioperative complications.

When considering overall risk stratification and perioperative management, anesthesiologists must understand and consider the risk associated with specific surgeries. Given the variety of head and neck lesions, surgery for head and neck cancer is heterogeneous and could be more or less invasive, but in general head and neck operations are considered intermediate risk surgeries ^[46]. This distinction is important for preoperative risk stratification, indications for further evaluation and medical optimization.



Fig. 15.1 Positioning of the surgical team during head and neck cancer surgery

Unless emergent (i.e., airway compromise, bleeding, etc.), patients with decompensated and symptomatic cardiac disease must be worked up, stabilized, and optimized before surgery. Patients with significant stable asymptomatic cardiac disease who report the ability to generate four metabolic equivalents (METS) (walk four flat blocks or walk up two flights of stairs ^[47]) can proceed to surgery without further workup. If the patient's functional status cannot be determined (limb loss, back pain, vertigo, etc.) or the patient cannot generate ≥ 4 METS without significant symptoms (shortness of breath, chest pain, palpitations), it is recommended to determine whether or not the patient has at least one or more clinical risk factors (Table 15.1). These patients might benefit from further noninvasive testing (stress test, echocardiograph), but only if it would affect management, otherwise they should proceed to surgery and be treated with perioperative beta blockade unless contraindicated (COPD, asthma) (see Chap. 7, Fig. 7.1).

Airway Considerations

Although this topic is covered in great detail in Chap. 8, it is clear that any patient presenting for surgery for head and neck cancer needs a thorough preoperative airway assessment that includes a review of any imaging studies with the otolaryngologic team (Chap. 2). The goals and objectives for this targeted preoperative evaluation should be to determine whether the patient can be intubated, ventilated, or is at risk to aspirate. The ASA Difficult Airway Algorithm ^[48] places significant weight on preoperative assessment and the ability

to recognize the difficult or impossible airway preoperatively. Interestingly, identifying those patients at risk for aspiration is not mentioned in the algorithm. This is, in our opinion, very important especially in head and neck cancer patients^[7], given the fact that this determination does have a major impact on management (awake vs. asleep intubation). Factors that are important to ascertain during this evaluation includes a history of prior head and neck surgery and subsequent treatments with chemotherapy and/or radiation, signs and symptoms such as cyanosis, hypoxemia, change in voice, positional dyspnea, or frank stridor, lesion location, anatomic involvement, and any compromise of normal anatomy without symptomatic disease.

Nutritional Status and Dehydration

There is a growing body of evidence suggesting the importance of one's nutritional status in risk reduction during the perioperative period ^[49-51]; however, anesthesiologists tend to overlook this parameter during their preoperative assessment. Although obesity is associated with adenocarcinoma of the esophagus ^[52], and associated with decreased survival rates in some cancers ^[53-55], a normal to high body mass index (BMI) is associated with improved survival for patients with head and neck cancer. Unfortunately, patients with head and neck cancer are at risk for malnutrition and tend to have normal to low BMIs. In general, smoking is associated with a lower BMI, alcoholism is associated with malnutrition, and patients with head and neck cancer generally have poor oral intake for a variety of reasons (painful or obstructing lesions limiting oral ingestion, radiation treatment obliterating taste, nausea, heightened metabolic rates, anorexia, fullness from alcoholic intake). Given the fact that patients with poor nutrition and hydration status are at increased risk for postoperative complications, it is important for both anesthesiologists and otolaryngologists to conduct a thorough evaluation of nutritional and hydration status. The diagnosis of malnutrition is based on objective measurements of nutritional status, including the assessments of oral intake, weight loss, biochemical parameters, and body composition. Therefore, if malnutrition is suspected a preoperative nutrition consultation may be warranted and when appropriate, the surgery delayed to optimize conditions with parental nutrition.

Radiation Therapy

It is important for every anesthesiologists and otolaryngologist to appreciate that radiation therapy (RT) causes tissue trauma and burns resulting in scaring, fibrosis, tissue immobility, and friability. RT of the head and neck causes these tissue changes in the airway. This makes patients treated with RT difficult or impossible to intubate using standard laryngoscopy AND difficult or impossible to ventilate via bag mask devices ^[56, 57]. One way to determine the degree of immobility is to apply minimal external force to the laryngeal structures. If the laryngeal cartilage is fixed, unless contraindicated, these patients' airways must be secured awake.

Chemotherapy

Patients presenting for recurrence of head and neck cancer need a thorough review of their chemotherapy history. The most common chemotherapy used today is platinum/5fluorouracil (5-FU). Cetuximab, a monoclonal antibody against epidermal growth factor receptor (EGFR), has been used for platinum-refractory disease and has significantly improved median survival when used in combination 5-FU ^[58]. 5-FU, irreversible inhibits thymidylate synthase and therefore eliminates RNA synthesis. 5-FU causes bone marrow suppression (anemia, thrombocytopenia, leukopenia), stomatitis, mucositis, and diarrhea. Cetuximab infusions can cause an acute anaphylactic reaction and pretreatment with diphenhyramine is recommended, chronic Cetuximab can cause magnesium wasting and hypomagnesemia ^[59] and rarely pulmonary and cardiac toxicity ^[60, 61].

Tobacco Cessation

Although advocating tobacco cessation during the perioperative period could reduce postoperative complications, the

Table 15.5 Prevention and treatment of alcohol withdrawal syndrome
(1) Brief intervention, consider detoxification for dependency before or after surgery
 (2) Perioperative and intraoperative prevention of delirium: (a) Premedication if indicated Long-acting benzodiazepine the evening before surgery (e.g., lorazepam) Short-acting benzodiazepine the morning of surgery (e.g., midazolam) (b) Before induction of anesthesia if not adequately premedicated Midazolam (0.5–5 mg i.v. titrated) (c) Initiated after induction of anesthesia Clondine (0.5 mg/kg per h) Haloperidol (up to 3.5 mg per day) Ketamine 0.5 mg/kg
(3) Prevention of Wernicke's encepaholpathy(a) Thiamine 200 mg parenterally for at least 3–5 days

duration of cessation for optimal postoperative benefits is currently unknown and remains controversial. Several studies suggested that a 2-week hiatus reduces pulmonary complications ^[62] while others suggested that bronchopulmonary airway reactivity may increase in the acute phase of abstinence leading to increased postoperative complications ^[63]. Although the optimal time to abstain from smoking is unclear most agree that abstaining from smoking for 12–24 h preoperatively assures the elimination of carbon monoxide thus improving oxygen delivery.

Alcohol Cessation

Acute alcohol withdrawal during the perioperative period can result in life-threatening complications ^[64]. Given the association with head and neck cancer, a thorough ethanol ingestion history is critical ^[65, 66]. Alcohol withdrawal syndrome (AWS) occurs in approximately 25% of intensive care patients and may lead to a life-threatening state, its mortality being significantly dependent on treatment: 15% mortality when untreated vs. 2% mortality when treated ^[12]. Table 15.5 outlines the recommendations for the perioperative prevention and treatment of acute alcohol withdrawal syndrome.

Intraoperative Considerations

Otolaryngologist's Perspective

Most surgeons prefer that muscle relaxation is avoided during neck dissection, so as to allow for monitoring of motor nerves in the surgical field. There are instances when a panendoscopy is performed prior to the neck dissection. In these cases, communication with the anesthesiologist is important so that paralytic requirements can be coordinated. During laryngoscopy, sufficient muscle relaxation may be needed especially in patients with history of prior radiation therapy to the upper aerodigestive tract. The average endoscopy will require approximately 10–15 min to complete. At that point, the patient is prepped and draped again in preparation for the neck dissection. In these cases, reversal of any muscle relaxant would be needed prior to the incision for the neck dissection. Similarly, a neck dissection may be performed along with other oral cavity procedures such as a glossectomy, floor of mouth resection, or pharyngectomy. In these cases, the surgeon must also communicate with anesthesiologist regarding the approximate time of this portion of the procedure, so that appropriate reversal of any paralytic agents can be timed correctly.

Standard prophylactic antibiotics are administered to prevent skin infection, as neck dissection alone is a clean surgery. In cases where a primary aerodigestive tumor is also being resected, this becomes a clean-contaminated case, and additional antibiotic prophylaxis is sometimes requested. Perioperative steroids are also sometimes administered to minimize inflammation to cranial nerves and to minimize postoperative edema (dexamethasone 10 mg prior to incision).

Neck dissection is performed under general anesthesia. To provide optimal exposure a "shoulder roll" is placed beneath the scapula and the patient is positioned with the neck extension. The head is stabilized by placing a foam or gel "donut" underneath. Sometimes, because of the location or volume of tumor, trismus, or prior radiation effects, awake fiberoptic intubation is indicated for the safest care of the patient. Nasotracheal intubation is often requested in patients with oral cavity or oropharyngeal tumors. Surgeons should be present for the induction of anesthesia, as head and neck cancer patients often have tenuous airways or mass effect on anatomic structures, such that an emergent surgical airway may be indicated. The team should always be prepared for such a scenario, and tracheostomy sets should be readily available, and when indicated, open and prepared. The endotracheal tube should be secured to the side contralateral to the tumor in order to allow for maximal exposure. A "gooseneck adapter" is often used to allow easier manipulation of the circuit and prevent circuit compromise. Frequently, surgeons will turn the bed 90-180° from the anesthesia workstation in order to maximize space for the surgical team and to improve access to the head and neck (Fig. 15.1).

Duration of the procedure generally ranges from 1 to 4 h, depending on the complexity of the case (tumor invasion, previous surgery, prior RT), concomitant procedures being performed, levels of lymph nodes removed, and the individual surgeon. Expected blood loss is usually less than 100 mL but large blood losses may be encountered if major vascular structures are breached. In larger cases involving extensive ablation or reconstruction, urinary catheters, arterial lines, and/or central venous lines may be needed irrespective of established patient comorbidities. Again, a thorough discussion between the surgical and anesthesia teams is important to clarify the need for these additional monitors and devices.

Special Situations: Total Laryngectomy and Tracheoesophageal Puncture

Total laryngectomy patients have, by definition, an endtracheostome, or in other words, a permanent surgical airway. Importantly, in emergency situations, it needs to be clearly communicated to the patient's treatment team that the absence of the larynx precludes oral or nasal intubation.

In addition, these patients are rendered aphonic by the surgery, but options exist for voice restoration. One common solution is the electrolarynx, a mechanical device that is placed near the mouth and is used to create vibrations through which the patient may produce speech. There is, however, a surgical method for voice restoration that creates a more natural-sounding voice, which can be modulated by the patient. This technique is essentially a variation of esophageal speech. The surgeon creates a small fistula through the back wall of the membranous trachea into the esophagus. Generally, the fistula is kept open with a red rubber catheter, directed into the distal esophagus. In the immediate postoperative period, this red rubber catheter may be used for enteral feeding. Once the tracheal stoma has matured and postoperative swelling subsides, a tracheoesophageal puncture (TEP) prosthesis is placed in the office setting. This prosthesis is a double-phalanged silicone device; one phalange sits on the posterior tracheal wall, and the other phalange on the anterior esophageal wall. Between the two phalanges is a one-way valve which, when the tracheal stoma is occluded manually, diverts airflow from the lungs through the trachea and into the esophagus. The patient can then force the air out of his mouth while enunciating, thus creating speech. The patient works closely with a speech pathologist in the postoperative period to learn this new manner of phonation.

TEP may be performed during the laryngectomy surgery, or as a secondary procedure. In either case, as with the laryngectomy itself, there will likely be many times when apneic anesthesia is necessary. The endotracheal tube will be removed during stoma creation, as well as during insertion of the red rubber catheter or voice prosthesis. Inhaled oxygen should be kept at a minimum level during these times, and the surgeon should also be cognizant of using electrocautery sparingly around the stoma as airway fire is a risk.

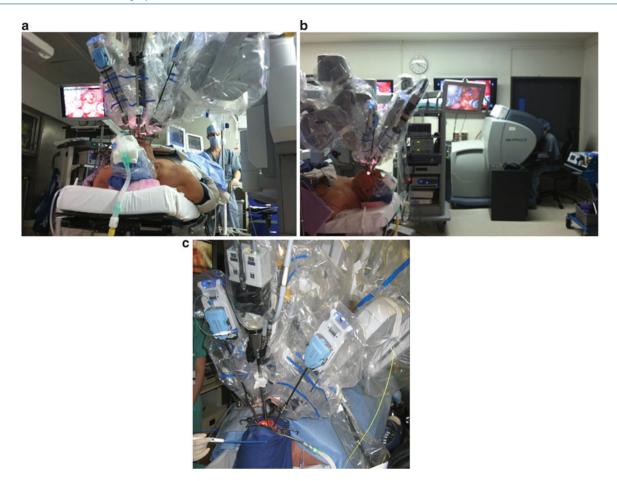


Fig. 15.2 Room arrangement for transoral robotic surgery. (**a**) The figure demonstrates the proper placement of routing of the circuit after nasotracheal intubation with the robotic cart in the background with viewing monitor for the assistant surgeon. (**b**) The figure demonstrates operating room setup with the surgeon console and patient with

placement of robotic instruments. (c) The close-up view demonstrates the number of instruments occupying the oral airway. In addition note the assistant surgeon holding a suction bovie to participate when needed during the procedure

Special Situations: Transoral Robotic Surgery

A quickly developing field in head and neck surgery is that of transoral robotic surgery (TORS). In the head and neck, it has been performed using the daVinci system, which consists of a surgeon console (manipulation of robotic arms) and the robotic cart, which has a stereoscopic camera and 2-3interchangeable instruments. An assistant sits at the head of the patient and helps facilitate the surgery with standard operating instruments when necessary (Fig. 15.2). This technique was first reported in 2007 for the treatment of supraglottic carcinoma ^[1]. Long-term data is not available; however, early follow-up studies have shown excellent local and regional control rates ^[2]. Numerous studies in the literature have reported their experience with this technique and although its place in the surgical management of head and neck cancer has yet to be determined, there is considerable excitement regarding this new technology and as a result

numerous centers will be implementing this technique and an understanding of the anesthetic issues unique to these cases is imperative.

Because of the large number of devices (retractor arms, camera, suction, instruments) that need to be placed through the oral cavity, it is critical to plan accordingly with the team. In general, nasotracheal intubation is preferred, as the endotracheal tube is routed away from the surgical field and equipment. Communication with the anesthesiologist regarding any alteration in nasal anatomy, such as a deviated septum or tumor extension to the nasopharynx, is imperative. Knowledge that a particular side of the nasal cavity would be easier for intubation or that friable tumor is in the path of the endotracheal tube can help avoid unnecessary bleeding that would obscure identification of the larynx and proper endotracheal tube placement. A tracheostomy is indicated in cases where a nasotracheal tube is not feasible, or if the tumor extent or anticipated postoperative edema dictates a surgical airway.

Anesthesiologist's Perspective

Surgery for head and neck cancer can be complex, lengthy, and associated with significant physiologic derangements of the cardiovascular, pulmonary, hematologic, and thermal regulatory systems. Additionally these surgeries can be quite destructive and may require significant surgical time and effort to perform the reconstruction during the initial resection surgery or at a later date (Chap. 16).

General Considerations

Depending on the patient's history and physical, the induction of general anesthesia will proceed or follow the airway management (see airway management below). Our team generally employs a balanced anesthetic technique with the plan to emerge and extubate the patient lucid, cooperative and breathing spontaneously while not bucking or becoming tachycardia or hypertensive in response to the endotracheal tube. Patients who have had a tracheostomy placed are allowed to breathe spontaneously at the conclusion of surgery and will be transferred to a monitored setting still under anesthesia in most cases. If laser surgery is planned nitrous oxide and high oxygen concentrations are avoided. Unless contraindicated 0.2-0.4 mg of glycopyrrolate is administered as an antisialagogue. This will facilitate airway management, intraoperative exposure, and postoperative care. It also has the added advantage of blocking parasympathetic tone associated with intraoperative laryngeal instrumentation or carotid body manipulation by the otolaryngologist. All patients receive an anti-inflammatory and antiemetic dose of dexamethasone (0.05-1 mg/kg, or 10 mg for most patients). Ondansetron 4 mg is also given routinely to prevent postoperative nausea and vomiting.

Neuromuscular Blockade Agents

While surgical exposure for intraoral procedures will be facilitated by the use of neuromuscular blockade agents (NMBA), they should be avoided during neck dissection surgery where nerve monitoring is planned. Given the avoidance of NMBA, it is critical that otolaryngologist appreciate that unparalyzed patients may move if the surgical stimulation is out of proportion to anesthetic depth. This can occur for a variety of reasons, but in general will occur during the initiation of stimulation after a hiatus in stimulation (e.g., after induction and airway management but before surgery has commenced, repositioning and draping during various stages of the surgery, waiting for frozen section results, etc.). This is especially true in a population where older patients with significant cardiovascular disease and questionable hydration status are the norm and tolerate only decreased levels of anesthesia. Many times these patients will require the use of vasopressor agents just to maintain cardiovascular stability while an adequate depth of anesthesia is administered to assure immobility. Given the complexity of balancing an immobile patient without the use of NMBA, everyone needs to keep open communication, and it is critical for the anesthesia team to be constantly aware of the surgical procedure and the otolaryngologic team needs to communicate to the anesthesiology team before commencing with surgery or about to start a particularly stimulating portion of the procedure.

During many head and neck procedures, both intra and extraoral procedures will be planned during the same surgery (e.g., trans-oral robotic surgery). In these cases NMBA will be employed during the intraoral portion and reversed during the extraoral portion of the procedure. When NMBAs are used, it is prudent to use a peripheral nerve stimulator and document resolution of the neuromuscular blockade and communicate this resolution with the surgical team.

Airway Management

This topic is covered extensively in Chap. 8, however due to the nature of the surgery and the airway complexity in these patients, the topic will be covered here emphasizing the unique aspect of airway management for patients presenting for head and neck cancer surgery. Although most patients presenting for a peripheral neck dissection with limited airway involvement can be intubated orally using a basic laryngoscope after a standard anesthetic induction, many patients will present with complex airway problems requiring a wellconstructed airway management plan. When planning the airway management several considerations need to be determined. These include (1) the timing of airway managing (before or after the anesthetic induction), (2) nasal or oral intubation, (3) choice of endotracheal tube, (4) equipment to be employed for successful intubation.

Awake Vs. Asleep Intubation

We recommend that most patients presenting with intraoral cancers of the base of the tongue, epiglottis, vallecula, pharynx, or larynx, be intubated awake. Those patients previously treated with radiation therapy will also be intubated awake given the known difficulties with both ventilation and intubation ^[56, 57]. Even small, non-obstructing intraoral lesions can be friable, bleed and lead to difficulties in ventilation and intubation (Fig. 15.3). When in doubt our team recommends performing the airway management before the induction of anesthesia using an effective and systematic method for

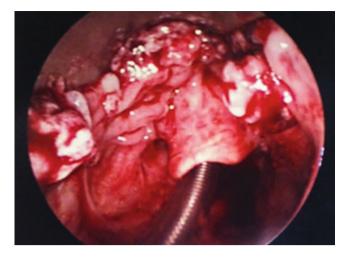


Fig. 15.3 Patient with a friable vallecula lesion intubated using flexible fiberoptic bronchoscopy in order to avoid direct trauma to the lesion with a laryngoscope

airway topicalization described in detail in Chaps. 8 and 10. We also recommend using minimal sedation to minimize respiratory depression and maintain patient cooperation. We have found that the ability for the patient to follow commands such as "take a deep breath," "stick out your tongue," "extend your jaw," is invaluable in the ability to identify and recognize anatomic structures and increase the ease of awake fiberoptic intubation.

Due to a lack of maturity (e.g., pediatrics) or cooperation (e.g., mental illness), an awake approach may not possible for all patients. Although not ideal, in these situations neuromuscular blocking agents must be avoided and spontaneous ventilation preserved until after the airway is secured. An induction of anesthesia with Sevoflurane or agents that do not blunt respiration (e.g., ketamine, dexmedetomidine) may be necessary. Since there is a high likelihood of laryngospasm when intubating spontaneously breathing patients we prepare a syringe of 4% lidocaine and spray the vocal cords (this can induce laryngospasm which is usually temporary) prior to entering the larynx.

Nasal or Oral Intubation

Although many intraoral resections for small tumors (e.g., partial glossectomy, tonsillectomy, palate resection) can be performed with an oral RAE (Ring, Adair and Elwyn or "right angle endotracheal") tube or a wire reinforced anode tube secured to the patient's chin, we prefer to nasally intubate most patients presenting for significant intraoral resections to assure a stable intraoperative airway, avoid surgical interference, and provide a comfortable stable airway post-operatively should the patient remain intubated.

When performing a nasal intubation, nasal patency, and tumor location help dictate placement. If patency is not an issue, the endotracheal tube is placed in the nostril opposite the side of the lesion. When performing the procedure awake we select one nostril to prepare with 0.25% phenylephrine spray and 4% atomized lidocaine. Limiting preparation to one nostril limits the dose of lidocaine and phenylephrine administered to the patient (Fig. 15.4).

Choice of Endotracheal Tube

Due to the complexity of airway pathology and the proximity of the airway to the surgical field, anesthesiologists must be knowledgeable about, and have available, a variety or endotracheal tube types to manage head and neck cancer patients. The choice of endotracheal tube style will depend on a number of issues including (1) the route (nasal or oral) of intubation, (2) the use of flexible bronchoscopy for intubation, and lastly (3) whether lasers will be used during the procedure.

Anode Tubes

Wire reinforced anode tubes can be placed both orally and nasally. Because of their flexibility, resistance to kinking and occlusion from outside forces, anode tubes can be placed in a variety of positions and are frequently used in head and neck surgery. One criticism of orally placed anode tubes is that they should never be left in postoperatively because once crushed they can cause total airway obstruction by remaining kinked. While it is true that anode tubes that are crushed (typically from the patient biting down during emergence ^[67]) stay crushed, they still remain patent and do not cause a total airway obstruction in most cases (Fig. 15.5). In addition anode tubes crushed in the postoperative period do not need to be emergently replaced, they simply need to be reformed by bending the wire reinforcement back using a device like a hemostat.

RAE Tubes

RAE tubes have preconfigured bends for both oral and nasal intubations. The preconfigured bend of a RAE tube has its advantages and its disadvantages. The preconfigured bend (Fig. 15.6) prevents kinking while directing the breathing circuit away from surgery performed on the palate, maxilla, nose, eyes, and cranium. The anesthesiologist must be aware that the internal diameter (ID) of a RAE tube corresponds to the position of the bend (depth the tube is secured at the lips) and the use of larger ID tubes may be necessary to decrease the

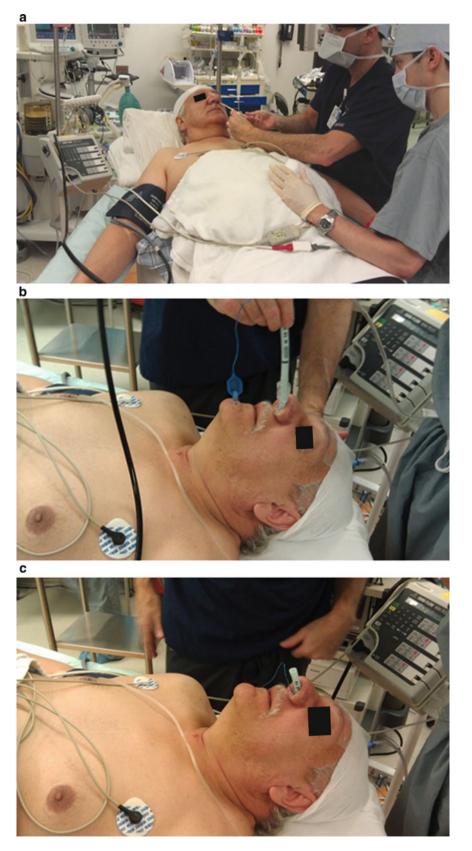


Fig. 15.4 Nasal topicalization and subsequent awake intubation prior to head and neck cancer surgery. (a) Patient semi-recumbent during nasal preparation. Note draping of monitors from patient's foot in

anticipation of 180° turn after induction. (**b** and **c**) Placement of endotracheal tube with a perpendicular angle to the floor and gentle pressure



Fig. 15.5 A hemostat is used to simulate a patient biting on an anode tube. Note that the tube is not completely occluded and can be reformed using a hemostat



Fig. 15.6 Standard oral RAE endotracheal tube

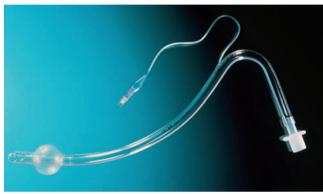


Fig. 15.8 A standard nasal RAE tube

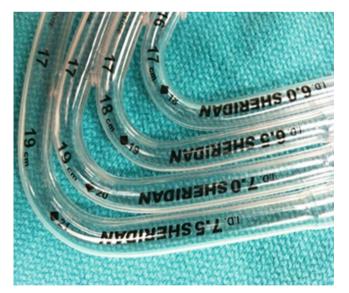


Fig. 15.7 A series of oral RAE tubes demonstrating the relationship between I.D diameter and preconfigured bend positioning

risk of an intraoperative extubation. For example, a RAE tube that bends at 21 cm (depth typically for women) corresponds to a 7.5 ID. A RAE tube that bends at 23 cm (depth typically for men) corresponds to an 8.5 ID diameter (Fig. 15.7).

For this reason we recommend using anode tubes of a desired ID for patients with extremes of height or where subsequent inadvertent extubation would be devastating (prior difficult intubation).

Nasal RAE tubes (Fig. 15.8) have a preconfigured bend that directs the breathing tube back toward the patient's forehead and away from surgeries of the oral cavity, mandible, neck, and thorax. The preconfigured bend also limits endotracheal tube kinking at the nostril, while preventing tissue trauma from undo pressure on the nasal cartilage. Due to the length, bend, and general bulkiness, our team prefers to use soft Portex[®] Blueline[®] PCV nasal tubes (Fig. 15.9a) especially if flexible bronchoscopy is used for the intubation as nasal RAE tubes' length and bend makes them a hindrance during fiberoptic bronchoscopy.

To minimize the chance of this tube kinking at the nostril, we replace the standard 15 mm adaptor with a metal right angle adaptor Fig. 15.9b, c. This adaptor provides a means to direct the circuit away from the operative site while minimizing kinking and tissue trauma. Using two corrugated "gooseneck" endotracheal tube extenders end to end allows the entire endotracheal tube and extensions to mount securely to a head drape (Fig. 15.10).

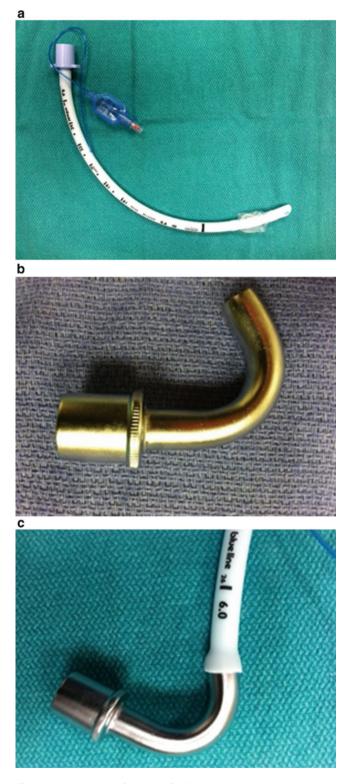


Fig. 15.9 (a-c) Portex[®] Blueline[®] PCV nasal tubes, right angle metal adaptor, and modified tube with adaptor

Laser Safe Tubes

When lasers are used, the anesthesiologists must use laser safe endotracheal tubes. There are several on the market including the stainless steel Mallinckrodt[®] Laser-Flex[®] endotracheal tube and the Medtronic[®] Laser Shield II[®] silicon tube shielded with aluminum and coated Teflon[®] (Fig. 15.11).

Due to a significant price differential we reserve the use of the Medtronic tubes for all nasal intubations and those requiring flexible fiberoptic placement. The steel Mallinckrodt tube is naturally abrasive and should never be placed nasally or risk significant nasal trauma. Because it is fairly stiff we also recommend avoiding this tube when flexible bronchoscopy is used for intubation. If this tube gets hung up on the larynx during placement, it will likely bend and stay bent preventing its smooth entrance into the airway. This situation can cause significant trauma at the level of the larynx. Our team has had greater success placing the Medtronic tubes fiberoptically, nasally, and through very friable lesions (Fig. 15.12).

Equipment to Be Employed: Standard Laryngoscope, Rigid Videoscope, or Flexible Bronchoscope

Patients with lesions of the base of tongue, epiglottis, or vallecula that interfere with the placement of traditional laryngoscopes or fixed videoscopes will require flexible video or fiberoptic bronchoscopic intubations. Flexible video or fiberoptic-mediated intubations will also be required when airway structures and tissue lack pliability or mobility due to invasive malignancy or scarring from previous surgery or radiation therapy. Therefore if the patient's airway structures or tissues are fixed and immobile, then the airway device used to navigate, visualize, and facilitate intubation must be flexible to effectively identify and traverse the larynx. Figure 15.13 demonstrates an awake flexible bronchoscopy intubation for a patient status post surgery and radiation therapy who presented with a large fixed right-sided mass.

Over the last decade rigid video laryngoscopes have become quite popular. Since these devices are fixed and require standard intraoral placement and tissue displacement for laryngeal visualization and intubation, they are not an acceptable alternative to flexible bronchoscopy for patients with head and neck pathology. In a recent study of 2004 patients, the risk of Glidescope[®] failure was high in patients with oral malignancies or those status post head and neck surgery or radiation therapy ^[68]. To reiterate, great care should be taken when managing patients who have been treated with

Fig. 15.10 Nasal tube modified with a metal right angle adaptor and mounted to a head drape



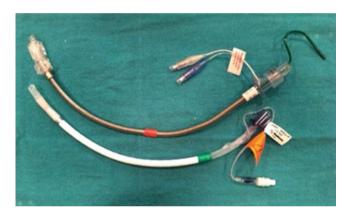


Fig. 15.11 Mallinckrodt[®] Laser-Flex[®] endotracheal tube (*top*) and the Medtronic[®] Laser shield II[®] (*bottom*)

radiation therapy of the head and neck. Not only are they difficult to intubate and ventilate, but fixed videoscopes prove to fail at a high rate and may make any subsequent "rescue" intubation attempts more difficult. Therefore, these patients require awake flexible video or fiberoptic bronchoscopicmediated intubations in order to assure patient safety. When performing TOR, our team of otolaryngologists finds tumor margin identification more challenging even after minor tissue disruption from standard laryngoscopy and prefers flexible video or fiberoptic airway management as a rule.

Intravenous Access

Due to superb dissection and surgical technique, even the most complex neck dissection surgery will typically be associated with minimal blood loss and limited insensible losses. However,

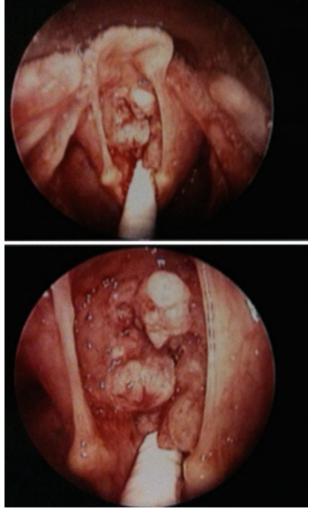


Fig. 15.12 Medtronic tube placed fiberoptically through friable laryngeal lesion without residual trauma

Fig. 15.13 An awake flexible bronchoscopy intubation for a patient status post surgery and radiation therapy who presents with a large fixed right-sided mass. (a) A endotracheal tube with embedded electrodes is placed because the patient had a left recurrent laryngeal nerve injury from their previous surgery and intraoperative nerve monitoring is planned (b–c)



because of the proximity of major vasculature to the surgical site, the possibility of sudden massive blood loss during invasive malignancy dissection, and the inaccessibility to place intravenous access intraoperatively due to patient positioning, we recommend placing at least one large bore (16 or 14 g) intravenous, and generally we have two IV sites available.

Monitoring

Standard ASA Monitoring

For a detailed discussion of intraoperative monitoring we refer the reader to Chap. 5, but will discuss the specifics of monitoring germane to patients undergoing head and neck cancer surgery here. Strategies for monitor placement that avoid the surgical field while limiting interference from surgical colleagues either touching (ECG) or leaning (noninvasive blood pressure cuff) on the equipment need to be employed. We recommend placing the ECG electrodes either on the chest below the level of the surgical site, on the patient's back or along their side (not over the chest or shoulders) and placing the BP cuff on the nonoperative side for unilateral neck dissections. For most bilateral neck dissections invasive arterial monitoring will likely be employed and the placement of the BP cuff will be less important. Using the lower extremity for BP cuff placement should be considered, but may not be accessible for cases requiring free tissue transfer reconstruction.

Intra-arterial Blood Pressure Monitoring

In most situations of major head and neck dissection an intraarterial (a-line) line will be placed for blood pressure

Fig. 15.14 Operating room organization for a right neck dissection



monitoring, frequent blood samples (arterial blood gas, hematocrit, electrolytes) and fluid replacement management. Placement will depend on surgical plan including anticipated free tissue harvest site. Typical sites for a-line placement include radial, axillary, femoral, or dorsalis pedis arteries. Placement before or after anesthetic induction will depend on the patient's comorbidities. A pre-induction a-line will be placed for those patients with significant cardiovascular or pulmonary disease or those suspected of being significantly dehydrated preoperatively.

Central Venous or Pulmonary Artery Pressure Monitoring

Central venous or pulmonary artery pressure monitors are usually not indicated for neck dissection surgery, unless the patient has significant pulmonary hypertension (Chap. 5). Although the use of central venous pressure (CVP) to determine and gauge intraoperative and postoperative fluid management has been called into question ^[69, 70], occasionally central venous access via the femoral vein will be required and is the only location available for intraoperative fluid and blood replacement. It should be noted that CVP monitoring from a femoral placed line is an especially poor guide of fluid management ^[71].

Although the relevance of CVP pressure monitoring and its correlation with intravascular fluid status has been called into question, it has been suggested that systolic (SPV) or pulse pressure variation (PPV) in intubated, ventilated patient correlates well with intravascular volume status and the responsiveness of fluid administration to optimize cardiac output ^[72, 73]. Simply SPV and PPV reflect the reduction in preload, stroke volume, and cardiac output with increased intrathoracic pressure associated with positive pressure ventilation. This reduction is greater in hypovolemic patients. Despite the fact that several companies have created equipment and algorithms to precisely interpret invasively and noninvasively SPV and PPV ^[74], to date this has not become standard of care intraoperatively. Although the use of intraoperative transesophageal echocardiography is considered the gold standard to determine and correlate volume status and optimal cardiac output, it requires skilled echocardiographers, is expensive and due to the surgical site, is impractical in head and neck cancer surgeries.

Positioning

Effective strategies to safely care for and monitor patients having surgery away from the anesthesia team is the hallmark of many otolaryngologic procedures, but given the surgical and patient complexity, it is particularly critical in surgeries for head and neck cancer. All neck dissection surgery will be conducted with the patient turned away from the anesthesiology team to some degree. Therefore, we recommend the use of extendible breathing circuits and the use of extra extension tubing for both intravenous and intra-arterial lines.

Unilateral neck dissection will usually be conducted with the patient turned 90° away from the anesthesiology team with the surgical site turned out to the room. Right neck dissections will be turned counterclockwise toward the anesthesia machine (Fig. 15.14) while left neck dissection will be turned 90° clockwise away from the anesthesia machine (Fig. 15.15). **Fig. 15.15** Operating room organization for a left neck dissection

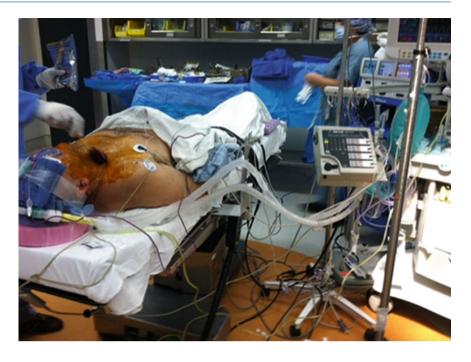




Fig. 15.16 Monitors are positioned and are applied from the foot of the bed up towards the patient

Bilateral neck dissections, major resections requiring free tissue reconstruction or those resections employing trans-oral robotic resections will be done with the patient turned 180° away from the anesthesiology team. While many anesthesiologists prefer to minimize patient movement post anesthetic induction, given the complexity of this patient population, our group prefers to keep the patient's head toward the anesthesiology equipment and only turn the patient once the airway is secure and the patient is stable under anesthesia. Although it has been suggested that this adds complexity, time and requires monitoring and lines to be disconnected prior to turning to avoid tangling and inadvertent line loss ^[75], our team has

developed an effective and time saving strategy that does not necessitate the removal of monitors or intravenous lines. This method affords us the ability to manage the airway and the anesthetic induction close to our equipment, and monitor the patient continuously during post induction positioning.

Strategy to Turn Patients 180°

We recommend placing all monitor cables along the right-hand side of the operating room bed. Intravenous and arterial lines are placed on the contralateral arm. All of the monitor cables should be looped down the operating room table and applied to the patient coming up from the foot of the bed (Fig. 15.16). The BP cuff cable should be directed toward the patient's feet to avoid kinking after the turn. Intravenous and arterial lines need to be long enough and applied straight down the arm without looping them back on themselves. Turning the operating table 180° can be done effortlessly and only requires that bed electrical cord be unplugged and endotracheal tube be disconnected. Figure 15.17 demonstrates the 180° turn in sequence.

Strategy to Turn Patients 90°

This strategy is identical to the 180° turn, but the monitor cables are brought in contact with the patient on the side opposite the surgery. If possible all intravenous and arterial lines are also placed in the arm opposite the surgical site. Turning the operating table 90° clockwise can then be done effortlessly.



Fig. 15.17 Demonstration of the sequence of events in a 180° turn following the induction of anesthesia (**a**) patient positioned with monitors draped properly. (**b**) Circuit disconnected. (**c**–**g**) Bed unlocked and

head of bed turned. (h) Circuit reconnected and bed locked in final position

Fluid and Transfusion Management

The goal of perioperative fluid and transfusion management is to optimize cardiac output and oxygen delivery (Chap.4). This optimization has been linked to reduced perioperative morbidity and mortality [76-78]. Although the importance of fluid and transfusion management is clear, there is conflicting and limited data to suggest the exact amount of intravenous fluid any one patient should receive and whether that fluid should be crystalloid, colloid, or contain red blood cells or other blood components ^[79]. Even though there is no exact formula, clinical judgment and evidence must guide fluid and transfusion management strategies [80]. While it is clear that giving too much intravenous fluid leads to significant postoperative complications including congestive heart failure, postoperative respiratory failure, and free flap failure [81], it is also apparent that inadequate fluid resuscitation causes hemodynamic instability and end-organ ischemia, increased blood viscosity, and possible hypercoagulability [82], and may lead to the increased use of vasopressor agents, which itself may be associated with increased morbidity and mortality ^[83, 84]. A recent study looked at the affect of using a conservative approach to transfusion protocols and found no adverse affect on free flap survival restricting transfusion to a hematocrit of 25 compared to 30^[85].

During all surgical cases including those of the head and neck, intraoperative fluid replacement and transfusion therapy are guided by a number of preoperative conditions and intraoperative events. Preoperative conditions include hydration, hematologic, cardiovascular, pulmonary, and renal status. Intraoperative conditions include insensible fluid loss and absolute blood loss. The rate of blood loss, the likelihood of further blood loss, and associated hemodynamic stability are also important to consider. Intraoperatively a number of measurable parameters will be monitored to gauge fluid resuscitative efforts. These include blood pressure, heart rate, urine output, hemoglobin, hematocrit, acid base balance, lactic acid, and electrolytes.

We generally use a balanced crystalloid solution for fluid replacement and gauge replacement with traditional methods of fluid assessment; heart rate, blood pressure, urine output, hemoglobin, hematocrit, and acid base status and lactate trends. Unless the patient has significant cardiovascular disease or becomes hemodynamically unstable we tolerate a lower hematocrit and generally don't transfuse patients with hematocrits greater than 25% unless hemodynamic instability develops or significant further blood loss is anticipated, or rapid blood loss has occurred.

Vasopressors

The use of intraoperative vasopressors seems to create significant conflict between anesthesiologists and otolaryngologists during head and neck reconstructive surgery. As

discussed, patients undergoing head and neck cancer resections are older with significant cardiovascular disease and questionable preoperative hydration status. Because intraoperative nerve monitoring will likely be employed, the use of neuromuscular blockade is contraindicated; therefore, a deep plane of anesthesia will be required to assure immobility. A deep plane of anesthesia in this patient population predictably causes hemodynamic instability. Although adequate fluid resuscitation will be employed (see above), over hydration increases postoperative complications. Therefore, the use of vasoactive substances will likely be needed during head and neck surgeries. Despite the fact that many otolaryngologists believe the use of such agents interferes with microvascular circulation causing vasospasm leading to flap failure, literature supports the use of such agents during free tissue transfer reconstruction surgery and has not been associated with such complications [86-88].

Temperature Management

The desired goal of temperature management is to maintain normothermia ^[89]. Both hyper ^[90] and hypothermia ^[91–93] can lead to significant intraoperative and postoperative complications. Generally limited neck dissection surgery is not associated with significant heat loss and temperature problems. In fact due to the limited exposure head and neck surgeries can be associated with hyper not hypothermia. In addition due to the limited surgical field, surface hot air systems can be applied and can effectively be used to maintain and re-warm patients ^[94].

This is not the case for the major head and neck reconstructive case requiring free tissue transfer surgery. In these cases the patient's head, neck, torso, and extremities are prepped. They get exceedingly cold even before the surgery has commenced. One solution is to minimize heat loss during the sterile prep by warming the room, but that might not be possible or practical. In these cases, surface hot air warming systems are inadequate due to the limited surface available for covering. We use a urinary catheter thermometer for accurate intraoperative temperature monitoring and an under body hot air system to maintain and re-warm patients after being exposed and prepped.

Postoperative Considerations

Otolaryngologist's Perspective

Patients in the immediate postoperative period are monitored in the recovery room and can be transferred to a standard room unless medical comorbidities require closer monitoring. Patients are checked in the immediate postoperative period for cranial nerve function, including the marginal mandibular, hypoglossal, and spinal accessory nerves. All patients will usually have a suction drain in place for 1-2 days, and the neck incision is monitored for evidence of hematoma or collection. Close monitoring of drain output for quantity and quality is performed. In the presence of a chyle leak, large cloudy output is appreciated and the fluid is sent to check for an elevated amylase level.

The morbidity of selective neck dissection, which is the most commonly performed surgery in contemporary times, is low. Only when the spinal accessory nerve is sacrificed, most patients have any shoulder discomfort and weakness ^[27, 95]. Furthermore, in rare instances where both internal jugular veins are either narrowed or sacrificed as a result of surgery, patients may experience significant postoperative facial edema. Most patients are back to work or their normal daily activities, in approximately 7–10 days after the surgery.

Anesthesiologist's Perspective

For most head and neck cancer resections, the patients are awake, alert, breathing spontaneously, extubated in the operating room and recovered in our standard Post Anesthesia Care Unit (PACU). Although rare, acute airway compromise represents the most catastrophic complication in head and neck surgery patients. Airway loss can be caused by a variety of issues including significant airway edema from local trauma, recurrent laryngeal nerve injury, aspiration of blood, close spaced bleeding causing tracheal compression and massive airway swelling from venous and lymphatic compression. Given the urgency and severity, it is critical for both anesthesiologists and otolaryngologist to work quickly and collaboratively. In most cases the airway will need to be secured. Given the possible anatomic derangements associated with surgery and bleeding, intubation using standard laryngoscopy, rigid video laryngoscopy and flexible bronchoscopy may prove difficult or impossible with a high failure rate. The use of an LMA should be considered and the placement of a surgical airway should not be delayed. Maintaining oxygenation, ventilation, and hemodynamic stability is paramount. Immediate re-exploration or evaluation will likely require an urgent return to the operating room.

Early hypertension may follow neck dissection possibly as a result of either carotid sinus denervation or a Cushing's reflex associated with intracranial hypertension ^[96]. If such hypertension is encountered its incidence is increased if the contralateral neck is subsequently dissected. Prolongation of the Q–T interval has been reported following right-sided neck dissection and under such some circumstances special attention should be given to potassium levels ^[97]. In most cases standard antihypertensive medications (e.g., nitrates, labetalol) are appropriate.

Conclusions

Head and neck cancer surgery can be a challenging undertaking for both surgeons and anesthesiologists. As with other head and neck procedures, most difficulties can be addressed through excellent communication and preparation.

Clinical Insights

For the Otolaryngologist (from the Anesthesiologist)

- Preoperative optimization of the cardiovascular, hematologic, and nutritional status will likely improve surgical outcome and decrease postoperative complications.
- Patients can be safely and efficiently intubated awake, don't underestimate the impact of prior head and neck surgery, radiation therapy, and the current airway pathology on the ability to intubate and ventilate the anesthetized patient.
- The necessary equipment and choice of endotracheal tube and intubation site depends on the surgical plan. For the anesthesiologist to be efficient and plan ahead there must be superb communication between the otolaryngologists and anesthesiologists.
- When the use of neuromuscular blocking agents are contraindicated patients require a significant level of anesthesia to assure immobility. This may cause significant cardiovascular instability requiring vasopressor agents to maintain homeostasis.
- Patient movement does not imply the patient is awake. If the patient begins to move during a surgical case, the best approach for the otolaryngologist is to inform the anesthesiologists.
- Good communication with the anesthesiology team and demarcation of the site of tissue harvest is critical to avoid wasted time and possibly the placement of compromising intravenous and arterial access.

For the Anesthesiologist (from the Otolaryngologist)

Head and neck cancer patients often present a special challenge to anesthesiologists, because these patients often have difficult airways that require preoperative planning. Depending on location or bulk of disease, these patients will require awake fiberoptic intubation or even awake tracheostomy. It is absolutely critical that the surgical and anesthesia teams communicate clearly about the anesthetic plan well in advance.

Some of the questions that can be useful to ask the surgeon are:

- Will we need to turn the patient once intubated?
- Will pathology specimens be sent for frozen section and will we be waiting for the results before extubation?
- Will you be planning to do a tracheostomy? Will this be at the beginning of the case, or more likely later? (Influences how to secure the endotracheal tube and circuit)
- What is the probability of cutting the jaw to access the tumor? (Influences whether nasotracheal intubation could be useful)
- Would you like to have muscle relaxation at the start of the case? If not, let me know when you would like to have the patient relaxed.
- In a patient with an existing tracheostomy: is this a simple trachestomy, or a laryngectomy? (Influences whether patient could be intubated from above in an emergency setting, as laryngectomy patients have end-tracheal stomas with no communication to the oral cavity and pharynx)
- Will this patient be undergoing microvascular reconstruction? (Influences length and complexity of surgery)

Clinical Pearls

For the Otolaryngologist (from the Otolaryngologist)

- Always consider awake fiberoptic (nasal or oral) intubation, or even awake tracheostomy, in patients with marginal airways or extreme trismus, especially as a result of prior surgery and/or radiation.
- When tracheostomy is indicated, its site should be excluded from surrounding neck incisions and skin flaps, in order to minimize the risk of wound infection from the clean-contaminated tracheotomy site.
- Large cranial nerves such as XI and XII typically stimulate noticeably even in the pharmacologically muscle-relaxed patient. CN VII (including the marginal mandibular branch) is not as reliable and can only be tested when the patient is not paralyzed.
- Spinal accessory nerve is subject to weakness even when anatomically preserved. Special care must be taken to avoid undue traction or cautery damage to the nerve. Postoperatively, CN XI function is best tested by shrugging against resistance, or lifting the arm over the head.

- Phrenic nerve injury results in ipsilateral paralysis of diaphragm. In most patients, this remains subclinical, but patients with chronic lung disease or marginal lung capacity may experience pulmonary complications.
- When possible, transection of motor nerves should be repaired by end-to-end anastomosis within 3 months.

For the Anesthesiologist (from the Anesthesiologist)

- Patients with a fixed larynx from radiation therapy are known to be difficult to ventilate and intubate and have a high GlideScope[®] failure rate.
- During an awake intubation, test the airway anesthesia with gentle oral pharyngeal suctioning with a yankauer. Don't proceed if the anesthesia is inadequate. Position the video screen directly in front of the videoscope operator, do not over sedate the patient, patient cooperation may be critical, don't inflate the endotracheal tube cuff until after the patient is asleep.
- After performing an awake intubation give a small dose of induction agent to initiate the general anesthetic. Large doses will cause hemodynamic instability.
- Airway compromise from mucus plugs and bronchospasm can still occur with a tracheostomy in situ.
- The use of neuromuscular blocking agents will likely be indicated during certain portions of a head and neck resection reconstruction surgery and contraindicated during others. If you have any doubt, do not use them and communicate with the otolaryngologists if you have or plan to use them.

References

- 1. Lore JM, Medina J. An atlas of head and neck surgery. Saunders, Philadelphia; 2004.
- Young JEM, Archibald SD, Shier KJ. Needle aspiration cytologic biopsy in head and neck masses. Am J Surg. 1981;142:484–9.
- Gleeson M, Herbert A, Richards A. Management of lateral neck masses in adults. BMJ. 2000;320:1521.
- 4. Jungehülsing M, Scheidhauer K, Damm M, et al. 2[F]-fluoro-2deoxy-D-glucose positron emission tomography is a sensitive tool for the detection of occult primary cancer (carcinoma of unknown primary syndrome) with head and neck lymph node manifestation. Otolaryngol Head Neck Surg. 2000;123:294.
- Pitman KT, Johnson JT, Myers EN. Effectiveness of selective neck dissection for management of the clinically negative neck. Arch Otolaryngol Head Neck Surg. 1997;123:917.
- Mendenhall WM, Amdur RJ, Williams LS, Mancuso AA, Stringer SP, Mendenhall NP. Carcinoma of the skin of the head and neck with perineural invasion. Head Neck. 2002;24:78–83.

- Jones HB, Sykes A, Bayman N, et al. The impact of lymphovascular invasion on survival in oral carcinoma. Oral Oncol. 2009;45:10–5.
- Ferlito A, Silver CE, Rinaldo A. Elective management of the neck in oral cavity squamous carcinoma: current concepts supported by prospective studies. Br J Oral Maxillofac Surg. 2009;47:5–9.
- Kowalski LP, Sanabria A. Elective neck dissection in oral carcinoma: a critical review of the evidence. Acta Otorhinolaryngol Ital. 2007;27:113–7.
- Genden EM, Ferlito A, Silver CE, et al. Contemporary management of cancer of the oral cavity. Eur Arch Otorhinolaryngol. 2010;267:1001–17.
- Asakage T, Yokose T, Mukai K, et al. Tumor thickness predicts cervical metastasis in patients with stage I/II carcinoma of the tongue. Cancer. 1998;82:1443–8.
- Vegers JW, Snow GB, van der Waal I. Squamous cell carcinoma of the buccal mucosa. A review of 85 cases. Arch Otolaryngol. 1979;105:192–5.
- Pradhan SA, D'Cruz AK, Gulla RI. What is optimum neck dissection for T3/4 buccal-gingival cancers? Eur Arch Otorhinolaryngol. 1995;252:143–5.
- Shah JP. Patterns of cervical lymph node metastasis from squamous carcinomas of the upper aerodigestive tract. Am J Surg. 1990;160:405–9.
- Gil Z, Fliss DM. Contemporary management of head and neck cancers. Isr Med Assoc J. 2009;11:296–300.
- Fein DA, Lee WR, Amos WR, et al. Oropharyngeal carcinoma treated with radiotherapy: a 30-year experience. Int J Radiat Oncol Biol Phys. 1996;34:289.
- 17. Shu CH, Cheng H, Lirng JF, et al. Salvage surgery for recurrent nasopharyngeal carcinoma. Laryngoscope. 2000;110:1483–8.
- Triboulet JP, Mariette C, Chevalier D, Amrouni H. Surgical management of carcinoma of the hypopharynx and cervical esophagus: analysis of 209 cases. Arch Surg. 2001;136:1164.
- Genden EM, Ferlito A, Rinaldo A, et al. Recent changes in the treatment of patients with advanced laryngeal cancer. Head Neck. 2008;30:103–10.
- Molinari R, Cantu G, Chiesa F, Grandi C. Retrospective comparison of conservative and radical neck dissection in laryngeal cancer. Ann Otol Rhinol Laryngol. 1980;89:578.
- Hirabayashi H, Koshii E, Uno K, et al. Extracapsular spread of squamous cell carcinoma in neck lymph nodes: prognostic factor of laryngeal cancer. Laryngoscope. 1991;101:502–6.
- 22. Baker SR, Krause CJ. Carcinoma of the lip. Laryngoscope. 1980;90:19–27.
- Shaha AR. Management of the neck in thyroid cancer. Otolaryngol Clin N Am. 1998;31:823.
- Messina JL, Reintgen DS, Cruse CW, et al. Selective lymphadenectomy in patients with Merkel cell (cutaneous neuroendocrine) carcinoma. Ann Surg Oncol. 1997;4:389–95.
- Yiengpruksawan A, Coit DG, Thaler HT, Urmacher C, Knapper WK. Merkel cell carcinoma: prognosis and management. Arch Surg. 1991;126:1514.
- 26. Veness MJ, Palme CE, Smith M, Cakir B, Morgan GJ, Kalnins I. Cutaneous head and neck squamous cell carcinoma metastatic to cervical lymph nodes (nonparotid): a better outcome with surgery and adjuvant radiotherapy. Laryngoscope. 2003;113:1827–33.
- 27. Ferlito A, Pellitteri PK, Robbins KT, et al. Management of the neck in cancer of the major salivary glands, thyroid and parathyroid glands. Acta Otolaryngol. 2002;122:673–8.
- Hocwald E, Korkmaz H, Yoo GH, et al. Prognostic factors in major salivary gland cancer. Laryngoscope. 2001;111:1434–9.
- Skolnik EM, Friedman M, Becker S, Sisson GA, Keyes GA. Tumors of the major salivary glands. Laryngoscope. 1977;87:843–61.
- Martin H, Martin HE. Surgery of head and neck tumors. New York: Hoeber-Harper; 1957.
- Fisch UP, Sigel ME. Cervical lymphatic system as visualized by lymphography. Ann Otol Rhinol Laryngol. 1964;73:870–82.

- 32. Rouviere H. Lymphatic system of the head and neck. Edwards Bros: Anatomy of the Human Lymphatic System Ann Arbor; 1938.
- Janfaza P, Nadol JB, Galla R, et al. Surgical anatomy of the head and neck. Philadelphia: Lippincott Williams & Wilkins; 2000.
- Ferlito A, Rinaldo A, Silver CE, et al. Neck dissection: then and now. Auris Nasus Larynx. 2006;33:365–74.
- 35. Medina JE. A rational classification of neck dissections. Otolaryngol Head Neck Surg. 1989;100:169–76.
- 36. Robbins KT, Clayman G, Levine PA, et al. Neck dissection classification update: revisions proposed by the American Head and Neck Society and the American Academy of Otolaryngology-Head and Neck Surgery. Arch Otolaryngol Head Neck Surg. 2002;128:751–8.
- Bocca E, Pignataro O, Oldini C, Cappa C. Functional neck dissection: an evaluation and review of 843 cases. Laryngoscope. 1984;94:942–5.
- Kreimer AR, Bhatia RK, Messeguer AL, et al. Oral human papillomavirus in healthy individuals: a systematic review of the literature. ALSex Transm Dis. 2010;37386–91.
- D'Souza G, Agrawal Y, Halpern J, et al. Oral sexual behaviors associated with prevalent oral human papillomavirus infection. J Infect Dis. 2009;1:1991263–9.
- Chaturvedi AK, Engels EA, Pfeiffer RM, et al. Human papillomavirus and rising oropharyngeal cancer incidence in the United States. J Clin Oncol. 2011;29:4294–301 [Epub 2011 Oct 3].
- Ries LAG, Eisner MP, Kosary CL, et al. SEER Cancer Statistics Review, 1975–2002. Bethesda: National Cancer Institute; 2005.
- 42. Ridge JA, Glisson BS, Lango MN, et al. Head and neck tumors. In: Pazdur R, Wagman LD, Camphausen KA, Hoskins WJ, editors. Cancer management: a multidisciplinary approach. 11th ed., Philadelphia; 2008.
- Castellsague X, Quintana MJ, Martinez MC, et al. The role of type of tobacco and type of alcoholic beverage in oral carcinogenesis. Int J Cancer. 2004;108:741–9.
- Copper MP, Smit CF, Stanojcic LD, et al. High incidence of laryngopharyngeal reflux in patients with head and neck cancer. Laryngoscope. 2000;110:1007–11.
- Lee TH, Marcantonio ER, Mangione CM, et al. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. Circulation. 1999;100:1043–9.
- 46. Fleisher LA, Beckman JA, Brown KA, et al. ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: a report of the American College of Cardiology/ American Heart Association Task Force on Practice Guidelines [published correction appears in J Am Coll Cardiol 2008; 52: 794–797]. J Am Coll Cardiol. 2007;50:1707–32.
- Reilly DF, McNeely MJ, Doerner D, et al. Self-reported exercise tolerance and the risk of serious perioperative complications. Arch Intern Med. 1999;159:2185–92.
- 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; 98:1269.
- 49. van Bokhorst-de van der Schueren MA, van Leeuwen PA, et al. Assessment of malnutrition parameters in head and neck cancer and their relation to postoperative complications. Head Neck. 1997;19:419–25.
- Ferrier MB, Spuesens EB, Le Cessie S, et al. Comorbidity as a major risk factor for mortality and complications in head and neck surgery. Arch Otolaryngol Head Neck Surg. 2005;131:27–32.
- de Cassia Braga Ribeiro K, Kowalski LP. APACHE II, POSSUM, and ASA scores and the risk of perioperative complications in patients with oral or oropharyngealcancer. Arch Otolaryngol Head Neck Surg. 2003;129:739–45.
- Oh SW, Yoon YS, Shin SA. Effects of excess weight on cancer incidences depending on cancer sites and histologic findings among men: korea national health insurance corporation study. J Clin Oncol. 2005;23:4742–54.

- Whiteman MK, Hillis SD, Curtis KM, et al. Body mass and mortality after breast cancer diagnosis. Cancer Epidemiol Biomarkers Prev. 2005;14:2009–14.
- Meyerhardt JA, Catalano PJ, Haller DG, et al. Influence of body mass index on outcomes and treatment-related toxicity in patients with colon carcinoma. Cancer. 2003;98:484–95.
- Amling CL, Riffenburgh RH, Sun L, et al. Pathologic variables and recurrence rates as related to obesity and race in men with prostate cancer undergoing radical prostatectomy. J Clin Oncol. 2004;22: 439–45.
- El-Orbany M, Woehlck HJ. Difficult mask ventilation. Anesth Analg. 2009;109:1870–80.
- Sachin K, Lizabeth M, Amy M, et al. Prediction and outcomes of impossible mask ventilation a review of 50,000 anesthetics. Anesthesiology. 2009;110:891–7.
- Shin DM, Khuri FR. Advances in the management of recurrent or metastatic squamous cell carcinoma of the head and neck. Head Neck. 2011. doi:10.1002/hed.21910 [November 2; Epub ahead of print]
- Fakih M. Management of anti-EGFR-targeting monoclonal antibody-induced hypomagnesemia. Oncology. 2008;22:74–6.
- 60. Klastersky J. Adverse effects of the humanized antibodies used as cancer therapeutics. Curr Opin Oncol. 2006;18:316–20.
- Hoag JB, Azizi A, Doherty TJ, et al. Association of cetuximab with adverse pulmonary events in cancer patients: a comprehensive review. J Exp Clin Cancer Res. 2009;28:113.
- Moores LK. Smoking and postoperative pulmonary complications: an evidence-based review of the recent literature. Clin Chest Med. 2000;21:139–46.
- 63. Warner MA, Offord KP, Warner ME, et al. Role of preoperative cessation of smoking and other factors in postoperative pulmonary complications: a blinded prospective study of coronary artery bypass patients. Mayo Clin Proc. 1989;64:609–16.
- Spies CD, Rommelspacher H. Alcohol withdrawal in the surgical patient: prevention and treatment. Anesth Analg. 1999;88:946–54.
- Gordon AJ, Olstein J, Conigliaro J. Identification and treatment of alcohol use disorders in the perioperative period. Postgrad Med. 2006;119:46–55.
- Kork F, Neumann T, Spies C. Perioperative management of patients with alcohol, tobacco and drug dependency. Curr Opin Anaesthesiol. 2010;23:384–90.
- 67. Haas RE, Kervin MW, Ramos P, et al. Occlusion of a wire-reinforced endotracheal tube in an almost completely edentulous patient. Mil Med. 2003;168:422–3.
- 68. Aziz MF, Healy D, Kheterpal S, et al. Routine clinical practice effectiveness of the Glidescope in difficult airway management: an analysis of 2,004 Glidescope intubations, complications, and failures from two institutions. Anesthesiology. 2011;114:34–41.
- 69. Magder S, Bafaqeeh F. The clinical role of central venous pressure measurements. J Intensive Care Med. 2007;22(1):44–51.
- Marik PE, Baram M, Vahid B. Does central venous pressure predict fluid responsiveness? A systematic review of the literature and the tale of seven mares. Chest. 2008;134(1):172–8.
- Desmond J, Megahed M. Is the central venous pressure reading equally reliable if the central line is inserted via the femoral vein. Emerg Med J. 2003;20(5):467–9.
- Michard F. Changes in arterial pressure during mechanical ventilation. Anesthesiology. 2005;103:419–28.
- Cannesson M, Attof Y, Rosamel P, Desebbe O, et al. Respiratory variations in pulse oximetry plethysmographic waveform amplitude to predict fluid responsiveness in the operating room. Anesthesiology. 2007;106:1105–11.
- Cannesson M, Musard H, Desebbe O, et al. The ability of stroke volume variations obtained with Vigileo/FloTrac system to monitor fluid responsiveness in mechanically ventilated patients. Anesth Analg. 2009;108:513–7.
- Chi JJ, Mandel JE, Weinstein GS. Anesthetic considerations for transoral robotic surgery. Anesthesiology Clin. 2010;28:411–22.

- Gan TJ, Soppitt A, Maroof M, et al. Goal-directed intraop- erative fluid administration reduces length of hospital stay after major surgery. Anesthesiology. 2002;97:820–6.
- 77. Pearse R, Dawson D, Fawcett J, Rhodes A, Grounds RM, Bennet ED. Early goal-directed therapy after major surgery reduces complications and duration of hospital stay. A randomised, controlled trial. Crit Care. 2005;9:R687–93.
- Wakeling HG, McFall MR, Jenkins CS, et al. Intraoperative oesophageal Doppler guided fluid management shortens postoperative hospital stay after major bowel surgery. Br J Anaesth. 2005;95:634–42.
- Bundgaard-Nielsen M, Secher NH, Kehlet H. 'Liberal' vs. 'restrictive' perioperative fluid therapy—a critical assessment of the evidence. Acta Anaesthesiol Scand. 2009;53:843–51.
- Practice Guidelines for Perioperative Blood Transfusion and Adjuvant Therapies. Anesthesiology. 2006;105:198–208.
- Namdar T, Bartscher T, Stollwerk PL, et al. Complete Free Flap loss due to extensive hemodilution. Microsurgery. 2010;30: 214–7.
- Doi T, Sakurai M, Hamada K, et al. Plasma volume and blood viscosity during 4 h sitting in a dry environment: effect of prehydration. Aviat Space Environ Med. 2004;75(6):500–4.
- Collier B, Dossett L, Mann M, et al. Vasopressin use is associated with death in acute trauma patients with shock. J Crit Care. 2010;25:173.e9–14.
- Sperry JL, Minei JP, Frankel HL, et al. Early use of vasopressors after injury: caution before constriction. J Trauma. 2008;64:9–14.
- Rossmiller S, Cannady S, Tamer G, et al. Transfusion criteria in free flap surgery. Otolaryngol Head Neck Surg. 2010;142: 359–64.
- Pattani KM, Byrne P, Boahene K, et al. What makes a good flap go bad? A critical analysis of the literature of intraoperative factors related to free flap failure. Laryngoscope. 2010;120:717–23.
- Monroe MM, Cannady SB, Ghanem TA, et al. Safety of vasopressor use in head and neck microvascular reconstruction: a prospective observational study. Otolaryngol Head Neck Surg. 2011;144:877–82.
- Monroe MM, McClelland J, Swide C, et al. Vasopressor use in free tissue transfer surgery. Otolaryngol Head Neck Surg. 2010;142: 169–73.
- Esnaola NF, Cole DJ. Perioperative normothermia during major surgery: is it important? Adv Surg. 2011;45:249–63.
- Liu YJ, Hirsch BP, Shah AA, et al. Mild intraoperative hypothermia reduces free tissue transfer thrombosis. J Reconstr Microsurg. 2011;27:121–6.
- Sumer BD, Myers LL, Leach J, et al. Correlation between intraoperative hypothermia and perioperative morbidity in patients with head and neck cancer. Arch Otolaryngol Head Neck Surg. 2009;135:682–6.
- Rajagopalan S, Mascha E, Na J, Sessler D. The effects of mild perioperative hypothermia on blood loss and transfusion requirement. Anesthesiology. 2008;108:71–7.
- Leslie K, Sessler DI. Perioperative hypothermia in the high-risk surgical patient. Best Pract Res Clin Anaesthesiol. 2003;17:485–98.
- Moola S, Lockwood C. Effectiveness of strategies for the management and/or prevention of hypothermia within the adult perioperative environment. Int J Evid Based Healthc. 2011;9: 337–45.
- Terrell JE, Welsh DE, Bradford CR, et al. Pain, quality of life, and spinal accessory nerve status after neck dissection. Laryngoscope. 2000;110:620–6.
- Celikkanat S, Akyol M, Koc C, et al. Postoperative hypertension after radical neck dissection. Otolaryngol Head Neck Surg. 1997;117:91–2.
- Otteni J, Pottecher T, Bronner G, et al. Prolongation of the Q-T interval and sudden cardiac arrest following right radical neck dissection. Anaesthesiology. 1983;59:458–61.

Head and Neck Cancer Surgery II: Reconstruction

16

Adam I. Levine, Samuel DeMaria, Jr., and Brett A. Miles

Introduction

Patients presenting for head and neck cancer resections may eventually require surgical reconstruction to fix anatomic defects and maintain aerodigestive and masseter function. Complex head and neck reconstructive surgery represents a unique interaction between the reconstructive surgeon and the anesthesiologist. The management of head and neck pathology including trauma, congenital deformities, as well as benign and malignant tumors requires a substantial array of reconstructive techniques. Over the last several decades the most significant reconstructive advancement from the surgical standpoint is the technique of free tissue transfer, the so-called "free flap." This procedure involves harvesting distant tissues and transferring reconstructive elements such as bone, soft tissue, and muscle to ablative defects of the head and neck (see Fig. 16.1).

Overview

Microvascular arterial and venous anastomoses are used to reestablish the blood supply. Thus, free tissue transfer is essentially an autotransplantation procedure. Significant advances in training and technology have allowed free tissue transfer to become the standard reconstruction in many institutions for major oncologic head and neck surgery and a variety of other reconstructive situations.

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The inherent complexity of these procedures coupled with the length of time required to complete the operation often results in anesthetic challenges. Perhaps the most important factor inherent to success of a free flap is communication between the reconstructive surgeon and anesthesiologist. The wide variety of pathology, reconstructive techniques, and previous therapy dictates a truly customized approach for the surgical planning for each new patient. This is especially relevant in the management of the airway, which is often affected by the primary pathology, previous radiotherapy and/or previous surgical manipulation. A formal preoperative discussion with members of the surgical team will improve surgical and anesthetic management, improve outcomes. and avoid conflicts regarding patient management.

The topic of Head and Neck Cancer Resection surgery is discussed in great detail in Chap. 15, from both the otolaryngologist and anesthesiologist perspective. This chapter will focus on elements of care related to complex head and neck reconstruction with specific focus on free tissue transfer techniques. An understanding of the surgical techniques and anesthetic factors affecting head and neck reconstruction is critical for successful management. Anesthetic topics unique to head and neck reconstruction and those that require additional emphasis will be discussed here.

Preoperative Considerations

Otolaryngologist's Perspective

The indication for complex reconstructive surgery often occurs in the context of the medically compromised patient. Patients requiring head and neck reconstruction are usually elderly and often have significant medical, nutritional, and social comorbidities. Although advanced age has been associated with adverse outcomes, it is not an independent risk factor for complications. However with advancing age comes increased medical comorbidities (Table 16.1)^[4, 17].

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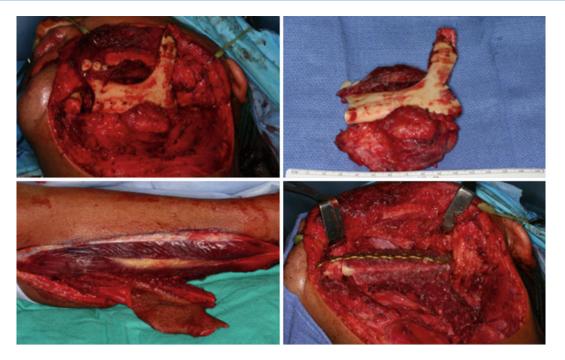


Fig. 16.1 Free tissue transfer reconstructive technique, in this case a fibular bone graft is transferred for mandibular reconstruction after resection of an oral cavity squamous cell carcinoma. (a) Mandibular

resection (*upper left*). (**b**) Tumor specimen (*upper right*). (**c**) Fibular free tissue harvest (*lower left*). (**d**) Mandibular reconstruction with fibular graft (*lower right*)

 Table 16.1
 Factors related to adverse outcomes in major head and neck surgery

Factors associated with adverse outcomes in major head and neck
reconstructive surgery
ASA status >3 ^[1]
High volume resuscitation >7 L $^{[2,3]}$
Length of surgery $(>10 h)$ ^[4-6]
Current smoking ^a ^[3, 7, 8]
Osteoradionecrosis ^[9, 10]
History of radiation therapy ^[6, 11, 12]
Vasopressor infusion ^[13]
Hypoalbuminemia ^[14]
Low volume medical centers ^[15]

^aIt should be noted that smoking has not been shown to significantly correlate with free tissue transfer failure ^[8, 16]

The risk factors associated with head and neck malignancy such as alcohol and tobacco abuse often result in secondary cardiopulmonary disease, and increase the risk of perioperative complications. Investigations have shown that the majority of patients undergoing free tissue transfer reconstruction have significant microvascular disease and have illustrated the negative impact of medical comorbidities, extended length of the procedure, and large volume fluid replacement in this patient population ^[3, 6, 18–22]. The predominant major complications associated with these reconstructions are rarely technical and pulmonary, cardiac, and infectious complications predominate ^[1]. Avoiding major medical complications is perhaps the most challenging aspect in the management of patients requiring complex head and neck reconstruction and can be addressed with preoperative medical evaluation and optimization.

Many patients undergoing major head and neck surgery have compromised nutrition. This is due to factors related to malignant processes, previous therapy/surgery compromising oral intake, and factors such as alcohol consumption and medical comorbidities ^[14]. Low plasma albumin concentration is associated with postoperative morbidity and increased mortality ^[23, 24].

Patients with a history of radiation therapy to the head and neck warrant special consideration as radiotherapy often results in significant fibrosis within the treatment fields. Standard pre-anesthetic airway assessments such as mouth opening, thyromental distance, and Mallampati classification may be deceptive in previously radiated patients who often have limited neck mobility, and fibrosis of tissues such as the tongue base and neck, prohibiting the use of standard airway techniques. This may be true even in the absence of significant trismus or obvious external airway deformities.

Anesthesiologist's Perspective

Preoperative assessment and medical optimization is critical for improved surgical outcome in patients having free tissue transfer and reconstructive surgery. Many patients presenting for reconstructive surgery have advanced age and significant cardiovascular, pulmonary, hematologic, hepatic, and renal disease. They may have nutritional deficits and significant exposure histories including tobacco, alcohol, and other carcinogens (asbestos). In many studies age, ASA score, low body mass index, anemia, and tobacco consumption correlated with development of major perioperative complications; recent weight loss and alcohol abuse correlated with prolonged length of hospital stay. Although not always possible (e.g., age), some of these parameters (e.g., medical comorbidities, tobacco and alcohol ingestion, nutritional status, preoperative hemoglobin) can be optimized preoperatively, which may lower perioperative risk and improve surgical outcome.

Although it is covered in great detail in the previous chapter (Chap. 15), it bears repeating that any patient presenting for major head and neck surgery needs a thorough airway examination including, a history, physical and imaging assessment. Tumors of the head and neck may be rapidly growing, compromise secretion management and are quite friable. In addition many of these patients present for cancer recurrence and have already undergone a combination of surgery, chemotherapy, and radiation therapy. Each one of these may be a relative indication for an awake intubation, but in combination more likely represent an absolute indication for an awake flexible bronchoscopic-mediated intubation. In our practice we make the assumption that patients presenting for a major head and neck cancer resection or reconstruction will require their airway managed awake unless a thorough evaluation of all existing data proves otherwise.

Many of these patients will present with an in situ tracheostomy. We refer the reader to Chap. 19 for a thorough discussion of tracheostomies and their management. Tracheostomy does not preclude a thorough airway examination including the tracheostomy site and the tracheostomy appliance (e.g., size and style; metal vs. plastic, cuffed vs. uncuffed). It is also critical to have a working knowledge of the patient's upper airway anatomy and whether or not they can be ventilated and/or intubated from above should that be necessary. This can be accomplished by discussing the case with the otolaryngologic team prior to managing the tracheostomy. Generally, these patients will present spontaneously breathing, and it is important to determine whether or not positive pressure ventilation can be delivered via the mouth and nose and/or the tracheostomy appliance.

Intraoperative Considerations

Otolaryngologist's Perspective

Major ablative and reconstructive surgery of the head and neck requires significant surgical planning and often the defect dictates the plan. The sequence of surgery is variable

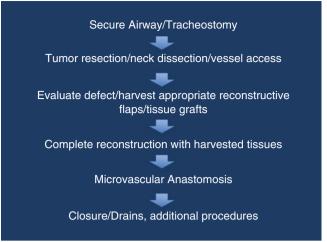


Fig. 16.2 General surgical sequence for major head and neck ablative and reconstructive surgery

and often two surgical teams will function together in order to save time. The general surgical sequence can be viewed in Fig. 16.2.

The operative times associated with each step in the sequence are extremely variable depending on the size, location, and type of pathology and the reconstructive plan. The tumor ablation may occur with simultaneous harvest of reconstruction flaps depending on the donor site. Donor sites such as the radial forearm, anterior lateral thigh, fibula and rectus abdominus are amenable to a two-team approach, which shortens the operative time (Fig. 16.3). Donor sites such as the lattissimus dorsi, or scapular system of flaps do not allow for a two-team harvest due to positioning issues and longer operative times are required for these reconstructive options (Fig. 16.4).

Positioning

Careful patient positioning allows the necessary surgical access while reducing the risk of peripheral nerve injury, ocular injury, or soft tissue pressure necrosis. A careful discussion regarding potential harvest sites will allow for placement of external warming devices, monitoring devices, arterial/central lines, and other required equipment without obstructing surgical access. The airway circuit should be positioned to allow for 180° rotation of the patient and provide access for trouble shooting monitoring/ventilation problems. Often the exposure of graft sites and potential donor sites will require exposure of a significant body surface area and careful planning will still allow for appropriate warming/monitoring without hindering the surgeon's ability to harvest reconstructive flaps and grafts. Warming devices should be of the "under body" type as most conventional warmers are not easily or appropriately placed on top of the patient with multiple harvest sites demarcated. In general it



Fig. 16.3 (a) The complex room setup for a double surgical team during a radial forearm free flap reconstruction. The operating microscope is at the far left, two surgical teams, one performing the microvascular anastomosis works under the microscope, while the other team can

operate on the forearm. The anesthesiologists are at the far right. (b) Notice the two surgical teams are able to work side by side and simultaneously during the free flap microvascular anastomosis and surgery on the forearm harvest site

is advisable to determine the potential harvest sites and allow this to drive monitor placement. Arterial lines, central lines, and other invasive monitors should be placed away from the proposed graft sites to simplify surgical preparation. Reconstructive flaps such as the fibular and radial forearm flap are often harvested under tourniquet control and therefore should not be used for invasive monitoring, external monitoring or venous access. Posterior harvest sites, such as the lattissimus dorsi and scapular flaps, generally require a vacuum-assisted surgical positioning device (i.e., bean bags, gel devices), which should be in place prior to patient transfer to the operating table. **Fig. 16.4** Positioning for scapular free tissue harvest. Two-team flap harvest is not possible with scapular or lattissimus harvest, due to arm retraction required. Note the significant exposure necessary in this type of reconstruction mandating appropriate intravenous access and warming device placement



Vascular Access

Vascular access in head and neck surgery is critical to provide reliable access for prolonged surgery without compromising donor site access. In general, peripheral intravenous lines should be discussed with the surgeon prior to placement to avoid placing the cannula in a proposed graft site and compromising venous anatomy, or creating logistical problems with donor site preparation. For central access, femoral lines contralateral to proposed donor sites such as the fibula or anterior lateral thigh are preferred (relative to subclavian, internal jugular lines) to avoid line management issues within the head and neck region. Regarding arterial line placement, the line should generally be placed in the radial artery opposite to the site of proposed donor sites if radial forearm free tissue transfer is performed. This will often be in the patient's dominant upper extremity as the flap harvest is generally performed on the nondominant side if possible.

Antibiotic Prophylaxis

Wound infection rates range from 20 to 25% in major head and neck surgery and gram positive and negative coverage is indicated. Interestingly, the type and location of the surgery are likely more important than the selection of the antibiotic coverage in terms of the infection risk ^[25, 26]. Due to the increased bacterial load, surgeries that violate the oral cavity, nasal cavity, and pharyngeal mucosa are particularly prone to postoperative wound infections, relative to procedures which do not violate the mucosa. Because the majority of head and neck malignancy (excluding thyroid malignancies and isolated neck dissections) occur on the mucosal surface of the upper aerodigestive tract, appropriate antibiotic prophylaxis is paramount. Optimal administration is 30 min prior to surgical incision with additional doses often required for prolonged procedures (>5 h), significant blood loss (>1500 mL), and tourniquet usage. The antibiotic coverage should continue for a total of 24 h after surgery ^[27, 28].

Management of Blood Pressure

While the intraoperative management of blood pressure generally falls to the anesthesiologist, there are several issues related to this of concern to the operating surgeon. Often the assumption is made that the surgeon merely wants the blood pressure as low as possible to prevent intraoperative blood loss. This is an incorrect assumption, however, and from a surgical standpoint the optimal blood pressure varies throughout the surgery. During stages of the surgery associated with significant blood loss such as maxillectomy, vascular tumor resections, or harvesting of reconstructive flaps associated with bone or muscle (which are not under tourniquet control) the maximum tolerable hypotension is desired from the surgeon's standpoint. This principle must be tailored to the individual as the various comorbidities inherent to this patient population may dictate that deliberate hypotension is untenable. Stages performed under tourniquet control are not associated with significant blood loss and may be safely performed

with an increased mean arterial pressure and more normotensive conditions.

A special situation occurs during free tissue transfer procedures with the specific aim of optimal blood flow through the microvascular anastomosis in order to perfuse the transplanted tissues. Generally, microvascular anastomosis of the vessels is performed with little patient stimulation, and therefore attention to the blood pressure during this portion of the procedure is critical to provide adequate perfusion pressure to prevent thrombosis and to maintain perfusion within the flap. Hyperoxia, hypocapnia, and hypovolemia (factors that variably result in decreased cardiac output or induce local vasoconstriction) should be avoided during this stage of the reconstruction ^[2].

Transfusion

The optimal transfusion trigger during major head and neck surgery to maintain perfusion and oxygen delivery remains unknown but a target hematocrit of >30 is generally suggested. Normovolemic hemodilution is performed by some groups but the benefits in terms of flap perfusion remain under investigation [29, 30]. Preoperative anemia has been associated with adverse outcomes yet overcorrection is unnecessary [11, 30]. There is some evidence that hematocrits > 25 will lead to decreased complication rates [31]. Therefore, current evidence indicates that a transfusion trigger hematrocrit range of 25-30 is reasonable during free tissue transfer procedures. Preoperative factors associated with transfusion include female sex, low body mass index, advanced tumor stage, and preoperative hemoglobin level. Additionally, the type of reconstruction affects the transfusion likelihood with osseous harvests predicting transfusion ^[32]. Interestingly, large volume transfusions (>3-4 U) during major head and neck surgery have been associated with adverse outcomes, which may be indicative of large blood loss, preoperative anemia, or prolonged surgery ^[33]. Historically, within the surgical literature, large volume transfusion was thought to be associated with adverse long-term survival outcomes in head and neck cancer surgery. Recent evidence indicates this is likely a reflection of advanced disease state driving major surgery rather than being directly attributable to the transfusion [34].

Fluid Management

In the setting of major head and neck surgery, intraoperative fluid management is often poorly understood by both the anesthesia and surgical teams. While opinions regarding fluid management vary widely, a review of the available scientific literature offers some insight into appropriate fluid management. Large volume fluid resuscitations are associated with adverse outcomes and flap complications ^[3, 22]. There are several misconceptions, which lead to inappropriate fluid administration during major head and neck reconstruction procedures. Preoperative "fluid loading" in patients who have been NPO prior to surgery or in anticipation of prolonged surgery is questionable in patients with a normal circulation. In most situations, crystalloid-loading results in an excess of fluid without improved cardiovascular or renal function. In fact, this fluid will be redistributed interstitially and results in unnecessary edema and reduced tissue oxygenation^[35]. Aggressive fluid administration early in the case in anticipation of significant third spacing, which is associated with other major surgery, is not appropriate for major head and neck surgery as the principle of third spacing is of minimal importance in the majority of these cases. Conservative fluid management is critical to improve outcomes in major head and neck reconstructive surgery ^[35–37]. Some centers advocate cardiac output monitoring to optimize fluid management; however, this has not gained widespread acceptance at this time, likely because of the cost of these devices ^[36, 38]. Interestingly, hypertonic saline may increase blood flow to microvascular transfers but cannot be recommended with currently available evidence [37, 39]. Moderate hemodilution with maintenance of cardiac output with low peripheral resistance should be the ultimate goal of fluid administration during major head and neck surgery ^[37].

The use of volume expanders such as synthetic colloids and albumin has not been extensively investigated in the setting of head and neck reconstruction. The majority of investigative work in this area remains controversial and there is no clearly superior volume expander currently recommended for major head and neck surgery ^[8, 40]. There is currently little evidence to support or discourage the use of colloid volume expanders in head and neck reconstructive surgery. Nevertheless, routine use of volume expanders is likely unnecessary and large amounts of volume expanders may alter coagulation and fluid physiology to the patient's detriment ^[41].

Intraoperative Vasopressors

Many microvascular surgeons discourage the use of intraoperative vasopressors in order to avoid microvascular spasm and peripheral vasoconstriction during free tissue transfer procedures. This clinical practice is not supported by the current literature however and the use of intraoperative, moderate dose bolus vasoactive drugs is not associated with adverse affects on flap perfusion ^[42]. Recent investigations have shown that the use of intraoperative vasopressors is more common than previously realized and does not result in an increase of free tissue transfer loss ^[43, 44]. The use of intraoperative vasopressor infusions may decrease flap blood flow and is generally discouraged given the available literature ^[13], however severe, life-threatening intraoperative hypotension requires systemic vasopressor infusion and the survival of the free tissue transfer becomes of secondary importance in this rare situation. In the setting of free tissue transfer there is some data that dobutamine may offer an advantage over dop-amine in these situations^[45].

Anticoagulation

Another controversial topic amongst reconstructive surgeons is the use of intraoperative/postoperative anticoagulation for free tissue transfer. The most common causes of free tissue transfer failure are vessel thrombosis and hematoma, which [12] represents an interesting clinical dichotomy Anticoagulation therapy may offer protective effects in terms of vessel thrombosis while predisposing to postoperative bleeding and hematoma simultaneously. Protocols ranging from subcutaneous heparin to intravenous heparin infusions have been utilized to prevent vascular thrombosis. Postoperative regimens including heparin, aspirin, and lowmolecular-weight heparins are common and varied. A recent survey of microvascular surgeons in North America revealed that >75% utilized some form of anticoagulation therapy ^[46]. Interestingly, despite multiple investigations, no clear anticoagulation protocol has been established and there are no formal guidelines. In fact the use of anticoagulation in the setting of microvascular reconstruction continues to remain controversial and no clear benefit in terms of overall reduction of thrombosis rates or flap failure have been definitively demonstrated^[47]. Therefore, while routine intraoperative use is not required the microvascular surgeon may request administration of heparin, aspirin, ketoralac, or other anticoagulation depending on the clinical situation.

Dextran

There exists a large emphasis on the use of low-molecularweight dextran in the microvascular surgery literature in order to decrease vascular thrombosis. The administration of intravenous dextran has multiple effects on the coagulation cascade and decreases platelet adhesion, erythrocyte aggregation and reduces von Willebrand factor. In addition, dextran possesses some thrombolytic activity via plasminogen activation ^[41]. Nevertheless, significant systemic morbidity including pulmonary and cardiac effects as well as anaphylaxis has been reported with the use of low-molecular-weight dextran. Randomized data indicates that the use of low-molecular-weight dextran confers a 3–7 times relative risk of systemic complications when compared to aspirin therapy ^[48, 49]. Additionally, no statistically significant differences in vessel patency rates or improved outcomes have been demonstrated ^[50–52].

Ventilation

The ventilation of patients undergoing reconstructive flap surgery is generally straightforward; however, some considerations warrant review. Significant hyperoxia causes vasoconstriction and reduced functional capillary density. While there is some evidence that oxygen supplementation may increase the amount of oxygen dissolved in plasma and increase oxygen tension within the flap, there is little evidence of improved outcome. Recent investigations interrogating the microcirculation have demonstrated that hyper-oxygenation results in arteriolar vasoconstriction, resulting in a reduction of microvascular flow, and therefore routine hyperoxia is not recommended ^[2].

Anesthesiologist's Perspective

Intraoperative Events

It is critical for the anesthesiology and otolaryngologic team to have open and continuous communication concerning the planned intraoperative events mentioned above (i.e., periods where hypotension/normotension are required). It is also critical for the anesthesiology team caring for patients undergoing major head and neck resection and reconstruction to be knowledgeable about the timing of intraoperative events, in order to best plan the anesthetic while maintaining hemodynamic stability.

Room Preparation

Unless the patient has a tracheostomy, we recommend maintaining the operating room table toward the anesthesiology machine during the induction of anesthesia. An underbody warming blanket is placed on the operating room table. Monitor cables are left hanging from an IV pole so they can be placed alongside the operating room table and attached to the patient from the feet up, which will facilitate turning the bed 180°. Two intravenous lines are prepared, at least one being a "blood set." An active type and cross is confirmed with the blood bank and two units of matched pack red blood cells are made immediately available. An arterial line is also prepared. If the patient does not have a tracheostomy, the flexible bronchoscope is prepared with an appropriate selected endotracheal tube (i.e., nasal, oral, or laser safe). The supplies and syringes are also prepared to perform the airway topicalization (Chap. 8) with atomization, superior laryngeal and recurrent laryngeal nerve blocks.

Besides preparing the intravenous anesthetic agents, we also set up intravenous infusion devices, ancillary syringes of dexamethasone, ondansetron and antibiotic prophylaxis, all of which can be given during the induction.

Airway Management and Anesthetic Induction

If the patient has a mature, adequately sized tracheotomy, the operating room bed may be turned 180° away from the anesthesiology machine as emergency airway intervention and proximity to the workstation are unlikely. An appropriately sized wire reinforced anode tube is prepared with adequate lubrication, a means to deliver laryngeal tracheal anesthesia is made available as is a soft tip suction catheter. The tracheostomy site is sprayed with local anesthesia, the stoma suctioned if necessary and the reinforced tube is placed. Once end tidal carbon dioxide is confirmed, general anesthesia is induced with a combination of intravenous and inhaled agents. An appropriately sized, well-lubricated anode tube is placed in the tracheostomy to the depth of the double black lines on the tube or when there is bilateral breath sounds and bilateral chest rise.

There is a high likelihood of a mainstem intubation when placing an endotracheal tube in a tracheostomy site. It is important to confirm endotracheal tube placement with bilateral breath sounds and chest rise since the ability to listen and examine the chest will be greatly reduced once the patient is prepped and draped. Peak airway pressures should be noted at this time since a mainstem intubation can occur at any time during the procedure and increasing airway pressures may be the first sign that this is occurring before a major oxygen desaturation. The tube will likely be sutured to the chest wall by the surgeons, but the ability to place the endotracheal tube deeper or withdraw it during the procedure should always be available.

The full details of performing an awake intubation are presented elsewhere in the text. For patients with painful, friable, and necrotic intraoral lesions, topical anesthesia alone may prove inadequate, presumably because of the tissue acid milieu surrounding the tumor. In this situation the use of superior and recurrent laryngeal nerve blocks may be necessary to reliably anesthetize the airway. It should be noted that these regional techniques are not without risks and should be avoided if there is infection at the site, tumor at the site, coagulopathy, or disrupted anatomy that would make localization of traditional landmarks impossible. If a trans-tracheal block of the recurrent laryngeal nerve is planned, the patient should not be an aspiration risk as the vocal cords will not adduct fully if the block is performed correctly. Figures 16.5 and 16.6 show these techniques.

While preparing a patient for an awake intubation it is critical to develop a good rapport ahead of time. Superb communication with the patient is important and letting the



Fig. 16.5 Performance of the superior laryngeal nerve block



Fig. 16.6 Performance of the recurrent laryngeal nerve block (trans-tracheal block)

patient know each step anticipated is vital to the success of the procedure. Glycopyrrolate (0.2–0.4 mg) is given well before topicalization begins. Although anesthetizing the airway can be done effectively and efficiently, it is important to take time and start the oral or nasal topicalization with brief episodes of local anesthesia atomization allowing the patient to get used to the bitter taste of local anesthetic. Initiating coughing or gagging at this time can create patient anxiety and may cause them to lose confidence in their caregivers. It is important to "test" the airway anesthesia before commencing with the awake intubation. A gentle oral pharyngeal suction with a Yankauer, serves to eliminate excessive secretions while testing the adequacy of the airway anesthesia. If inadequate, further topicalization, the use of nerve blocks (if not previously done), or a judicious dose of an opioid can be considered.

For successful intubation, the room ergonomics are critical. The video screen should be placed directly in front of the operator (Fig. 16.7). Suction or blow-by oxygen (our preference) should be attached to the working channel of the scope. Additional local anesthesia should be attached to the working channel either directly or via a threaded epidural catheter (Figs 16.8 and 16.9) (our preference) to provide anesthesia during the procedure while providing a means to clean off the scope should fog, blood or secretions obscure the view. The anesthetic induction agent should be attached to the intravenous before starting the intubation so there is no delay in induction once intubation has been confirmed.

After successfully performing the endotracheal intubation, confirming tube position visually with bronchoscopy and detecting the presence of end tidal carbon dioxide, anesthesia is induced using a small dose of intravenous propofol (20–50 mg) and the patient is allowed to breathe sevoflurane spontaneously. Due to the fact that the patient's airway is anesthetized, larger doses of propofol may cause hemodynamic instability and are unnecessary. We have found that once the endotracheal tube is positioned in the trachea, the patients are more comfortable if the cuff remains deflated. We only inflate the endotracheal tube cuff after the induction of anesthesia. Inflating the endotracheal tube cuff while the patient is awake initiates patient coughing and anxiety. After intubation and anesthetic induction the patient is turned 180 away from the anesthesia machine. If planned, the surgeon will place a tracheostomy and Foley catheter at this time, while the anesthesiologist places an arterial line and an addition intravenous line. A percutaneous feeding tube may also be placed at this time by gastroenterologists or general surgeons.

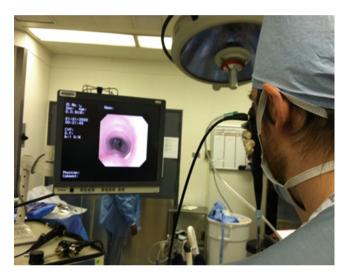


Fig. 16.7 The recommended position of the video screen during a fiberoptic intubation. Note the supplemental oxygen tubing attached to the working channel of the bronchoscope. This allows oxygen "blow-by" during the procedure and helps blow secretions away from the camera lens

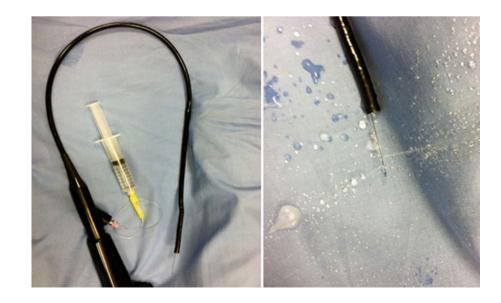


Fig. 16.8 Passage of an epidural catheter through the working channel of the fiberscope

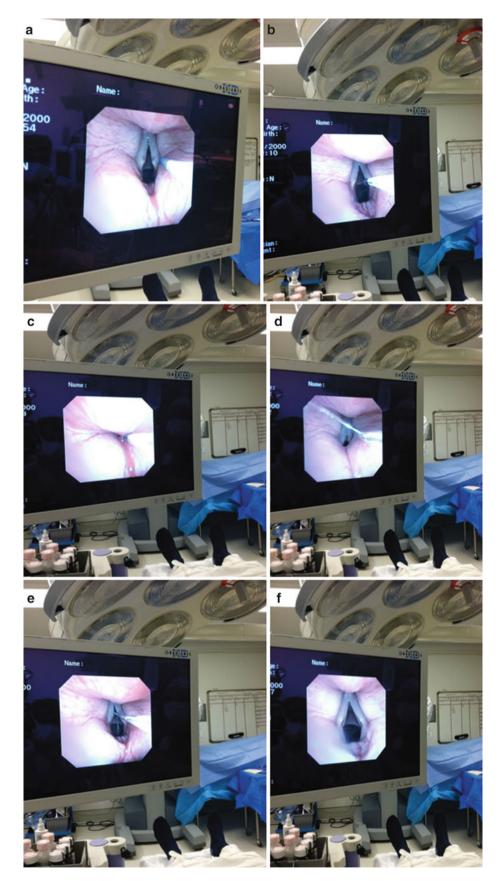


Fig. 16.9 The use of an epidural catheter threaded through a bronchoscope to administer supplemental anesthesia and perform a trans-tracheal block (recurrent laryngeal nerve block) under direct bronchoscopic view. (\mathbf{a} , \mathbf{b}) Note the epidural catheter is easily positioned through the vocal cords. (\mathbf{c}) Despite placing a trans-tracheal block externally, the

patient still coughs in response to the administration of additional local anesthesia. (d-f) Before proceeding with intubation an additional dose of anesthesia reveals adequate topical anesthesia without cough. This additional step assures the patient will tolerate the placement of the bronchoscope and endotracheal tube through their larynx

Intravenous and Monitor Placement

The placement of appropriately sized and positioned intravenous and arterial lines will be dictated by the surgical plan. Generally the limb(s) used for tissue harvest will be well demarcated. In our institution the limb(s) are wrapped in a curlex bandage; however, it is critical that the anesthesiologists and otolaryngologists communicate and are aware of the surgical plan and the intended harvest site(s).

The Resection

The resection represents a very stimulating portion of the surgical case where significant blood loss can be anticipated. Although the avoidance of neuromuscular blocking (NMB) agents is generally a good plan during the resection, depending on the surgical site, the use of neuromuscular blockade maybe required (intraoral) or contraindicated (extraoral). In many head and neck resection cases both intraoral and extraoral procedures are planned sequentially (e.g., transoral robotic resections) and the timing and use of NMB must be precise and agreed upon ahead of time. In most situations NMB will be employed during intraoral evaluation (e.g., laryngoscopy) or resection. During resections around major nerves including parotid tumors and neck dissections the use of NMBs is contraindicated since intraoperative nerve monitoring (IONM) will likely be used. For younger, healthier patients, we generally use a large dose of remifentanil (0.2-5 µg/kg/min) and propofol (25-100 µg/kg/min) to facilitate exposure during intraoral portions of the procedure. This eliminates the need to monitor or confirm the reversal of NMB during portions of the surgery when IONM is used.

For older patients with significant comorbidities who would otherwise not tolerate a high dose of anesthesia, a judicious dose of NMB agents is used and either allowed to auto-reverse or is actively reversed pharmacologically. Either way, NMB is monitored and its reversal is confirmed with the surgical team and documented in the anesthesia record.

Because NMB agents are avoided during the majority of these procedures, the surgical team needs to communicate with the anesthesiology team before initiating surgical stimulation or commencing with a very stimulating portion of the surgery or risk patient movement. The maintenance of immobility requires a deep level of anesthesia. Patient movement does not imply the patient is awake. If the patient begins to move during surgery, the best approach for the otolaryngologist is to inform the anesthesiologists without proclaiming the patient is "awake." Doing so is generally received as unprofessional since it implies the anesthesiologist is committing malpractice or at the very least, doing a poor job. Many of the head and neck patients cannot tolerate deep levels of anesthesia and maintain hemodynamic stability. In this case, vasoactive agents will be used to assure adequate cardiac output and tissue oxygenation and allow for a deep plane of anesthesia.

Tissue Harvest

This portion of the surgery is very stimulating and will be facilitated by the use of NMB, but will generally not be associated with significant blood loss due to the use of a tourniquet (obviously not the case with a scapular harvest).

Microvascular Anastomosis

The microvascular anastomosis is not a stimulating portion of the procedure and if the anesthetic level is not titrated down a decreased blood pressure can be anticipated. The use of NMB is recommended, since sudden movement during the microvascular anastomosis could be catastrophic. When the otolaryngologist is working under a microscope, it is important for the anesthesiologists to appreciate that minor motion of the operating room bed creates distracting, magnified, and significant motion in the surgical field being viewed. Care should be taken to avoid making any contact with the bed and if it is necessary to touch the bed for whatever reason, communication with the surgical team ahead of time is critical.

Emergence and Monitor Transfer

Spontaneous ventilation is initiated in patients with tracheostomies. Patients nasally intubated are left intubated and can be mechanically ventilated, or left spontaneously breathing. The anesthetic is maintained and the patients are transferred to a monitored setting, generally an intensive care unit, via a monitored transport bed. Because of significant airway edema, patients are rarely extubated at the end of major head and neck resection and reconstruction surgery.

Postoperative Considerations

Otolaryngologist's and Anesthesiologist's Perspectives

The majority of postoperative management strategies including pain control and monitoring are relatively routine in patients undergoing head and neck reconstruction. Antiemetic prophylaxis is extremely important in this population to avoid complications related to pharyngeal and oral cavity reconstructions that can occur from retching. This is especially true of tracheal reconstructions in which case postoperative vomiting can be catastrophic. Postoperative corticosteroids are often indicated to minimize postoperative edema with 24-h administration sufficient for the majority of cases. Cases involving airway reconstruction are a notable exception and may require longer administration. Postoperative shivering must be avoided, as it increases the oxygen consumption, causes peripheral vasoconstriction, and reduces the blood flow in transplanted tissues.

Regarding free tissue transfer there was a historical belief that patients undergoing these complicated procedures benefited from sedation and ICU observation for 48-72 h to prevent movement and possible microvascular anastomotic compromise. This has been recognized to be false and the goal should be early extubation with spontaneous ventilation with the majority of patients not requiring sedation or prolonged stays in the intensive care monitoring postoperatively. Unnecessary sedation in the intensive care unit increased the risk of hospital-acquired infections, complications such as DVT and pneumonia, deconditioning, increased use of anxiolytics/restraints, and prolonged hospital stay ^[53]. Additionally, unnecessary sedation can decrease perfusion pressure to transplanted tissues and lead to flap compromise. Investigations have shown that perfusion of transplanted tissue improves over the course of 72 h after transplantation [54, 55], therefore maintaining blood pressure and perfusion pressure in the first several days is important for flap viability and prevention of stasis and microvascular thrombosis.

Postoperative Complications

Occasionally patients status post free-flap reconstruction will return to the operating room emergently because of free flap failure secondary to bleeding, infection, or compromised vascular integrity. In such cases the anesthesiology and otolaryngology team need to work quickly and precisely to best care for the patient to assure the best surgical outcome. Occasionally patients will not have a tracheostomy, in this situation an awake intubation must be planned.

Bleeding patients will need adequate intravenous access, an active type and cross and packed red blood cells prepared and delivered to the operating room. Infected patients may be septic and showing signs of shock and will need adequate access, invasive monitoring, broad antibiotic coverage, and vasopressor support.

When free flap viability is in question from a vascular cause it can be from the arterial supply or the venous drainage. Many times a simple thrombectomy or removal of a vascular kink can correct the problem; however, the anesthesiologists should prepare for a prolonged procedure should a vascular reanastomosis be necessary or a completely new free flap revision requiring a new tissue harvest be indicated. In such a case adequate intravenous access, invasive monitoring, underbody hot air warming devices, and blood products need to be available.

Conclusions

Head and neck free flap transfer is a challenging surgical procedure for both the surgeon and anesthesiologist. As with other head and neck procedures, most of the difficulties can be addressed by thorough patient evaluation, adequate preparation and of course, excellent communication between teams.

Clinical Insights

For the Otolaryngologist (from the Anesthesiologist)

- Preoperative optimization of the cardiovascular, hematologic, and nutritional status will likely improve surgical outcome and decrease postoperative complications.
- Patients can be safely and efficiently intubated awake, don't underestimate the impact of prior head and neck surgery, radiation therapy, and the current airway pathology on the ability to intubate and ventilate the anesthetized patient.
- When the use of neuromuscular blocking agents is contraindicated, patients require a significant level of anesthesia to assure immobility. This may cause significant cardiovascular instability requiring vaso-pressors agents to maintain homeostasis.
- Patient movement does not imply the patient is awake. If the patient begins to move during a surgical case, the best approach for the otolaryngologist is to inform the anesthesiologists.
- Good communication with the anesthesiology team and demarcation of the site of tissue harvest is critical to avoid wasted time and possibly the placement of compromising intravenous and arterial access.

For the Anesthesiologist (from the Otolaryngologist)

• Medical comorbidity is the rule rather than the exception in patients undergoing major head and neck reconstruction.

- Anesthesia techniques for head and neck reconstruction cannot follow standard protocol as in other surgical specialties due to the unique elements of the operation involving the airway, upper aerodigestive tract, and various donor sites required for reconstruction.
- Communication with the surgical team will allow for appropriate airway management, positioning, monitoring, and warming. All intravenous and arterial lines should be reviewed with surgical team prior to placement to avoid donor site compromise of vascular pedicles.
- Large volume resuscitations are generally unnecessary and should be avoided and lead to increased edema and complications. There is no current evidence to support the routine use of volume expanders in major head and neck surgery.
- Moderate hemodilution with transfusion to maintain hematocrit 25–30 is generally preferred.
- Intermittent intraoperative vasopressor use is acceptable. The timing of administration should not coincide with microvascular anastomosis if possible. Vasopressor infusions should be avoided if possible.

Clinical Pearls

For the Otolaryngologist (from the Otolaryngologist)

- The majority of reconstructive complications are related to medical comorbidity and less related to technical aspects of the reconstruction.
- Clear communication with the anesthesiology team is paramount to avoid airway complications and donor site compromise.
- Intermittent intraoperative vasopressor use is acceptable and does not appear to affect outcomes in free tissue transfer procedures.
- Appropriate surgical site planning will allow for vascular access and warming device placement.
- Two-team approaches should be utilized when possible to limit operative time (<10 h) and minimize blood loss and associated resuscitation.
- Transfusion should be anticipatory rather than reactionary with a goal hematocrit range of 25–30.

For the Anesthesiologist (from the Anesthesiologist)

- Patients with a fixed larynx from radiation therapy are known to be difficult to ventilate and intubate and have a high glidescope failure rate.
- During an awake intubation, test the airway anesthesia with gentle suction with a yankauer. Don't proceed if the anesthesia is inadequate. Position the video screen directly in front of the videoscope operator, do not over sedate the patient, patient cooperation may be critical, don't inflate the endotracheal tube cuff until after the patient is asleep.
- After performing an awake intubation, give a small dose of induction agent to initiate the general anesthetic. Large doses will cause hemodynamic instability.
- Airway compromise can still occur with a tracheostomy in situ.
- The use of vasopressors is not associated with free flap failure.
- Overhydration is associated with postoperative complications, increased hospital stays and free flap failure.
- The use of neuromuscular blocking agents will likely be indicated during certain portions of a head and neck resection reconstruction surgery and contraindicated during others. If you have any doubt, do not use them and communicate with the otolaryngologists if you have or plan to use them.
- Under body hot air warming blankets may be the only effective means to rewarm and maintain normothermia during head and neck resection and reconstruction surgeries.
- While working under a microscope any minor motion of the operating room bed creates distracting, magnified and significant motion in the surgical field being viewed. Care should be taken to avoid making any contact with the bed and if it is necessary to touch the bed for whatever reason, communicate with the surgical team ahead of time.

References

- Suh JD, Sercarz JA, Abemayor E, et al. Analysis of outcome and complications in 400 cases of microvascular head and neck reconstruction. Arch Otolaryngol Head Neck Surg. 2004;130(8):962–6.
- Hagau N, Longrois D. Anesthesia for free vascularized tissue transfer. Microsurgery. 2009;29(2):161–7.
- Haughey BH, Wilson E, Kluwe L, et al. Free flap reconstruction of the head and neck: analysis of 241 cases. Otolaryngol Head Neck Surg. 2001;125(1):10–7.

- Serletti JM, Higgins JP, Moran S, Orlando GS. Factors affecting outcome in free-tissue transfer in the elderly. Plast Reconstr Surg. 2000;106(1):66–70.
- Rosenberg AJ, Van Cann EM, van der Bilt A, Koole R, van Es RJ. A prospective study on prognostic factors for free-flap reconstructions of head and neck defects. Int J Oral Maxillofac Surg. 2009;38(6):666–70.
- Singh B, Cordeiro PG, Santamaria E, Shaha AR, Pfister DG, Shah JP. Factors associated with complications in microvascular reconstruction of head and neck defects. Plast Reconstr Surg. 1999;103(2):403–11.
- Chang LD, Buncke G, Slezak S, Buncke HJ. Cigarette smoking, plastic surgery, and microsurgery. J Reconstr Microsurg. 1996;12(7):467–74.
- Pattani KM, Byrne P, Boahene K, Richmon J. What makes a good flap go bad? A critical analysis of the literature of intraoperative factors related to free flap failure. Laryngoscope. 2010;120(4): 717–23.
- Baumann DP, Yu P, Hanasono MM, Skoracki RJ. Free flap reconstruction of osteoradionecrosis of the mandible: a 10-year review and defect classification. Head Neck. 2011;33(6):800–7.
- Cannady SB, Dean N, Kroeker A, Albert TA, Rosenthal EL, Wax MK. Free flap reconstruction for osteoradionecrosis of the jaws– outcomes and predictive factors for success. Head Neck. 2011;33(3):424–8.
- Clark JR, McCluskey SA, Hall F, et al. Predictors of morbidity following free flap reconstruction for cancer of the head and neck. Head Neck. 2007;29(12):1090–101.
- Pohlenz P, Blessmann M, Blake F, Li L, Schmelzle R, Heiland M. Outcome and complications of 540 microvascular free flaps: the Hamburg experience. Clin Oral Investig. 2007;11(1):89–92.
- Banic A, Krejci V, Erni D, Wheatley AM, Sigurdsson GH. Effects of sodium nitroprusside and phenylephrine on blood flow in free musculocutaneous flaps during general anesthesia. Anesthesiology. 1999;90(1):147–55.
- Platek ME, Reid ME, Wilding GE, et al. Pretreatment nutritional status and locoregional failure of patients with head and neck cancer undergoing definitive concurrent chemoradiation therapy. Head Neck. 2011;33(11):1561–8.
- Gourin CG, Forastiere AA, Sanguineti G, Marur S, Koch WM, Bristow RE. Impact of surgeon and hospital volume on short-term outcomes and cost of oropharyngeal cancer surgical care. Laryngoscope. 2011;121(4):746–52.
- Khouri RK, Cooley BC, Kunselman AR, et al. A prospective study of microvascular free-flap surgery and outcome. Plast Reconstr Surg. 1998;102(3):711–21.
- Shestak KC, Jones NF. Microsurgical free-tissue transfer in the elderly patient. Plast Reconstr Surg. 1991;88(2):259–63.
- Stavrianos SD, McLean NR, Fellows S, et al. Microvascular histopathology in head and neck oncology. Br J Plast Surg. 2003; 56(2):140–4.
- Howard MA, Cordeiro PG, Disa J, et al. Free tissue transfer in the elderly: incidence of perioperative complications following microsurgical reconstruction of 197 septuagenarians and octogenarians. Plast Reconstr Surg. 2005;116(6):1659–68 [discussion 1669–71].
- Patel RS, McCluskey SA, Goldstein DP, et al. Clinicopathologic and therapeutic risk factors for perioperative complications and prolonged hospital stay in free flap reconstruction of the head and neck. Head Neck. 2010;32(10):1345–53.
- Ferrier MB, Spuesens EB, Le Cessie S, Baatenburg de Jong RJ. Comorbidity as a major risk factor for mortality and complications in head and neck surgery. Arch Otolaryngol Head Neck Surg. 2005;131(1):27–32.
- Farwell DG, Reilly DF, Weymuller Jr EA, Greenberg DL, Staiger TO, Futran NA. Predictors of perioperative complications in head and neck patients. Arch Otolaryngol Head Neck Surg. 2002;128(5):505–11.

- Dequanter D, Lothaire P. Serum albumin concentration and surgical site identify surgical risk for major post-operative complications in advanced head and neck patients. B-Ent. 2011;7(3): 181–3.
- Story DA. Postoperative mortality and complications. Best Pract Res Clin Anaesthesiol. 2011;25(3):319–27.
- Skitarelic N, Morovic M, Manestar D. Antibiotic prophylaxis in clean-contaminated head and neck oncological surgery. J Craniomaxillofac Surg. 2007;35(1):15–20.
- Phan M, Van der Auwera P, Andry G, et al. Antimicrobial prophylaxis for major head and neck surgery in cancer patients: sulbactam-ampicillin versus clindamycin-amikacin. Antimicrob Agents Chemother. 1992;36(9):2014–9.
- Reiffel AJ, Kamdar MR, Kadouch DJ, Rohde CH, Spector JA. Perioperative antibiotics in the setting of microvascular free tissue transfer: current practices. J Reconstr Microsurg. 2010;26(6):401–7.
- Simo R, French G. The use of prophylactic antibiotics in head and neck oncological surgery. Curr Opin Otolaryngol Head Neck Surg. 2006;14(2):55–61.
- Schramm S, Wettstein R, Wessendorf R, Jakob SM, Banic A, Erni D. Acute normovolemic hemodilution improves oxygenation in ischemic flap tissue. Anesthesiology. 2002;96(6):1478–84.
- Velanovich V, Smith Jr DJ, Robson MC, Heggers JP. The effect of hemoglobin and hematocrit levels on free flap survival. Am Surg. 1988;54(11):659–63.
- Rossmiller SR, Cannady SB, Ghanem TA, Wax MK. Transfusion criteria in free flap surgery. Otolaryngol Head Neck Surg. 2010;142(3):359–64.
- 32. Shah MD, Goldstein DP, McCluskey SA, et al. Blood transfusion prediction in patients undergoing major head and neck surgery with free-flap reconstruction. Arch Otolaryngol Head Neck Surg. 2010;136(12):1199–204.
- Szakmany T, Dodd M, Dempsey GA, et al. The influence of allogenic blood transfusion in patients having free-flap primary surgery for oral and oropharyngeal squamous cell carcinoma. Br J Cancer. 2006;94(5):647–53.
- 34. Fenner M, Vairaktaris E, Nkenke E, Weisbach V, Neukam FW, Radespiel-Troger M. Prognostic impact of blood transfusion in patients undergoing primary surgery and free-flap reconstruction for oral squamous cell carcinoma. Cancer. 2009;115(7): 1481–8.
- Chen HC, Coskunfirat OK, Ozkan O, et al. Guidelines for the optimization of microsurgery in atherosclerotic patients. Microsurgery. 2006;26(5):356–62.
- Chappell D, Jacob M, Hofmann-Kiefer K, Conzen P, Rehm M. A rational approach to perioperative fluid management. Anesthesiology. 2008;109(4):723–40.
- Sigurdsson GH. Perioperative fluid management in microvascular surgery. J Reconstr Microsurg. 1995;11(1):57–65.
- 38. Chalmers A, Turner MW, Anand R, Puxeddu R, Brennan PA. Cardiac output monitoring to guide fluid replacement in head and neck microvascular free flap surgery—what is current practice in the UK? Br J Oral Maxillofac Surg. Sep 20, 2011. Epub ahead of print
- Scholz T, Evans GR. Impact of hypertonic and hyperoncotic saline solutions on ischemia-reperfusion injury in free flaps. Plast Reconstr Surg. 2008;122(1):85–94.
- 40. Boldt J. New light on intravascular volume replacement regimens: what did we learn from the past three years? Anesth Analg. 2003;97(6):1595–604.
- 41. Arellano R, Gan BS, Salpeter MJ, et al. A triple-blinded randomized trial comparing the hemostatic effects of large-dose 10% hydroxyethyl starch 264/0.45 versus 5% albumin during major reconstructive surgery. Anesth Analg. 2005;100(6):1846–53.
- Hiltunen P, Palve J, Setala L, et al. The effects of hypotension and norepinephrine on microvascular flap perfusion. J Reconstr Microsurg. 2011;27(7):419–26.

- 43. Monroe MM, Cannady SB, Ghanem TA, Swide CE, Wax MK. Safety of vasopressor use in head and neck microvascular reconstruction: a prospective observational study. Otolaryngol Head Neck Surg. 2011;144(6):877–82.
- Chen C, Nguyen MD, Bar-Meir E, et al. Effects of vasopressor administration on the outcomes of microsurgical breast reconstruction. Ann Plast Surg. 2010;65(1):28–31.
- 45. Suominen S, Svartling N, Silvasti M, Niemi T, Kuokkanen H, Asko-Seljavaara S. The effect of intravenous dopamine and dobutamine on blood circulation during a microvascular TRAM flap operation. Ann Plast Surg. 2004;53(5):425–31.
- Spiegel JH, Polat JK. Microvascular flap reconstruction by otolaryngologists: prevalence, postoperative care, and monitoring techniques. Laryngoscope. 2007;117(3):485–90.
- Hanasono MM, Butler CE. Prevention and treatment of thrombosis in microvascular surgery. J Reconstr Microsurg. 2008;24(5): 305–14.
- 48. Disa JJ, Polvora VP, Pusic AL, Singh B, Cordeiro PG. Dextranrelated complications in head and neck microsurgery: do the benefits outweigh the risks? A prospective randomized analysis. Plast Reconstr Surg. 2003;112(6):1534–9.

- Hardin CK, Kirk WC, Pederson WC. Osmotic complications of low-molecular-weight dextran therapy in free flap surgery. Microsurgery. 1992;13(1):36–8.
- Sun TB, Chien SH, Lee JT, Cheng LF, Hsu LP, Chen PR. Is dextran infusion as an antithrombotic agent necessary in microvascular reconstruction of the upper aerodigestive tract? J Reconstr Microsurg. 2003;19(7):463–6.
- 51. Jallali N. Dextrans in microsurgery: a review. Microsurgery. 2003;23(1):78-80.
- 52. Ridha H, Jallali N, Butler PE. The use of dextran post free tissue transfer. J Plast Reconstr Aesthet Surg. 2006;59(9):951–4.
- Allak A, Nguyen TN, Shonka Jr DC, Reibel JF, Levine PA, Jameson MJ. Immediate postoperative extubation in patients undergoing free tissue transfer. Laryngoscope. 2011;121(4):763–8.
- Schultze-Mosgau S, Wiltfang J, Birklein F, Neukam FW. Micro-lightguide spectrophotometry as an intraoral monitoring method in free vascular soft tissue flaps. J Oral Maxillofac Surg. 2003;61(3):292–7.
- Hanasono MM, Ogunleye O, Yang JS, Hartley CJ, Miller MJ. Changes in blood velocity following microvascular free tissue transfer. J Reconstr Microsurg. 2009;25(7):417–24.

Neurosurgical Otolaryngology I: Neurotology/ Posterior Fossa Surgery

Jess W. Brallier and Eric E. Smouha

Introduction

In neurotologic surgery, the temporal bone is the gateway to the posterior cranial fossa and skull base. Neurotologists collaborate with neurosurgeons to treat a variety of neoplastic, inflammatory, and traumatic disorders of this anatomically complex area, of which acoustic neuroma is the most common. Posterior fossa surgery presents significant perioperative challenges to both the surgeon and anesthesiologist. In addition to containing the medulla, pons and cerebellum, this compartment houses cranial nerve nuclei and centers crucial to respiratory and cardiovascular function. The pathology as well as surgical manipulation can lead to potential hemodynamic instability, compression of vital areas, and damage to important neural pathways. These factors combined with the limited size of the posterior fossa compartment can make this surgery tenuous.

During neurotologic surgery, the surgeon may become focused on preservation of function, such as facial nerve and (when possible) hearing. But the first, often unstated, goal of surgery is to ensure the patient avoids major neurologic morbidity or mortality^[1]. Regardless of the approach used, surgery of the posterior cranial fossa exposes the brainstem and its circulation to the risk of surgical injury, and as such, can be life threatening. For this reason, close interaction between the surgeon and anesthesiologist is essential to achieving satisfactory outcome. Patient positioning, hemodynamic lability, intraoperative monitoring, and the potential for complications such as venous air embolism and pneumocephalus necessitate communication and collaboration between the surgical and anesthetic teams.

E.E. Smouha

Overview

Depending on the nature of the lesion, patients presenting for skull base surgeries may have a decreased level of consciousness, elevated intracranial pressure (ICP) secondary to obstructive hydrocephalus, or cranial nerve palsies. Such individuals may require mechanical ventilation preoperatively and postoperatively. Maintaining continual, flexible communication with surgeons and staff throughout the perioperative period is paramount when administering anesthesia for posterior fossa surgery. Likewise, appreciation of the often urgent surgical requirements of these surgeries is important to anesthesiology staff.

Acoustic Neuroma

Acoustic neuroma (vestibular schwannoma) is the most common condition encountered in neurotologic surgery (Fig. 17.1). Acoustic neuroma is a benign neoplasm of the VIIIth cranial nerve that occurs sporadically with an incidence of about 1:100,000. It most often presents with progressive unilateral sensorineural hearing loss, although this is variable; sudden hearing loss and normal hearing can also be associated with this tumor. Tinnitus and vague disequilibrium are common. True vertigo (spinning) is not common but can occur. Facial twitching or involuntary blepharospasm can sometimes occur, but facial paralysis is distinctly rare and its presence should raise the suspicion of facial nerve schwannoma or malignancy. Although benign, larger acoustic neuromas can cause significant neurologic sequelae including facial hypesthesia and loss of corneal reflexes; gait ataxia; cerebellar signs; hydrocephalus; mental confusion, obtundation, and nystagmus. Brun's nystagmus is described as a bidirectional nystagmus caused by cerebellar compression with coarse and slow eye movement when looking toward the side of the tumor, and fine and rapid eye movement when looking away.

These tumors occupy the internal auditory canal (IAC), and cerebellopontine angle (CPA), and are easily demonstrated by

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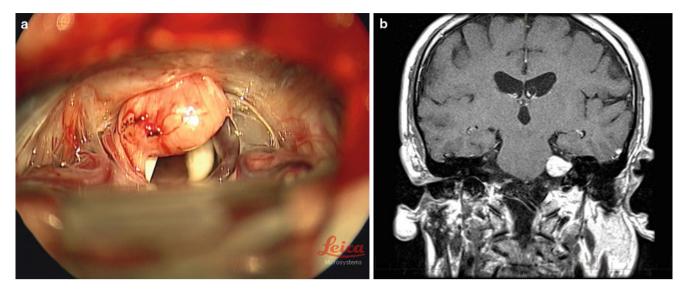


Fig. 17.1 (a) Acoustic neuroma (vestibular schwannoma) seen under the operating microscope. (b) Frontal MRI view of the same neuroma

gadolinium-enhanced magnetic resonance imaging (MRI). They can be intracanalicular, small (<1.5 cm), medium (1.5– 2.5 cm), or large (>2.5 cm in greatest dimension) at the time of diagnosis. Treatment options include serial observation, surgical excision, or stereotactic radiotherapy. The selection of treatment is based on tumor size, hearing status, age and health of the indiviual-, and patient preference. Because these tumors usually grow slowly, 2 mm/year on average, serial observation is acceptable in older patients with smaller tumors, especially when hearing is preserved. MRI scans are repeated at 6 months then at yearly intervals until interval tumor growth of 2 mm or greater occurs. Multiple studies have shown that about 50% of patients managed with serial observation will not require surgery ^[2]. Patients must understand that the success of this strategy is contingent on regular follow-up and that hearing may be permanently lost during the period of observation.

Stereotactic radiotherapy (also called "radiosurgery," a misleading term) is a treatment modality that administers a sharply contoured beam of ionizing radiation to the tumor. Treatment planning is done with the head in a rigid stereotactic frame, and the radiation is administered either by a precisely tuned array of cobalt sources ("Gamma knife") or a moving electron beam (such as "Cyber knife," or "LINAC," a linear accelerator) controlled by a computer. Stereotactic radiotherapy is successful in arresting the further growth of the tumor in about 90% of cases, but does not eliminate the tumor ^[3]. After treatment, the tumor must be followed by serial MRI. Tumor shrinkage and cystic degeneration sometimes occurs. Transient increase in tumor size also commonly occurs during the first year after treatment, but persistent regrowth generally mandates surgery. Hearing preservation is occasionally successful, especially when smaller treatment dosages are used [<13 Gray (Gy)] to the tumor margin) and the radiation field excludes the inner ear. Stereotactic radiotherapy is most clearly appropriate for older patients with smaller tumors ^[4], but is often selected by younger patients because it can obviate the need for surgery. Radiation treatment may not be effective for tumors greater than 3 cm in size. Other tumors of the cerebellopontine angle include meningiomas, facial nerve schwannomas, epidermoid tumors, lipomas, and aneurysms. These are all benign soft tissue lesions that are extraaxial to the brain. They present with similar symptoms and signs as acoustic neuromas and are removed through similar surgical approaches.

Glomus Tumors

Glomus tumors are paragangliomas ("chemodectomas"), benign growths of neuroendocrine origin ^[5], which arise along the course of certain nerves in the head and neck as well as elsewhere in the body (Fig. 17.2).

In and around the temporal bone, glomus tympanicum tumors are most common, beginning in the middle ear along the course of Jacobson's nerve, and usually appearing as a red mass behind the tympanic membrane ^[6]. They first appear in the hypotympanum, the inferior portion of the middle ear, and they can usually be excised via a transmastoid or trans-middle ear approach. Glomus jugulare tumors begin in the jugular foramen and are generally more formidable than glomus tympanic tumors because they involve the great vessels and nerves and the bone of the skull base. Glomus vagale tumors arise from the Xth cranial nerve in the upper neck. These are parapharyngeal space masses that can grow upward to involve the skull base at the jugular foramen, and occasionally require combined approaches to the skull base and neck for removal.

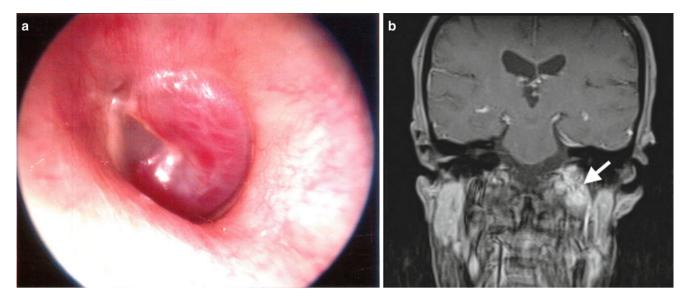


Fig. 17.2 (a) Glomus tumor seen under the operating microscope. (b) MRI imaging of a glomus tumor

Carotid body tumors arise from the neuroendocrine cells at the bifurcation of the internal and external carotid arteries, and rarely involve the bone of the skull base. Paragangliomas can also occur in the para-aortic organs of Zuckerkandel in the abdomen and thorax and may be part of the multiple endocrine neoplasia (MEN) type II syndrome ^[7].

Preoperative Considerations

Otolaryngologist's Perspective

The diagnostic workup of acoustic neuroma usually begins with an audiogram; if a unilateral sensorineural hearing loss is found, especially with a decreased speech discrimination score, then further workup is initiated (Fig. 17.3).

Auditory brainstem response (ABR, synonymous with brainstem auditory evoked potential, BAEP or BAER) testing uses repetitive click and tone stimuli to elicit time-locked, summated electrical responses from the auditory nerve. These will usually show abnormal delay of the waveforms elicited from the tumor ear as compared to the normal ear, but the test is not infallible. MRI with gadolinium enhancement will always demonstrate the acoustic neuroma, and this test is the gold-standard for diagnosis.

Preoperative evaluation of glomus jugulare tumors should include CT and MRI of the head and neck, chest X-ray, as well as urinary levels of the catecholamine metabolites, vanilmandelic acid (VMA) and metanephrine ^[8]. Early stage glomus jugulare tumors are confined to the temporal bone and jugular bulb but larger tumors may track within the jugular lumen into the upper neck, intracranially through the jugular foramen into the posterior cranial fossa, and anteriorly toward the petrous apex and infratemporal fossa. Because these tumors have rich vascularity, preoperative angiography and embolization are usually performed.

Anesthesiologist's Perspective

A thorough preoperative evaluation is essential to the delivery of any safe anesthetic. The preoperative assessment provides the anesthesiologist with the opportunity to gain a complete understanding of an individual's neurological status and medical comorbidities and to recommend further workup when necessary. "Clearing" a patient for surgery is not the objective of a preoperative evaluation. This terminology is misleading and implies the absence of risk should the "go ahead" be given. Instead, practitioners should realize that risk always exists and that the primary purpose of a detailed preoperative assessment is to identify modifiable risk factors and to provide medical optimization when necessary ^[9].

In the case of elective surgery, the preoperative assessment should occur in a preoperative evaluation clinic prior to the day of surgery. This minimizes redundant medical provider interviews and allows for targeted, economical diagnostic testing (3). Assessing patients in advance also assists in uncovering comorbidities not easily identified during the cursory interview performed on the day of surgery ^[10]. Evaluating individuals prior to surgery also improves patient safety and satisfaction ^[11–13] while decreasing operating room delays and reducing lengths of hospital stays ^[14, 15].

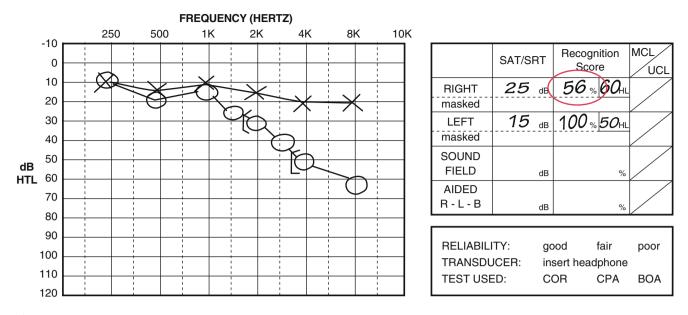


Fig. 17.3 Typical audiogram report demonstrating a right (circle) normal sloping to severe sensorineural hearing loss. There is normal hearing in the left (X) ear

Not all clinical scenarios lend themselves to a pre-admission assessment. Formulating an appropriate anesthetic plan for an emergent procedure may involve a cursory history and physical with minimal opportunity for medical optimization.

Central to the preoperative assessment is a thorough but focused history and physical exam. Emphasis should be placed on evaluating the neurologic status, airway, cardiovascular system, pulmonary system, endocrine system and fluid/ electrolyte balance. Recent laboratory work and diagnostic examinations should be reviewed at this time with additional diagnostics ordered when necessary. Records of prior anesthetics are also valuable.

Neurologic System

Patients with posterior fossa pathology vary widely in their preoperative neurologic presentation. Some are alert and coherent, while others have depressed levels of consciousness and profound neurological deterioration. Such patients may have cranial nerve palsies or elevations in ICP due to obstructive hydrocephalus or brainstem compression.

A careful history begins with questions regarding the type and location of the lesion and symptomatology. Querying patients regarding seizure history (though less likely with infratentorial surgery than supratentorial surgery) and symptoms of elevated ICP including nausea, vomiting, headaches, and neurological deterioration are important. Documentation of preoperative neurological deficits is essential for intraoperative management and postoperative comparison. When the preoperative assessment has been completed in advance, a careful review of the neurologic system should be

Table 17.1 Glasgow coma scale

Chasgow conta scale			
Eye response (E)	Open spontaneously	4	
	Open to verbal command	3	
	Open in response to pain	2	
	No response	1	
Verbal response (V)	Talking/oriented	5	
	Confused speech/disoriented	4	
	Inappropriate words	3	
	Incomprehensible sounds	2	
	No response	1	
Motor response (M)	Obeys commands	6	
	Localizes to pain	5	
	Flexion/withdrawal	4	
	Abnormal flexion	3	
	Extension	2	
	No response	1	
	Total	3-15	

performed on the day of surgery to screen for new or worsening symptoms.

The neurological exam should start by assessing the patient's mental status and level of consciousness. The Glasgow coma scale is a valuable tool in evaluating a patient's mental status. It provides a standard means of assessing the neurological status and also assists the anesthesiologist in determining the need for preoperative intubation and the urgency of neurosurgical intervention ^[16] (Table 17.1).

In the conscious patient, performing a mental status exam is invaluable to uncovering even subtle deficits in a patient's neurological state. Evaluation of cranial nerve function is also crucial as these nuclei may be affected by the lesion or become compromised during surgery. Examination of sensory and motor function with documentation of any loss of sensation or weakness is also important prior to surgery. Review of diagnostic imaging including MRIs or CT scans are also indicated at this time.

The Airway

Careful attention to airway management is also imperative. In addition to asking about intubation history and reviewing prior anesthetic records, questions pertaining to the presence or absence of elevated ICP are particularly relevant in patients with posterior fossa pathology.

Patients with depressed neurological status or who have pathological involvement of the primary respiratory centers may already be intubated. In the case of elective surgery, a thorough airway exam should be completed. Despite the inability of any one maneuver to consistently predict difficult laryngoscopy, assessment of Mallampati score, thymomental distance, sternomental distance and inter-incisor distance are recommended components of a complete airway examination. Two recent studies emphasize the inability to bite one's upper lip (known as the upper lip bite test or ULBT) as a more reliable predictor of difficult laryngoscopy. Khan et al. showed that the ULBT is significantly more specific and accurate in predicting difficult laryngoscopy compared to other airway assessment tests (Mallampati score, thymomental distance, sternomental distance, inter-incisor distance) and that the ULBT in conjunction with these other tests more reliably predicts uncomplicated laryngoscopy or intubation [17, 18].

Additionally, a patient's mouth opening and range of neck mobility should also be assessed. Finally, factors which might lead to difficult mask ventilation such as obesity, large facies, craniofacial abnormalities, or obstructive sleep apnea should be addressed.

Compared to other individuals, patients with elevated ICP have a low tolerance for apnea (apnea leads to increases in arterial CO_2 pressure (PaCO₂) which in turn increases cerebral blood flow and increases in ICP) highlighting the need for an efficient airway securement. Additional challenges to airway management include dysphagia, impaired gag reflex, and laryngeal nerve dysfunction secondary to cranial nerve deficits. In anticipation of a difficult airway, an awake fiberoptic intubation or use of other advanced airway equipment is indicated. The presence of additional anesthesia personnel is also appropriate when managing a potentially difficult airway.

Cardiovascular System

In addition to the cardiovascular disease present in the general population, patients with posterior fossa pathology have additional considerations. A thorough preoperative evaluation of the cardiovascular system is essential.

During the preoperative interview, patients should be asked about comorbid cardiac disease such as hypertension, cardiac arrhythmias, valvular disease, and ischemia. Ouestions about exercise tolerance (i.e., "can you walk up a flight of stairs without experiencing chest discomfort or shortness of breath) can yield valuable insight into one's functional capacity. The 2007 ACC/AHA guidelines recommend routine preoperative ECG testing for patients with known coronary heart disease (CHD) presenting for intermediate-risk surgery. Posterior fossa surgery definitely falls into this category. Additionally, the guidelines state that a routine electrocardiogram (ECG) may be indicated in patients with one clinical risk factor presenting for intermediate-risk surgery ^[19]. A baseline ECG is valuable not only for preoperative screening but also for postoperative comparison should an untoward cardiac even occur. Patients taking beta-blockers and those with evidence of ischemia on preoperative cardiac testing should continue taking them throughout the perioperative period. The preoperative history and physical should guide the anesthesiologist as to whether additional diagnostic testing or consultation with a cardiologist is necessary. A complete review of the perioperative cardiovascular evaluation is beyond the scope of this chapter but the 2007 ACC/ AHA guidelines provide an excellent summary of established practice recommendations for patients presenting for noncardiac surgery. The preoperative history and physical should guide the anesthesiologist as to whether additional diagnostic testing or consultation with a cardiologist is necessary.

Patients with posterior fossa tumors may demonstrate baseline hypertension and hemodynamic instability for a number of reasons (pheochromocytoma). A subgroup of these patients exhibit hypertension due to direct mechanical compression on the pressor zone in the rostral ventrolateral medulla. The hypertension usually resolves with successful surgery ^[20]. In addition to direct compression of central medullary centers, other factors may also contribute to hypertension and hemodynamic lability. Elevated ICP and crowding within the posterior fossa cause further disturbance to the vasomotor center. Regardless of hypertension etiology, knowledge of a patient's preoperative blood pressure range is useful for intraoperative and postoperative management. It is generally more accurate to obtain these blood pressure values prior to the day of surgery. Patients may be anxious so values obtained may not accurately reflect his or her normal

cardiac complications. According to ACC 2007 guidelines, there is no evidence that delaying surgery is beneficial for mild to moderate hypertension without evidence of metabolic or cardiac disturbance. Patients with established hypertension should continue their antihypertensive medications throughout the perioperative period. This is especially true for patients taking beta-blockers and clonidine. Cessation of these medications can cause rebound hypertension that could be more dangerous than any hypotensive episode experienced by continuing such medications.

For stage 3 hypertensive patients (SBP greater than or equal to 180 and DBP greater than or equal to 110) the decision to delay surgery in favor of establishing improved blood pressure control with antihypertensive medications needs to be weighed against the risk of postponing surgery. One randomized control trial involving 989 patients treated for chronic hypertension showed there was no benefit to delaying surgery. These patients presented on the day of surgery with DBP between 110 and 130. Patients were randomized into two groups. In one group surgery was canceled and the patient admitted for blood pressure control. The other group received nifedipine and proceeded to surgery. No significant differences in postoperative outcomes were noted ^[21]. Of note, with rapidly acting intravenous antihypertensives, reasonable blood pressure control can generally be attained within a few hours.

Respiratory System

Patients presenting with posterior fossa pathology should also be optimized from a respiratory standpoint. As mentioned, patients with tumors of the posterior fossa may present with preoperative deficits secondary to invasion of vital respiratory centers. These individuals may already be intubated and ventilated prior to surgery.

Patients presenting for elective surgery should be asked about respiratory comorbidities including asthma, chronic obstructive pulmonary disease (COPD), obstructive sleep apnea, and smoking history. Individuals with signs and symptoms of acute respiratory illness such as pneumonia should have their surgery delayed until the infection has been treated.

Other Considerations

A plethora of clinical studies have illustrated that poor perioperative glucose control leads to increased incidence of postoperative wound infection, longer hospital stays, and increased mortality ^[22, 23]. Questions regarding blood glucose control and medication regiments are important when assessing a patient with diabetes. These patients are also more likely to present with comorbid cardiovascular disease.

Patients presenting for posterior fossa surgery also have special concerns regarding their fluid and electrolyte status. They are often dehydrated secondary to fluid restriction, vomiting, diuretic administration, and even bleeding. Use of isoosmotic replacement fluids should be used to replete fluid and electrolyte loss with careful avoidance of hypo-osmolar/ hyper-osmolar solutions and those containing glucose.

A list of the patient's current medications also needs to be reviewed. Although most should be continued through the perioperative period, certain medications such as antiplatelet therapy or NSAIDS may be discontinued by recommendation of the surgeon or cardiology consultant.

Intraoperative Considerations

Otolaryngologist's Perspective

For many years, surgical removal has been the textbook standard of treatment of acoustic neuroma. Surgery results in complete tumor removal with a very low chance of regrowth (\sim 3%) in the majority of individuals. Technical progress has made this a safe procedure with excellent results in experienced hands. Three potential routes exist for surgical access: translabyrinthine, suboccipital (synonymous with retrosigmoid), and middle cranial fossa (or subtemporal).

In the translabyrinthine approach, the bone of the mastoid and bony labyrinth is removed to gain access to the internal auditory canal and cerebellopontine angle. This is a direct approach with very little cerebellar retraction, but that routinely sacrifices hearing because the inner ear is violated. It can be used for tumors of any size, but is best suited for small and medium-sized tumors due to the limitation of exposure allowed by the jugular bulb.

The suboccipital approach is a craniotomy placed just posterior to the sigmoid sinus that provides exposure of the entire posterior cranial fossa. It can be used to remove tumors of any size, but is most appropriate for larger tumors where broad exposure is desirable, and for smaller tumors when hearing preservation is a goal.

The middle cranial fossa approach is a craniotomy through the squamosa of the temporal bone centered above the preauricular line, which provides access to the internal auditory canal from superiorly ^[24]. It is appropriate for intracanalicular tumors when hearing preservation is desired. Division of the tentorium and superior petrosal sinus can give access to the posterior cranial fossa for tumors with extracanalicular extension, but this approach is not suitable for larger tumors. Acoustic neuroma surgery seeks to achieve total tumor removal with preservation of facial nerve function. Intraoperative facial nerve monitoring and stimulation is routinely used. The success rate of facial nerve preservation is correlated with tumor size, although the adherence of the tumor to the nerve and the experience and technical skill of the surgeon also play an important role.

For glomus tumors, surgical removal is through an "infratemporal fossa" approach. So named by Fisch ^[25], this approach begins with a mastoidectomy and facial nerve transposition to expose the jugular bulb and internal carotid artery at the skull base. An upper neck dissection exposes the internal jugular vein and cranial nerves IX through XII, and the internal jugular vein is ligated. The approach can be extended anteriorly by retracting the condyle of the mandible forward and following the eustachian tube to the petrous apex and infratemporal fossa. Intracranial exposure can be gained by combining this with a suboccipital craniotomy or, if the hearing is expendable, by adding a translabyrinthine or transcochlear approach.

Glomus jugulare surgery is complex and lengthy because of the challenges associated with working in a tight anatomical area. Collaboration between neurotology, neurosurgery, head and neck surgery, and interventional radiology teams is routine ^[26]. Risks of surgery include blood loss requiring transfusion, facial nerve weakness related to anterior transposition of the nerve (usually temporary), conductive hearing loss from middle ear obliteration, lower cranial nerve injury form tumor dissection at the jugular foramen resulting in vocal cord weakness and aspiration, and carotid injury resulting in stroke. Tumors adherent to the carotid adventitia cannot always be eradicated, and adjuvant radiation therapy is sometimes employed. Primary radiation therapy has also been employed in patients in fragile health who are unable to undergo surgery. Radiation therapy limits further tumor growth by creating obliterative endarteritis, but is not directly tumoricidal. Doses of 50-60 Gy are typical; stereotactic modalities have also recently been advocated.

Positioning

Surgical positioning for the translabyrinthine approach, as well as other trans-temporal approaches, is similar to mastoid surgery, with the patient in a supine position with the head turned 45° away from the surgeon. The head can be secured on a foam donut pillow, or a horseshoe head holder or even Mayfield pins may be used. Because these operations typically take several hours, adequate padding under pressure points (elbows, heels) should be ensured. The surgeon usually sits behind the patient's head, with the scrub nurse across the table, and the microscope stand at the top of the head. The anesthesiologist then is usually at the patient's feet, and an "ether screen" is created by clipping the drape to IV poles. Because the anesthesiologist does not have ready access to the patient's head or arms during the procedure, he/ she must be certain that the endotracheal tube is well secured and that intravenous lines are sufficient. The body must also be held securely, since the table will be tilted or "airplaned" during the operation. Straps are routinely used around the patient's torso to prevent the patient from sliding. Care should be taken to avoid wrapping too snugly around the chest, which might restrict lung inflation (recognized by high airway pressures that could result in hypercarbia and hypoxemia).

For the suboccipital/retrosigmoid approach, different surgeons have different preferences for surgical positioning. In our practice, the most common is to have the patient in a supine position with their head turned away from the surgeon. Mayfield pins are most often used, but a horseshoe head holder may be adequate if the neck is flexible. Because muscle relaxants are avoided during neurotologic surgery, it is important that a sufficient depth of anesthesia be maintained because head movement while the head in the Mayfield apparatus may result in injury. Obviously, the head must be securely pinned because slippage can occur as the operating table is moved during surgery.

Access to the cerebellopontine angle demands that the head be positioned with the neck somewhat flexed and tilted away from the surgeon; this gives the best surgical trajectory when a suboccipital (retrosigmoid) craniotomy is used. The head position should be checked and rechecked prior to draping. It is important that the surgeon have sufficient access, but it is even more important that the airway is not pinched and that venous outflow is not occluded for the duration of surgery. Visualizing the porus acousticus from a suboccipital approach demands that the patient be tilted away from the surgeon.

Other surgical positions are also commonly employed for access to the posterior fossa: the lateral decubitus ("park bench") allows the patient's head to be directed away from the surgeon with less neck flexion than the supine-head turned position. The drawback is that the patient's ipsilateral shoulder is prominent and may block the surgeon's hand (especially true for a right-handed surgeon trying to access the patient's right side). Adequate padding is paramount, and proper positioning of both upper extremities is needed to avoid pressure or traction neurapraxia. The undermost arm is usually extended forward at the shoulder so that the weight of the patient is not directly over it. The uppermost arm can be placed on a sled-type armrest or Mayo stand. Sequential stockings are routinely used for deep venous thrombosis prophylaxis. Subcutaneous heparin is avoided in neurologic surgery because of the risk of intracranial bleeding.

The sitting position is still preferred by some surgeons. The patient is kept upright with the head flexed forward, and the upper extremities and chest supported on a Mayo stand with adequate padding. This position is good for surgical access to the posterior fossa because blood and cerebrospinal fluid (CSF) drain away from the area of interest. This not only creates space for the surgeon to work, but also permits the use of two hands for surgical dissection (because one hand is freed from using the suction) ^[27]. Venous air embolism (VAE) is a potential hazard, because in the sitting position the venous system in the head is at a negative pressure, a venous tear can suck air into the right atrium of the heart ^[28]. This can be a lethal complication if not recognized and treated quickly. Some would use frequent auscultation and a central venous catheter to quickly remove air from the right atrium should this become necessary. The sitting position is contraindicated in any patient with a patent foramen ovale. Present in 25% of the population, a patent foramen ovale places the patient at increased risk for a catastrophic paradoxical left sided air embolus and stroke. There have also been cases of quadriparesis resulting from upper spinal cord infarction caused by excessive head flexion ^[29], and seizures have also been found to occur ^[30].

The middle cranial fossa approach requires that the surgeon sit at the top of the head, looking down at the temporal bone form a superior vantage. The craniotomy is made in the temporal squamosa, centered on the pre-auricular line. An extradural approach is used to access the roof of the temporal bone, requiring retraction of the temporal lobe of the brain. Mannitol is given to decrease ICP by osmotic diuresis. Excessive retraction against a tense brain can result in a temporal hematoma, a potentially devastating complication. A drill is used to open the internal auditory canal from above, using the arcuate eminence and the greater petrosal nerve as landmarks. Dissection of the tumor from the lateral end (fundus) of the internal auditory canal is easier from this approach than from a suboccipital approach, but the facial nerve is at greater risk because it is exposed immediately upon opening the dura. This approach is useful for intracanalicular tumors, and for smaller tumors with extracanalicular extension. Access to the CPA can be obtained by coagulating the superior petrosal sinus and splitting open the tentorium, but the exposure is limited and excessive retraction on the temporal lobe can be hazardous.

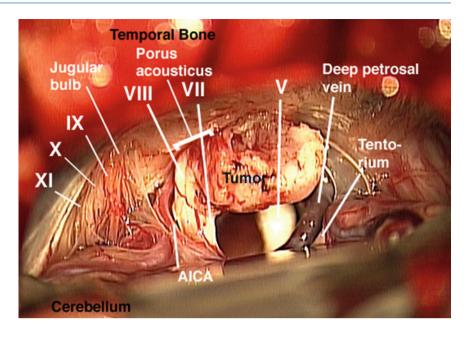
Surgical Exposure of the Posterior Cranial Fossa

The suboccipital, or retrosigmoid, approach is the most versatile surgical approach to the posterior fossa, although other approaches through the temporal bone also exist. Mannitol is usually given intravenously at the start of the case. The suboccipital area is accessed via a linear or curvilinear incision in the posterior scalp, and the nuchal muscles are detached from the occipital bone or partially divided. Branches of the occipital artery are typically encountered and coagulated during the surgical entry. The vertebral artery is vulnerable at the point that it exits the vertebral canal just before it enters the skull. A large self-retaining ("cerebellar") retractor is introduced. A silver-dollar size craniotomy is created in the occipital bone, and enlarged superiorly and anteriorly until the transverse and sigmoid sinuses are just exposed. In so doing, the mastoid air cells are usually opened, and these need to be sealed with bone wax to prevent a potential pathway for spinal fluid leakage. A dural incision is made parallel to the posterior edge of the sigmoid/transverse sinus, leaving a 3 mm cuff of dura. Retention sutures can be placed at the free dural edge to provide anterior retraction. The larger dural flap is reflected posteriorly and kept moist during the procedure. The arachnoid is incised at the inferiorly and the basal cistern is opened and drained of CSF, allowing the cerebellum to relax. Next the cerebellopontine angle cistern is opened by bluntly dissecting the arachnoid, allowing more CSF to drain. At this point, the cerebellum usually falls away, and a good view of the posterior cranial fossa is obtained. Figure 17.4 demonstrates the posterior fossa exposure during surgery.

The translabyrinthine, retrolabyrinthine, and transcochlear approaches are pre-sigmoid approaches that remove parts of the temporal bone to access the cerebellopontine angle. In the translabyrinthine approach ^[31, 32], typically used for acoustic neuromas with non-serviceable hearing, the mastoid bone is widely removed, the vestibular labyrinth is sacrificed, and the IAC is skeletonized. In the retrolabyrinthine approach [33], used for vestibular nerve section for treatment of vertigo, posterior fossa dura is exposed but the labyrinth is maintained. In the transcochlear approach ^[34], used for access to the petrous apex and pre-pontine cistern, the cochlea is also sacrificed and the facial nerve transposed. The posterior fossa dura is exposed and opened anterior to the sigmoid sinus and inferior to the tentorium, creating a posteriorly base dural flap. Because the dural opening is more anterior than in the retrosigmoid/suboccipital approach, less (if any) cerebellar retraction is needed, and the facial nerve is readily identified earlier in the procedure [35]. However, the vertical extent of the dural opening is limited by the height of the jugular bulb, and the surgical exposure is not as panoramic as with the retrosigmoid approach.

The transpetrosal approach is a hybrid approach for larger skull base tumors that require exposure above and below the tentorium ^[35, 36]. Bone is removed widely from the temporal squamosa to gain access to the middle cranial fossa, and the mastoid is also removed but the labyrinth is typically spared. The dura is incised anterior to the sigmoid sinus and over the temporal lobe, and the tentorium can then be split longitudinally to gain access to both the posterior and middle cranial fossae; the IVth cranial nerve is at risk during this maneuver. The approach provides exposure for lesions such as meningiomas that traverse the tentorium and also gives access to neurovascular structures near the clivus.

Fig. 17.4 A microsurgical view of the CPA on the left side, as seen from a suboccipital approach, showing a small acoustic neuroma ("tumor"), cranial nerves V through XI, and the anterior inferior cerebellar artery (AICA). The cerebellum is gently retracted medially



The extended middle fossa approach creates surgical access to the petrous apex and the pre-pontine cistern. A subtemporal (middle fossa) craniotomy is performed by removing the temporal squamosa. The temporal lobe is elevated from the petrous bone with dura intact. The internal auditory canal is opened superiorly. The bone anteromedial to the internal carotid artery (Kawase's triangle) is then removed, providing exposure of the anterior portion of the posterior cranial fossa.

Anatomy of the Posterior Cranial Fossa

Cranial Nerves

The anatomic structures of the posterior cranial fossa include the brainstem and cerebellum, cranial nerves IV through XII, and certain arteries and veins, as illustrated in Fig. 17.4. From the surgeon's vantage, looking into the cerebellopontine angle, the cerebellum will be at the bottom of the surgical field (posterolateral), the brainstem will be deep (posteromedial), and the cranial surface of the temporal bone will be at the top (anterolateral).

Superiorly, the tentorium cerebelli forms the roof of the posterior fossa. The deep petrosal vein travels parallel to the tentorium, and joins the superior petrosal sinus at the junction of the tentorium and superior edge of the temporal bone. The trochlear nerve (IV) runs medially near the free edge of the tentorium and is not routinely visualized. The trigeminal nerve (V) is large, exits the pons, and is situated just inferior to the tentorium; it travels anteriorly and will form the trigeminal (Gasserian) ganglion in Meckel's cave, a dural pouch on the dorsal surface of the temporal bone which is not seen in this approach. The abducens nerve (VI) travels in a very medial position from the pons to the anterior-most edge of the tentorium; it exits the cranial cavity near the posterior clinoid process through a dural sleeve named Dorello's canal. The vestibulocochlear nerve (VIII) will be at the center of the surgical field. It exits the brainstem at the pontomedullary junction and enters the porus acousticus of the temporal bone. From there it travels through the internal auditory canal to supply the end-organs of hearing and balance in the inner ear. The facial nerve (VII) also exits the pontomedullary junction and travels to the internal auditory canal through the porus, but it is anterior to the VIII nerve and is hidden from the surgeon's view until VIII is reflected inferiorly. The internal auditory canal can be opened by incising and elevating the dura circumscribing the porus and drilling the posterior bony lip in a media-to-lateral direction. This is routinely done in acoustic neuroma surgery to expose the canalicular portion of the tumor.

Inferiorly, the lower cranial nerves IX, X, and XI (glossopharyngeal, vagus, and accessory) arise from the medulla and exit the skull at the jugular foramen. The accessory nerve has a spinal and a cranial component; the spinal cord and enters the skull through foramen magnum to join the cranial component near the jugular foramen. The jugular bulb itself can usually be seen as a bluish structure through the dura, as can the sigmoid sinus that feeds into it. Above the jugular foramen is a white fold of dura on the temporal bone surface, which corresponds to the endolymphatic sac. The hypoglossal nerve (XII) is very inferior and medial and exits the skull through the hypoglossal canal, which is part of the occipital bone just above foramen magnum.

Arterial Supply

The arteries of the posterior fossa arise from the vertebrobasilar trunk. The anterior inferior cerebellar artery (AICA) is routinely seen; it forms a loop near the VII-VIII nerve complex and gives rise to an internal auditory branch that exits at the porus acousticus. AICA supplies circulation to the brainstem and so injury to it can have grave consequences. The posterior inferior cerebellar artery (PICA) is tortuous and of variable size, and travels more inferiorly in the cerebellopontine angle. The superior cerebellar artery (SCA) is more superior and medial and is not routinely encountered except during trans-cochlear approaches. The basilar artery itself occupies a median position and can sometimes be seen from this surgical vantage. The internal carotid artery enters the skull anterior to the jugular bulb in the temporal bone, and travels superiorly and medially through the carotid canal within the temporal bone to exit near foramen lacerum where it enters the cavernous sinus. The internal carotid artery is not visualized through a suboccipital craniotomy, but is encountered during infratemporal fossa approaches for glomus jugulare tumors.

Venous Drainage

The venous drainage of the posterior fossa is mainly through the transverse and sigmoid sinuses into the jugular bulb and then the internal jugular vein. The mastoid emissary vein is a large diploic vein that is often encountered when removing bone near the sigmoid sinus. It is usually managed by occluding it with bone wax. The deep petrosal vein is a large intracranial tributary, which joins the superior petrosal sinus to empty anteriorly into the cavernous sinus. The inferior petrosal sinus runs between the jugular bulb and cavernous sinus and travels inferiorly within the temporal bone. It is not encountered in a suboccipital approach but does have to be dealt with when the jugular bulb is opened during glomus tumor surgery. The vein of Labbe is the dominant drainage of the temporal lobe of the brain. It enters the transverse sinus from superiorly, near its junction with the sigmoid sinus. The vein of Labbe is at risk during middle cranial fossa surgery, and injury to this vein can result in venous infarction of the temporal lobe, a grave sequela.

Cranial Nerve Monitoring During Neurotologic Surgery

The facial nerve supplies motor function to the muscles of facial expression, stapedius, and posterior digastric muscles. It also supplies common sensation to the external ear meatus, taste sensation (via chorda tympani branch) to the anterior two thirds of the tongue, parasympathetic fibers to the submandibular and sublingual salivary glands and nasal glands. The facial nerve is at risk of surgical trauma during posterior skull base procedures and especially during acoustic neuroma surgery. All degrees of surgical trauma are possible stretch injury can lead to neurapraxia (reversible bruising of the nerve), while more severe manipulation can lead to axonotmesis (disruption of axons with preservation of neural tubules), and surgical transection of the nerve can lead to neurotmesis (disruption of axon and axon sheath) and more severe grades of injury (disruption of nerve fascicles or transection of the entire nerve).

Continuous monitoring of the facial nerve has become routine during acoustic neuroma surgery and is also used during most neurotologic procedures ^[37]. The most common method is to use needle electromyographic (EMG) electrodes in the orbicularis oris and orbicularis oculi muscles. These will detect the action potentials caused by contraction of the facial muscles. Commercially available EMG units will display the EMG tracings and produce an audible signal in response to facial muscle contraction. The audible signal provides live feedback to the surgeon during the dissection. Continuous intraoperative monitoring and stimulation of VII demands that patient not be paralyzed during surgical dissection.

EMG monitoring produces various types of responses ^[38, 39] "Burst" responses are brief, non-repetitive signals that occur in response to brief mechanical stimulation of the nerve, such as when brushing against an exposed nerve with a blunt instrument. These do not usually cause permanent injury unless the disturbing activity is forceful or sustained. "Train" responses are repetitive signals that occur in response to mechanical traction on the nerve, or after thermal stimulation, such as saline irrigation. The surgeon should cease whatever activity produced the train response to avoid permanent injury.

The facial nerve can also be electrically stimulated during surgery using a monopolar probe to apply a small current to the nerve ^[40]. This provides immediate information about the anatomic location and the functional integrity of the nerve. Applying the stimulator probe proximal to where the surgeon is working assures him or her that the nerve has functional integrity. Stimulators are usually set to deliver a constant current, and the current level is pre-selected. Typically, a very small current (0.1 mA) will stimulate a bare nerve. Direct electrical stimulation of the nerve by an electrical probe elicits a "pulse" response, which is a synchronous repetitive signal. In the setting of facial nerve injury, the nerve will continue to stimulate distal to the site of the lesion for up to 3 days, even after complete transaction. Stimulation as to

the degree of injury; brisk stimulation at a low current level implies functional continuity of the nerve, while a high stimulation threshold or complete absence of stimulation implies a severe conduction block or a transected nerve. Constantcurrent stimulation is susceptible to shunting, wherein the current is dissipated by any electrolytic fluid (blood, saline, CSF) in the surgical field; the field should be dried with suction before attempting to stimulate the nerve.

The VIIIth nerve mediates hearing and balance function. While the vestibular nerve is routinely sacrificed during acoustic neuroma surgery, preservation of hearing can be achieved in certain patients with smaller tumors and serviceable preoperative hearing. Preoperative auditory brainstem response (ABR) testing helps determine neural integrity and is a good predictive test of hearing preservation. Intraoperative recording of auditory function is valuable to providing feedback to the surgeon during neurotologic surgery or acoustic neuroma and other posterior fossa tumors. This can be done with continuous auditory brainstem evoked potentials (ABR), direct cochlear nerve action potentials, or electrocochleography (ECoG). All three modalities use sound stimuli delivered to the operative ear through an insert earphone; the contralateral ear can be used as a control. ECoG is a near-field recording of the electrical discharges of the cochlea using a trans-tympanic needle electrode on the promontory, but is not reliable for determining hearing preservation because ECoG activity continues even after the cochlear nerve is severed ^[41].

Direct eighth nerve recording is instantaneous and robust and can be captured by a cotton-tipped bare wire electrode placed directly on the cochlear nerve near its entry point in the brainstem. Certain authors have shown that direct eighth nerve recording can improve hearing preservation outcomes in acoustic neuroma surgery ^[42], and that the response is more reliable than ABR recording during surgery ^[43]. While the rapidity and strength of the response are beneficial, electrode migration can make this modality cumbersome during surgery. The surgeon must be comfortable performing tumor dissection with the electrode in the surgical field, and must stop and reposition the electrode when the waveforms are lost during surgery.

Intraoperative ABR testing in the most widely used modality for measuring intraoperative auditory function. ABR is a far-field response, recorded by surface electrodes on the scalp. The response is an evoked potential, elicited in response to repetitive click or tone-burst stimuli (clicks give a more robust response), which are time-locked and summated to produce a patterned response consisting of up to five waves, generated at different points in the auditory neural pathway, occurring between 1 and 10 ms after the onset of the stimulus. The latency and amplitude of the most robust waves, 1 and 5, are followed continuously during surgery. Loss of amplitude or rise in latency alerts the surgeon to an adverse surgical event and may allow the nerve to recover before irreversible damage occurs. Because ABR is a far-field recording, there is a time delay between the surgical event and a change in the waveform. While this is a notable disadvantage, the stability of the electrode positioning makes this a valuable adjunct during surgery ^[44]. Typically, more than 1,000 repetitions are necessary to obtain a recognizable response; continuous "real-time" recording makes it possible to sample waveforms continuously during the surgical dissection. Preservation of ABR responses is usually but not always predictive of postoperative hearing preservation. Loss of intraoperative ABR waveforms is usually but not always associated with postoperative hearing loss.

None of these auditory evoked potentials are affected by general anesthesia, but hypothermia may diminish or abolish the response. The quality of recording can be corrupted by ambient acoustic or electrical noise. The OR suite is by nature a noisy and hostile recording environment. Anesthetic monitoring equipment can interfere with VIIIth nerve monitoring, and adequate isolation and grounding may be difficult to achieve.

Lower Cranial Nerves (IX, X, XI)

These are at theoretical risk during acoustic neuroma surgery, but can usually be identified anatomically before tumor removal and protected with cottonoids. The lower cranial nerves are at significant risk during glomus jugulare surgery when dissecting tumor from the jugular foramen. Intraoperative monitoring is difficult. A silver-foil recording electrode wrapped around the endotracheal tube can be used to monitor vocal cord function during surgery, and this is commercially available (Medtronic[®] Nims tube). This is used by some surgeons during thyroidectomy (Chap. 14) but is rarely used during skull base surgery. In cases with suspected vagus nerve injury, a nasogastric feeding tube should be placed intraoperatively and the patient kept NPO postoperatively until a swallowing evaluation is completed.

V Nerve

Large acoustic neuromas can cause facial hypesthesia if they grow upward to press on the trigeminal nerve. The trigeminal cannot be monitored intraoperatively, although electrical stimulation of the motor branch of V3 can cause masseter muscle contraction which can be misinterpreted as a facial nerve response.

CSF and ICP

CSF is contained within the subarachnoid space. Posterior fossa surgery is often done through a limited bony opening, and it is therefore important to control ICP at the time of the dural opening. Mannitol is usually given at the start of surgery to decrease ICP. Hyperventilation is also helpful. Lumbar CSF drains are not routinely used. After dura is opened, gentle dissection with a sharp instrument is used to open arachnoid and drain the CPA cistern. More inferior exposure is needed to drain the basal cistern, which is the larger CSF reservoir. Once this is done, the brain softens and little pressure is needed for cerebellar retraction.

Infectious Considerations

Most neurotologic surgery is done for tumors in a closed cavity and therefore "clean." The ear communicates with the upper aerodigestive tract through the eustachian tube, but is normally sterile. Antibiotic prophylaxis is routinely givencefazolin for surgery confined to the mastoid, and ceftriaxone or nafcillin and vancomycin when the cranial cavity is entered. Clindamycin can be substituted in penicillin-allergic individuals. Antibiotics are not continued postoperatively, per current guidelines. If the patient has an active infection, appropriate antibiotic coverage as determined by culture and sensitivity supersedes these recommendations, and is continued until the infection resolves. Patients with CSF leaks of traumatic or post-surgical origin are not routinely given antibiotic prophylaxis coverage for the leak as this probably does not prevent meningitis and may select for resistant bacterial strains. However, if an active infection is present, antibiotics should not be withheld.

Intraoperative Blood Loss

Blood loss during acoustic neuroma surgery is usually manageable and does not require transfusion. In translabyrinthine surgery, the sigmoid sinus is unroofed and gently compressed, and venous bleeding may occur at the margins where the vein is tethered to the bone. If not controlled by immediate pressure, venous hemorrhage may quickly result in significant blood loss. Surgeons should be aware that applying suction to the bleeding site may result in greater blood loss than is appreciated. A better method is to apply gentle compression to the vein with finger or blunt instrument and then slowly release pressure to isolate the point of bleeding. The bleeding can then be controlled by placing thrombin-soaked gelatin (Gelfoam[®]) within the venous tear. Intraluminal packing may embolize, so this should be avoided unless the distal internal jugular has been ligated. Glomus tumors are vascular by nature and the surgical blood loss may be substantial. Preoperative angiography and embolization help to minimize the intraoperative blood loss. Patients should be routinely typed and cross-matched before surgery. These tumors frequently occupy the lumen of the jugular bulb. The internal jugular vein is ligated in the neck, and the sigmoid sinus is compressed with extraluminal Surgicel[®] packing before opening the jugular bulb. Backbleeding from the inferior petrosal sinus occurs as the tumor is lifted out of the jugular lumen and is controlled by Surgicel packing. After the tumor is removed, the inferior petrosal lumen can be more precisely occluded with gelatinthrombin.

Dissection of tumors from the carotid adventitia can be treacherous. Preoperative arteriography can help to estimate the degree of carotid involvement. In cases where the carotid is encased in tumor, total tumor removal might be impossible. Strategies for dealing with carotid artery include preoperative stenting, intraoperative shunting and extra-intracranial arterial bypass, or carotid sacrifice (a preoperative trial balloon occlusion and measurement of backflow pressures in the angiography suite under local anesthesia might help determine the patient's ability to tolerate carotid occlusion). Intraoperative carotid bleeding can be life threatening. Proximal control of the vessel can be achieved with vascular loops or atraumatic clamps, but distal control of the vessel might be impossible where it enters the base of skull. In these cases, bleeding should be controlled temporarily by external compression and definitively by endovascular means.

Anesthesiologist's Perspective

Pre-induction

Positioning

One of the challenges of operating in the posterior fossa involves the choice of patient positioning. The infratentorial compartment can be accessed via the sitting position as well as a number of horizontal positions (prone, lateral, and parkbench), each with their associated advantages and disadvantages.

Historically, the "sitting" or "head-up" position was chosen by surgeons for patients undergoing posterior fossa surgery. This position offers superior surgical exposure, improved venous and CSF drainage, and reliable hemostasis. Advantages to the anesthesiologist include better access to the upper extremities and endotracheal tube and improved ventilation ^[28]. Use of this position has declined in recent years, however, due to the potential for serious complications. Furthermore, improvements in surgical technology have made use of the horizontal position more practical. Complications of sitting craniotomy include an increased incidence of venous air embolism, pneumocephalus, hemodynamic lability, quadriplegia lingual and laryngeal edema, and peripheral nerve palsies. Given the higher incidence of VAE in the sitting versus horizontal position, presence of a patent foramen ovale (PFO) represents a significant contraindication to surgery in this position. Preoperative screening for PFOs via transesophageal echocardiography and subsequent closure have been recommended where utilization of the sitting position for posterior fossa surgery has overwhelming advantages [45]. One group comparing posterior fossa surgery in 260 patients performed in the sitting versus horizontal position found that operating in the sitting position resulted in significantly less blood loss and transfusion as well as improved preservation of cranial nerve function. The same study illustrated that the incidence of VAE was significantly higher in the sitting versus horizontal position and that the incidence of postoperative seizures (although low for intratentorial surgery) was higher in patients in the "head-up" position. Interestingly, the incidence of hemodynamic instability and need for postoperative ventilation was similar for both groups.

Another pitfall encountered in surgery of the posterior fossa is pneumocephalus (intracranial air entry). In addition to delayed emergence it can lead to postoperative lethargy, headaches, confusion, cranial nerve deficits, and hemiparesis. The incidence varies with position and is estimated at 100% in the sitting position ^[46].

Each position offers advantages and disadvantages and position selection must be performed on a case-by-case basis weighing the overall risks and benefits of each (Table 17.2).

Monitoring

As with any anesthetic, standard American Society of Anesthesiologists (ASA) monitoring is performed on a continual basis to ensure adequate oxygenation, ventilation, circulation, and temperature. These monitors include the five-lead ECG, blood pressure measurement, pulse oximetry, and capnography. Additionally, specific monitoring modalities exist for posterior fossa surgery to aid in diagnosis and treatment of VAE and to ensure adequate central nervous system perfusion.

Invasive Monitors and VAE detection

Invasive blood pressure monitoring with an arterial line is indicated for posterior fossa surgery. Beat-to-beat monitoring of the blood pressure helps optimize cerebral perfusion pressure (CPP) while avoiding increases in ICP. Sampling of

 Table 17.2
 Advantages and disadvantages to the "sitting" position during posterior fossa surgery

Advantages	Disadvantages
Optimal surgical exposure	Venous air embolism
Improved venous and CSF drainage	Paradoxical air embolism
Hemostasis	Airway obstruction
Improved ventilation	Hemodynamic instability
Airway accessibility	Macroglossia
	Pneumocephalus
	Cranial nerve palsy
	Quadriplegia

 Table 17.3 Monitoring modalities available for the diagnosis of venous air embolism

Monitoring modality		
Transesophageal echocardiography		
Precordial Doppler		
Capnography (end-tidal carbon dioxide)		
Esophageal stethoscope		
End-tidal nitrogen fraction		

arterial blood also assists with ventilation, fluid, electrolyte, and transfusion management. A central venous catheter is often placed after induction. Besides providing ideal access for administration of medications, fluids, and blood products, it also acts as a means by which to aspirate air should a VAE occur. Assuming adequate venous access can be obtained peripherally, insertion of a central venous catheter is more critical in cases where surgery is to be performed in the sitting position.

To expedite diagnosis and treatment of VAE, a precordial Doppler ultrasound probe or transesophageal echocardiogram (TEE) is often utilized. Although more sensitive, the TEE is also more invasive and expensive. With proper placement, the precordial Doppler can consistently detect small amounts of air entrainment at low infusion rates ^[47] and is often more practical. Incorporating either one of these monitoring modalities is recommended when anesthetizing a patient for posterior fossa surgery in the sitting position (Table 17.3).

Neurophysiologic monitoring modalities such as somatosensory evoked potentials (SSEPs), auditory brainstem responses (ABR), motor evoked potentials (MEPs), and electrocephalogram (EEG) are being used with increasing frequency in neurosurgery. Posterior fossa surgery is no exception. These modalities are used to continuously monitor the integrity of the central nervous system during surgery. ABRs have been widely used for posterior fossa surgery and are valuable in preservation of the eighth cranial nerve during microvascular decompression and surgery for CPA tumors ^[48]. Continuous electromyographic monitoring of the VIIth cranial nerve (facial) is a sensitive and reliable method of identifying and preserving neuronal function during CPA surgery ^[49]. Somatosensory evoked potentials and MEPs have also shown to be valuable in monitoring for cortical and motor compromise during posterior fossa surgery ^[50]. Knowledge of which monitoring modalities are to be utilized during the operation is important in planning the anesthetic as avoidance of certain agents and the utilization of others may be necessary.

Induction

Patients, understandably, are often nervous prior to surgery. Premedication can be a helpful adjunct in mitigating preoperative anxiety. However, if not done vigilantly and selectively, premedicating patients with elevated ICP can lead to further elevation if patients are allowed to hypoventilate and $PaCO_2$ to rise. When appropriate, small doses of midazolam or fentanyl can be carefully titrated in a monitored setting.

Prior to induction, all ASA standard monitors need to be applied. If, during the preoperative assessment, the patient was known or suspected of having a difficult airway, emergency equipment and additional personnel should be available. Information regarding preoperative elevation of ICP and baseline blood pressure should be established. The decision for placement of a preinduction arterial line should be based on the patient's clinical status and medical comorbidities. Should the patient present with clinical signs and symptoms of elevated ICP, labile hemodynamic status, or other potential for end-organ damage (recent MI, etc.), then a preinduction arterial line should be placed. Due to the many potential reasons for delayed emergence (brainstem swelling, surgical retraction, etc.), it is sensible to use short-acting agents that will allow for rapid emergence without confounding the postoperative neurological exam. Choosing an induction agent such as propofol or etomidate that preserves cerebral autoregulation and promotes rapid emergence at the conclusion of the case is reasonable. Titration of a short-acting opioid such as fentanyl or remifentanil at induction assists in deepening the anesthetic and helps blunt the sympathetic response to laryngoscopy. Selection of an intermediate acting, non-depolarizing muscle relaxant should be used to assist laryngoscopy but redosing is best avoided if facial nerve monitoring is to be utilized. Prior to facial nerve monitoring, one should confirm that twitches have returned via use of a train-of-four monitor. Hemodynamic lability is a concern during the induction period. Additional opioids or IV induction agents as well as antihypertensive medications should be readily available

should the patient become hypertensive. Conversely, sympathomimetics such as phenylephrine and ephedrine should be used to treat hypotension.

Regardless of which surgical position is to be utilized, the patient's airway will more than likely be away from the anesthesiologist which makes vigilant endotracheal tube securement after intubation very important. Confirmation of bilateral breath sounds, appropriate airway pressures and that the endotracheal tube is free from kinks or obstruction should be checked.

Ventilation

Positive-pressure ventilation offers numerous advantages compared to spontaneous ventilation in patients undergoing posterior fossa surgery. In addition to promoting cerebral vasoconstriction and lowering ICP it helps to minimize blood loss. In cases where muscle relaxation is appropriate (i.e., operations not involving facial nerve monitoring) positivepressure ventilation allows for lighter levels of anesthesia with less concern for patient movement.

Anesthetic Agents

A variety of factors play a role in determining which agents are most appropriate for anesthesia maintenance in posterior fossa surgery. Selecting maintenance agents that will facilitate rapid emergence after potentially lengthy surgery is important for early postoperative neurologic assessment. The ideal anesthetic should preserve coupling between cerebral blood flow and cerebral metabolic rate (CMRO₂), maintain cerebrovascular autoregulation, and minimize increases in cerebral blood flow and CMRO₂. The use of electrophysiologic monitoring modalities such as SSEPs may limit the concentration of volatile anesthetics that can appropriately be used. And facial nerve electromyography may preclude the use of muscle relaxants for the majority of the case.

TIVA Vs. Volatile agents

Volatile anesthetics increase cerebral blood flow while decreasing CMRO₂. One review illustrated that the vasodilatory effects of sevoflurane are less than those of other commonly used volatile agents such as desflurane and isoflurane making it potentially more appealing for neurosurgical procedures ^[51]. The same group compared sevoflurane and propofol as maintenance agents for craniotomies. The neurophysiologic parameters reviewed included effects on cerebral blood flow and metabolism, cerebral blood flow autoregulation and cerebral blood volume and ICP. They concluded that the coupling between regional and cerebral

blood flow and metabolism is maintained with both sevoflurane and propofol. They also found that cerebral autoregulation was well maintained under 1.0 MAC of sevoflurane and that propofol does not impair cerebrovascular autoregulation regardless of concentration. Propofol was found to be superior to sevoflurane in its ability to decrease cerebral blood volume and ICP making it a better choice in cases where ICP elevation is concerning. This group also concluded that sevoflurane may be better suited for patients with preexisting cardiovascular morbidity and propofol superior in those with a history of nausea and vomiting.

Another review found that TIVA (usually administered as propofol with a synthetic opioid such as fentanyl or remifentanil) is similar to volatile anesthetics in terms of hemodynamic stability, emergence and extubation times, early cognitive function, and adverse events. Highlighted, however, is the fact that results from several randomized trials have suggested that ICP is decreased and CPP is increased in patients receiving TIVA compared to those receiving volatile anesthetics during elective craniotomy. Regardless of which anesthetics are chosen, the delivery of a carefully titrated anesthetic is more important than the individual agent selected.

Opioids

Opioids have a reliable history when it comes to neurosurgery. Physiologically, they decrease CMRO2 and ICP and negligibly effect cerebral blood flow ^[52]. Using short-acting agents such as fentanyl or remifentanil are preferred in order to facilitate rapid emergence at the end of posterior fossa surgery. Morphine is best avoided due to its more sedative qualities, longer duration of action, and vasodilatation secondary to histamine release resulting in increased cerebral blood flow.

Muscle Relaxation

As mentioned, utilization of facial nerve monitoring may preclude use of muscle relaxants for major portions of posterior fossa operations. However, medium-acting non-depolarizing agents such as vecuronium, rocuronium, or cisatricurium are acceptable for induction and intubation or periods where the facial nerve is not being monitored.

N_2O

Much controversy exists around the use of nitrous oxide in neurosurgery. Due to its ability to expand air filled spaces, many clinicians discourage its use due to higher incidences of VAE and pneumocephalus while others still support its use. The advent of newer, more blood insoluble volatile agents such as desflurane, make the use of nitrous oxide less appealing.

Fluid and Transfusion Management

Few subjects are more controversial in neurosurgery and neuroanesthesia than fluid and transfusion management. In spite of the large volume of literature that has been published over the past 15-20 years regarding blood transfusion practices, precisely when a blood transfusion should be administered to a neurosurgical patient is unclear. What most people tend to agree with is that the decision to transfuse should be based on an individual's comorbidities, the clinical scenario (ongoing bleeding), and evidence of inadequate perfusion and oxygenation of vital organs. The ASA practice guidelines for intraoperative transfusion guidelines recommend monitoring for blood loss via visual assessment of the surgical field as well as keeping an eye on suction canisters and sponges. Monitoring for inadequate perfusion and oxygenation of vital organs is done by monitoring blood pressure, heart rate, O2 saturation, pH from an arterial blood gases, lactic acid, mixed venous O₂ saturation, and echocardiography. Most experts agree that transfusion below a hemoglobin level of 6 is indicated for healthy individuals and that most patients do not necessitate transfusion above a hemoglobin level of 10. Between 6 and 10. it is pertinent to consider the patient's risk factors for inadequate oxygen perfusion (i.e., low cardiopulmonary reserve or increase oxygen demand), the risk of ongoing bleeding, and the patient's intravascular volume status (ASA guidelines). Evidence also exists that hemoglobin concentrations below 6 g/dl (hematocrit of approximately 18%) impairs cognitive function and memory in humans and that maintenance of a hematocrit level of 30-33% improves neurologic outcome [53, 54].

For decades, restrictive fluid management has been the treatment of choice for patients with mass lesions, cerebral edema, and intracranial hypertension. The logic being that overzealous fluid administration would cause new or worsening cerebral edema. However, extreme fluid restriction can lead to hypotension and compromised cerebral perfusion pressure which can be equally devastating. Generally, intraoperative fluid administration should be given at a rate adequate to replace urinary output and insensible losses. Hypo-osmolar crystalloid and glucose-containing solutions should be avoided, instead utilizing iso-osmolar crystalloids such as plasmalyte for adequate resuscitation. The use of hypertonic solutions may be indicated in order to decrease ICP in cases where mannitol or furosemide has failed but should generally be avoided as first-line therapy.

Postoperative Considerations

Otolaryngologist's Perspective

The specific complications of acoustic neuroma surgery are posterior fossa hemorrhage, CSF otorhinorrhea, facial nerve weakness, vertigo and imbalance, hearing loss, headache, facial numbness, and other cranial neuropathies. Patients routinely kept in the intensive care unit (ICU) for 24 h postoperatively, for continuous monitoring of vital signs and neurologic status. Patients are extubated in the operating room whenever possible, or as soon after surgery as their respiratory status allows. Blood pressure control is crucial, and the mean arterial pressure (MAP) should be kept below 100 mm Hg using antihypertensive drugs. An arterial line may be helpful. The level of consciousness is evaluated as early as the patient awakens and monitored hourly for the first 24 h. Narcotic analgesics are given judiciously to avoid blunting the mental status. Declining mental status is of grave concern after posterior fossa surgery and should mandate imaging to search for hemorrhage or stroke. Posterior fossa hemorrhage is a dire emergency after neurotologic surgery, and should be treated by urgent decompression in the operating room.

CSF leaks occur in about 10% of cases, in most surgical series. Meticulous dural closure and sealing of air cells at the end of surgery is imperative. Serial lumbar taps in the first three postoperative days may reduce the total incidence. Nevertheless, CSF can find its way through air cell tracts in the mastoid. Most leaks occur in the first few days after surgery but delayed CSF leaks also occur. CSF leaks are heralded by clear fluid from the nose or wound edges, or a fluid collection under the wound. When they are recognized, CSF leaks must be treated promptly because of the risk of meningitis. Conservative treatment with diuretics, stool softeners, and lumbar drainage are occasionally effective, but most patients with voluminous leaks will have to return to the operating room for surgical repair. This consists of reexploration of the wound and packing with fat or a tissue flap. Exposed air cells should be resealed with bone wax. Leaks originating from the internal auditory canal can be repaired extracranially using a translabyrinthine approach. Care should be taken to avoid disrupting the facial nerve during this procedure.

Headache is common in the first few days postoperative, but severe or prolonged headache is uncommon, and can compromise an otherwise good surgical result. Bone dust in the surgical field and muscle tension at the wound closure site appear to be the causes of disabling headaches after acoustic neuroma surgery. The first factor can be controlled by using Gelfoam-thrombin at the margins of the surgical field to absorb bone dust during drilling. The second factor can be lessened by performing a cranioplasty at the end of surgery to prevent nuchal muscles from attaching to dura. Hearing loss remains common after acoustic neuroma surgery, despite efforts to preserve hearing. Partial hearing loss can be habilitated with hearing aids, especially if adequate speech discrimination is maintained. Total hearing loss has traditionally been habilitated with the use of a CROS hearing aid (contralateral routing of signals), but long-term patient satisfaction is low. The osseointegrated hearing device (Baha) is a recent development that has earned greater acceptance. This requires implantation of a titanium screw ("flange fixture") into the postauricular bone, a minor surgical procedure, attached to a metal abutment to which a boneoscillator hearing aid is attached. Sound is captured from the deaf side and is efficiently routed to the normal side through bone conduction.

Loss of vestibular function routinely occurs after acoustic neuroma surgery but the impairment is usually temporary as compensation occurs. The degree of postoperative imbalance depends on the amount of remaining preoperative vestibular function. Patients usually awaken from surgery with vertigo and nausea, and this typically lasts from 3 to 5 days. Anti-vertigo and antiemetic drugs such as droperidol, ondansetron, dexamethasone, and prochlorperazine can ameliorate this. Patients are encouraged to get out of bed with assistance as early as they can, usually by the third postoperative day. Gait ataxia occurs to a variable degree, and recovers over a period of 3-6 weeks. Ambulation and balance exercises are important to ensure complete and rapid recovery. Younger patients usually recover faster. Vestibular physical therapy may be beneficial in patients who are slow to progress.

Facial nerve paralysis after acoustic neuroma may be temporary or permanent. The immediate postoperative status of the facial nerve is an important predictor of final outcome; the presence of facial movement implies nerve continuity and a good prognosis even if function declines in the first few days. Full recovery of facial nerve function can be rapid after neuropraxic injury, but can take 9 months or longer after more severe nerve injury. Eye protection using artificial tears, lacrilube, and a bubble chamber or lid taping is important to prevent exposure keratitis. A tarsorrhaphy can be performed if exposure develops. Insertion of a gold eyelid weight ("lid loading") is a simple and helpful adjunct that should be done if the paralysis is expected to last longer than a few months. This procedure can be reversed in the office if necessary. Older patients with lower lid laxity or persistent scleral show should have oculoplastic consultation and undergo a lower lid suspension procedure as well as a gold weight or eyelid spring.

Permanent facial paralysis can be treated by a variety of facial reanimation procedures including hypoglossal-facial nerve transfer, masseter or temporalis sling, gracilis muscle transfer. These procedures are selected individually based on the degree and regional distribution of weakness and are usually in the province of the facial reconstructive surgeon.

Anesthesiologist's Perspective

Emergence after posterior fossa surgery requires planning and exquisite attention to detail. Facilitating rapid emergence, preventing hypertension, and minimizing endotracheal tube stimulation are important objectives.

Emergence

Emergence from posterior fossa surgery requires careful planning. Facilitating rapid emergence, preventing hypertension, minimizing strain on the endotracheal tube and preventing nausea and vomiting are important objectives. As mentioned, the use of short-acting anesthetics and opioid agonists such as fentanyl and remifentanil help to ensure a timely emergence. The use of antihypertensives such as labetalol and nitroglycerin should be employed to treat emergence hypertension and antiemetics should be used liberally to help prevent postoperative nausea and vomiting. Patients undergoing posterior fossa surgery have many reasons for delayed emergence. For this reason, the use of short-acting anesthetics is recommended in order to facilitate timely neurological assessment. The decision to extubate the patient should be based upon the preoperative neurologic status, extent of the surgery, and the amount of postoperative brainstem swelling. Failure to emerge following discontinuation of anesthesia in a previously neurologically intact patient should prompt immediate diagnostic imaging.

Postoperative Period

Proper care for the patient undergoing posterior fossa surgery extends beyond the operating room and into the postoperative period. It is recommended that all patients undergoing posterior fossa surgery be admitted to the neurosurgical intensive care unit (or similar monitored setting) for management by a critical care team. Regardless of the postoperative clinical status, such patients will have neurologic, respiratory, and fluid and electrolyte needs best managed by a specialist in a controlled, monitored setting.

Pain Management

Pain after craniotomy remains a clinical dilemma for both patients and physicians and remains poorly managed and undertreated. Often, clinicians erroneously believe that craniotomies cause little pain and that liberal use of opioid medications will preclude neurological evaluation and cause neurological deterioration. Besides being unpleasant, postcraniotomy pain has been shown to worsen perioperative morbidity and mortality due to elevation in sympathetic tone. Such sympathetic stimulation increases the risk of cardiac complications and secondary intracranial hemorrhage. Current literature suggests analgesia after craniotomy is best accomplished using a combination of opioids, acetaminophen, anticonvulsants, and possibly NSAIDS. Although, scalp infiltration with local anesthesia has not been shown to consistently reduced acute postoperative pain, one group recently showed its value in decreasing the incidence of chronic pain ^[55].

Conclusion

Neurotology can present several challenges for the perioperative team. Proper and thorough communication between surgeons and anesthesiologists, as with many complex procedures, can save time and assure safe care.

Clinical Insights

For the Otolaryngologist (from the Anesthesiologist)

• Due to the proximity of the surgery to the facial nerve, neuromuscular blocking agents will not be used. Therefore the patient could move if sudden stimulation occurs. Communicate with the anesthesiology team prior to initiating surgery is critical.

For the Anesthesiologist (from the Otolaryngologist)

- The anatomy of the posterior fossa and the vital structures contained within it make it a precarious place in which to operate.
- Issues such as patient positioning, the potential for hemodynamic lability and cranial nerve damage, as well as lengthy surgery make these cases very challenging.
- Neurotologic surgery involves the skull base and the posterior cranial fossa. Airway management is usually uncomplicated, but facial nerve monitoring, performed almost routinely, requires that muscle relaxants be avoided. This means maintaining an adequate plane of anesthesia for an often lengthy procedure using inhalational and intravenous anesthetics and narcotics without the use of paralytic agents.

- Mannitol is usually administered at the start of surgery to soften the brain. Hyperventilation also helps reduce CSF tension until the CP angle cistern can be drained.
- The anesthesiologist should be vigilant of changes in vital signs during dissection near the brainstem.
 Brainstem stimulation leads to bradycardia and hypotension; prolonged stimulation can lead to death.
- Air embolism is possible if a large vein is lacerated, especially if the patient is in a sitting position.
- In glomus tumor surgery, intraoperative hemorrhage can be significant, so rapid fluid repletion may be needed, and typed and cross-matched blood should be available.
- Surgical injury to the lower cranial nerves can result in swallowing impairment and aspiration.

Clinical Pearls

For the Otolaryngologist (from the Otolaryngologist)

- Having a regular team of perioperative care providers (surgeons, anesthesiologists, nurses and technicians) involved in the management of these patients is important. Such consistency promotes not only excellent care but also fosters superior communication between all involved personnel.
- The anesthesiologist's primary objective is to safely see the patient through a difficult and potentially dangerous operation. Central to this objective is providing the surgeon with ideal operating conditions while maintaining patient safety. Communication of needs as to operating conditions is paramount to this, and it is your job to communicate your surgical needs early.

For the Anesthesiologist (from the Anesthesiologist)

- Continual communication with the surgeons and neuromonitoring personnel are vital to good outcomes.
- These are intracranial procedures, treat them accordingly (i.e., arterial line, potential for large blood loss, potential for raised intracranial pressure and mental status changes).
- Neuromuscular blockade must be avoided. If used for airway management, they should be reversed and resolution of blockade confirmed.

References

- Evaluation ASoATFoP. Practice advisory for preanesthesia evaluation: a report by the american society of anesthesiologists task force on preanesthesia evaluation; 2002.
- Smouha EE, Yoo M, Mohr K, Davis RP. Conservative management of acoustic neuroma: a meta-analysis and proposed treatment algorithm. Laryngoscope. 2005;115:450–4.
- Kondziolka D, Lunsford LD, McLaughlin MR, Flickinger JC. Long-term outcomes after radiosurgery for acoustic neuromas. N Engl J Med. 1998;339:1426–33.
- Sekhar LN, Gormley WB, Wright DC. The best treatment for vestibular schwannoma (acoustic neuroma): microsurgery or radiosurgery? Am J Otol. 1996;17:676–82.
- Lawson W. The neuroendocrine nature of the glomus cells: an experimental ultrastructural and histochemical tissue culture study. Laryngoscope. 1980;90:120–44.
- Rosenwasser H. Carotid body tumor of the middle ear and mastoid. Arch Otolaryngol. 1945;41:64.
- 7. Pellitteri PK, Rinaldo A, Myssiorek D, et al. Paragangliomas of the head and neck. Oral Oncol. 2004;40:563–75.
- Schwaber MK, Glasscock ME, Jackson CG, et al. Diagnosis and management of catecholamine secreting glomus tumors. Laryngoscope. 1984;94:1008.
- Schiavi A, Papangelou A, Mirski M. Preoperative preparation of the surgical patient with neurologic disease. Med Clin N Am. 2009;93:1123–30.
- Hepner DL. The role of testing in the preoperative evaluation. Cleve Clin J Med. 2009;76:S22–7.
- 11. Parsa P, Sweitzer B, Small SD. The contribution of a preoperative evaluation to patient safety in high-risk surgical patients: a pilot study (abstract). Anesth Analg. 2004;100:147.
- Hepner DL, Bader AM, Hurwitz S, et al. Patient satisfaction with preoperative assessment in a preoperative assessment testing clinic. Anesth Analg. 2004;98:1099–105.
- Parker BM, Tetzlaff JE, Litaker DL, Mauer WG. Redifining the preoperative evaluation process and the role of the anesthesiologist. J Clin Anesthe. 2000;12:350–6.
- Ferschl MB, Tung A, Sweitzer B, et al. Preoperative clinic visits reduce operating room cancellations and delays. Anesthesiology. 2005;103:855–9.
- Halaszynski TM, Juda R, Silverman DG. Optimizing postoperative outcomes with efficient preoperative assessment and managment. Crit Care Med. 2004;32(Suppl):S76–86.
- Sivanaser V, Manninen P. Preoperative assessment of adult patients for intracranial surgery. Anesthesiol Res Pract. 2010.
- 17. Khan ZH, Kashfi A, Ebrahimkhani E. A comparison of the upper lip bite test (a simple new technique) with modified Mallampati classification in predicting difficulty in endotracheal intubation: a prospective blinded study. Anesth Analg. 2003;96:595–9.
- Khan Z, Mohammadi M, Rasouli M, et al. The diagnostic value of the upper bite test combined with sternomental distance, thyromental distance, and interincisor distance for prediction of easy laryngoscopy and intubation: a prospective study. Anesthe Analg. 2009;109:822–4.
- Fleisher LA, Beckman JA, Brown KA, et al. ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for non-cardiac surgery: a report of the american college of cardiology/ american heart association task force on practice guidelines (writing committee to revise the 2002 guidelines on perioperative cardiovascular evaluation for non-cardiac surgery. Circulation. 2007;116:e418–500.
- 20. Kan P, Couldwell WT. Posterior fossa brain tumors and arterial hypertension. Neurosurg Rev. 2006;29:265–9.

- Weksler N, Klein M, Szendro G. The dilemma of immediate preoperative hypertension: to treat and operate or to postpone surgery? J Clin Anesth. 2003;15:179–83.
- 22. Frisch A, Chandra P, Smiley D, et al. Prevalence and clinical outcome of hyperglycemia in the perioperative in non-cardiac surgery. Diabetes Care. Published online April 30, 2010 [Publish Ahead of Print].
- Smiley DD, Umpierrez GE. Perioperative glucose control in the diabetic or non-diabetic patient. South Med J. 2006;99:580–9.
- Shelton C, Brackmann DE, House WF, Hitselberger WE. Middle fossa acoustic tumor surgery: results in 106 cases. Laryngoscope. 1989;99:405–8.
- Fisch U, Pillsbury HC. Infratemporal fossa approach to lesions in the temporal bone and base of the skull. Arch Otolaryngol. 1979;105:99–107.
- Weber PC, Patel S. Jugulotympanic paragangliomas. Otolaryngol Clin North Am. 2001;34:1231–40.
- Samii M, Gerganov V, Samii A. Hearing preservation after complete microsurgical removal in vestibular schwannomas. Prog Neurol Surg. 2008;21:136–41.
- Rath GP, Bithal PK, Chaturvedi A, Dash HH. Complications related to positioning in posterior fossa craniectomy. J Clin Neurosci. 2007;14:520–5.
- 29. Hitselberger WE, House WF. A warning regarding the sitting position for acoustic tumor surgery. Arch Otolaryngol. 1980;106:69.
- Suri A, Mahapatra AK, Bithal P. Seizures following posterior fossa surgery. Br J Neurosurg. 1998;12:41–4.
- Montgomery WW. Translabyrinthine resection of the small acoustic neuroma. Arch Otolaryngol. 1969;89:319–25.
- Hitselberger WE, House WF. Surgical approaches to acoustic tumors. Arch Otolaryngol. 1966;84:286–91.
- Silverstein H, Norrell H, Smouha E. Retrosigmoid-internal auditory canal approach vs. retrolabyrinthine approach for vestibular neurectomy. Otolaryngol Head Neck Surg. 1987;97:300–7.
- House WF, Hitselberger WE. The neuro-otologist's view of the surgical management of acoustic neuromas. Clin Neurosurg. 1985;32:214–22.
- House WF, Hitselberger WE. The transcochlear approach to the skull base. Arch Otolaryngol. 1976;102:334–42.
- Canalis RF, Black K, Martin N, Becker D. Extended retrolabyrinthine transtentorial approach to petroclival lesions. Laryngoscope. 1991;101(1 Pt 1):6–13.
- Smouha EE. Facial nerve monitoring and stimulation during surgery for chronic ear disease. Oper Tech Otolaryngol. 1992;3:43–7.
- Niparko JK, Kileny PR, Kemink JL, et al. Neurophysiologic intraoperative monitoring: II facial nerve function. Am J Otol. 1989;10:55–61.
- Prass RL, Lüders H. Acoustic (loudspeaker) facial electromyographic monitoring: Part 1. Evoked electromyographic activity during acoustic neuroma resection. Neurosurgery. 1986;19:392–400.

- Kartush JM, Niparko JK, Bledsoe SC, Graham MD, Kemink JL. Intraoperative facial nerve monitoring: a comparison of stimulating electrodes. Laryngoscope. 1985;95:1536–40.
- 41. Møller AR. Monitoring auditory function during operations to remove acoustic tumors. Am J Otol. 1996;17:452–60.
- Danner C, Mastrodimos B, Cueva RA. A comparison of direct eighth nerve monitoring and auditory brainstem response in hearing preservation surgery for vestibular schwannoma. Otol Neurotol. 2004;25:826–32.
- Colletti V, Fiorino FG, Carner M, Cumer G, Giarbini N, Sacchetto L. Intraoperative monitoring for hearing preservation and restoration in acoustic neuroma surgery. Skull Base Surg. 2000;10:187–95.
- Matthies C, Samii M. Management of vestibular schwannomas (acoustic neuromas): the value of neurophysiology for intraoperative monitoring of auditory function in 200 cases. Neurosurgery. 1997;40:459–66.
- 45. Fathi AR, Eshtehardi P, Meier B. Patent foramen ovale and neurosurgery in sitting position: a systematic review. Br J Anaesth. 2009;102:588–96.
- 46. Sloan T. The incidence, volume, absorption and timing of supratentorial pneumocephalus during posterior fossa neurosurgery conducted in the sitting position. J Neurosurg Anesthesiol. 2010;22:59–66.
- Schubert A, Deogaonkar A, Drummond JC. Precordial doppler probe placement for optimal detection for venous air embolism during craniotomy. Anesth Analg. 2006;102:1543–7.
- Watanabe E, Schramm J, Strauss C, Fahlbusch R. Neurophysiologic monitoring in posterior fossa surgery II. BAEP-waves I and V and preservation of hearing. Acta Neurochir. 1989;98(3–4):118–28.
- Kombos T, Suess O, Kern BC, et al. Can continuous intraoperative facial electromyography predict facial nerve function following cerebellopontine angle surgery. Neurol Med Chir (Tokyo). 2000;40:501–7.
- Kang DZ, Wu ZY, Lan Q, et al. Combined monitoring of evoked potentials during microsurgery for lesions adjacent to the brainstem and intracranial aneurysms. Chin Med J. 2007;120:1567–73.
- Engelhard K, Werner C. International or Intravenous Anestretic for craniotomes? Pro-international. Curn Open Anaestresiol 2006; 19;504–8.
- Cole CD, Gottifred ON, Gupta DK, Gonldwell WT. Total Intravenous Anestretic. Advantages for International Surgery. Neurosurgery 2007;61:369–78.
- Weiskopf RB, Kramer JH, Viele M, et al. Acute severe isovolemic anemia impairs cognitive function and memory in humans. Anesthesiology. 2000;2000:1646–52.
- Tommasino C. Fluids and the neurosurgical patient. Anesthesiol Clin N Am. 2002;20:329–46.
- Batoz H, Verdonck O, Pollerin C et al. The analges is properties of scalp infiltions with is provider after intracranial tumor resection. Anesth Analg 2009;109:240–61.

Neurosurgical Otolarynology II: Endoscopic Skull Base and Pituitary Surgery

Stacie G. Deiner, Kalmon D. Post, and Satish Govindaraj

Introduction

Successful endoscopic surgery of the skull base requires a rigorous understanding and comfort level with the surgical anatomy of the paranasal sinuses and adjacent vital structures including the orbit, optic nerve, carotid artery, cavernous sinus, and meninges. Since these structures are often protected by a thin barrier of mucosa-covered bone that may be violated by inflammation, neoplasm, or trauma, every case requires comprehensive presurgical imaging and planning. Anesthesiologists must also respect these unique anatomic relationships, as a surgery that is "going well" may quickly turn difficult in an area where the margin for error is small. Continued and effective communication between anesthesiology and surgical teams throughout the perioperative period helps ensure a safe and efficient surgical experience for the patient.

Overview

The base of the skull is made up of five bones including the ethmoid, sphenoid, occipital, paired frontal, and paired parietal bones and forms the floor of the cranial cavity that separates the brain from other facial structures (Fig. 18.1). The trans-nasal endoscopic surgical approach to the skull base has become popular amongst neurosurgeons and otolaryn-gologists. This approach, which used to be reserved

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for pituitary and sella turcica lesions (Fig. 18.2), has been successfully employed to manage lesions extending ventrally, laterally including the infratemporal fossa and the petrous apex, and the orbit ^[1, 2].

Advanced transnasal surgical techniques that include image-guided triplanar navigation, microdebriders, and suction irrigating drills have allowed the development of minimally invasive approaches to lesions that normally would require a craniotomy. Changes in surgical techniques have naturally resulted in unique anesthetic considerations required to create a surgical environment that allows adequate visualization while at the same time maintaining the principles of neurosurgical anesthesia. The focus of the anesthetic management has shifted from intraoperative management of blood loss, venous air embolism, and other catastrophic events to optimization of surgical conditions that afford superb visualization. Intraoperatively, endoscopic techniques require that the anesthesiologist understands the planned approach to facilitate a bloodless field. If neuromonitoring is required, the anesthesiologist must also provide an anesthetic conducive to accurate monitoring. Finally, the procedure often requires the patient to be positioned with the head away from the anesthesia machine, and often with arms tucked at the patient's side. Limited access to the patient during the duration of the procedure means that the anesthesiologist must anticipate appropriate venous access and monitoring prior to the surgical start. While surgery for skull base tumors has become less invasive, sudden catastrophic events can still occur (e.g., carotid artery injury), and it is clear that a complete understanding of the procedure is required by the anesthesiologist to ensure a safe and smooth perioperative course.

The transnasal approach to the skull base has evolved from the endoscopic sinonasal surgery performed by otolaryngologists and the transsphenoidal approach to the pituitary performed by neurosurgeons utilizing the operative microscope to encompass a broad range of surgical procedures. Currently the most common endoscopic skull base surgeries performed include tumor biopsy, excision of both benign and malignant neoplasms, orbital and optic nerve decompressions, CSF leak repairs, and pituitary surgery. These procedures and their anesthetic requirements will form the scope of this chapter.

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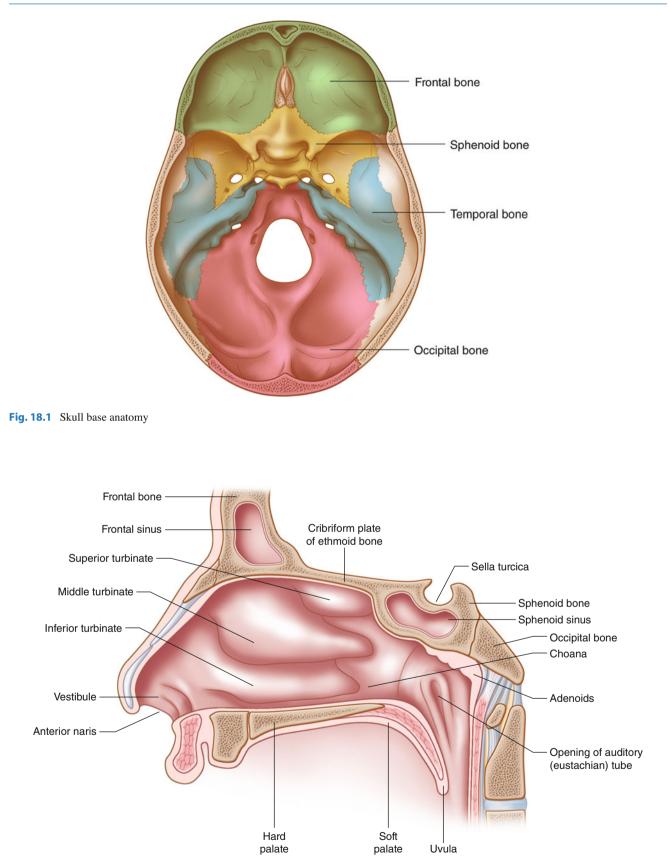


Fig. 18.2 Sella turcica and paranasal anatomy

Preoperative Considerations

Otolaryngologist's Perspective

As mentioned above the main endoscopic skull base surgeries performed are tumor biopsy, excision of both benign and select malignant neoplasms, orbital and optic nerve decompressions, CSF leak repairs, and pituitary surgery. In the case of skull base biopsies, lesions may be inflammatory such as granulomatous disease, pseudotumor, or Tolosa Hunt Syndrome ^[3, 4]; infectious such as invasive fungal sinusitis ^[5, 6]; or neoplastic. Certain neoplastic lesions such as lymphoma do not require surgery for treatment. Depending on the extent of the disease process and its location, these patients may present with preoperative cranial nerve palsies, orbital entrapment, diplopia or impaired vision due to invasion of the cavernous sinus and orbital apex. At times, there is extensive involvement of surrounding vital structures and imaging is not able to confirm a diagnosis.

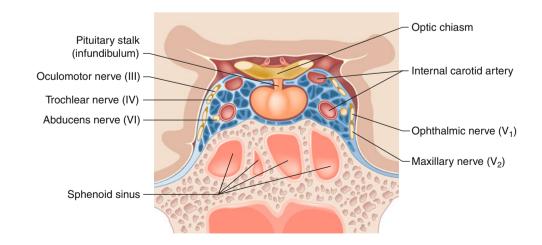
Because this region contains many critical structures especially in and around the cavernous sinus (Fig. 18.3), the clinical presentation in these patients depends on the area of involvement. Lesions may extend from an intracranial location into the paranasal sinuses such as meningiomas or pituitary lesions. Tumors that involve the sella may cause pituitary hypofunction or hyperfunction with variable presentations such as hypothyroidism, loss of libido, acromegaly, menstrual irregularity, or lactation, to name a few. The proximity of the optic chiasm to the pituitary gland may cause visual field defects or classically bitemporal hemianopsia (Fig. 18.4). Cranial nerve dysfunction is also seen in tumors that involve the skull base. Some examples are diplopia secondary to cranial nerves III, IV, and VI traveling through the cavernous sinus; jaw numbness due to involvement of the mandibular division of the trigeminal nerve as it exits the foramen ovale;

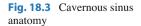
or aspiration or dysphagia secondary to cranial nerve IX and X compression at the jugular foramen.

Lesions that involve the orbital apex, infratemporal fossa, petrous apex, clivus, and sellar/parasellar regions are ideal for an endoscopic biopsy. In these cases if there is an access point via the paranasal sinuses, usually the sphenoid sinus, an endoscopic procedure is an ideal surgical option to obtain tissue for diagnosis. If the diagnosis requires only medical treatment, these surgeries may entail only removing a sample of tissue for pathologic examination or in some cases involve extensive surgical resection if negative margins are possible while preserving vital structures.

Cerebrospinal fluid (CSF) leaks and encephaloceles are now managed almost exclusively through an endoscopic approach except for select cases [7-12]. Patients commonly present with clear rhinorrhea that increases with coughing or Valsalva maneuver. CSF leaks may be posttraumatic, postsurgical, or spontaneous. Although 80% of posttraumatic leaks will heal with conservative management such as bed rest and lumbar drainage, there is a 30% long-term risk of ascending meningitis ^[13, 14]. The patient with a spontaneous CSF leak is considered a separate entity and believed to be a variant of benign intracranial hypertension ^[10, 15, 16]. It is imperative that the underlying pressure elevation be managed either medically with acetazolamide or surgically with a ventriculoperitoneal (VP) shunt; otherwise, the risk of repair failure or a new CSF leak is ongoing. There is no clear intracranial pressure level where a VP shunt is recommended in the literature with respect to the management of CSF leaks. At our institution, if the CSF pressure is above 30 cm H₂O, a shunt procedure is recommended.

Pituitary tumors and sellar lesions such as Rathke cleft cysts or craniopharyngiomas may present with headaches or visual field defects due to silent growth and pressure on the optic apparatus. Pituitary dysfunction may be seen as well, as mentioned earlier. Preoperative imaging is imperative in





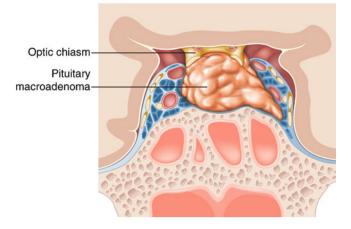


Fig. 18.4 Pituitary macroadenoma compressing the optic chiasm and extending into the left cavernous sinus

all of these lesions so as to determine the extent of surrounding tissue involvement and to plan a surgical approach.

Medical management and evaluation of these patients prior to surgery focuses on obtaining appropriate laboratory investigations and imaging before the surgery so the anesthesiologists can review these data. A full panel including a complete blood count and comprehensive metabolic panel are necessary at a minimum. Also, depending on the age of the patient or comorbid status, further workup may be involved as these surgeries are not without risk. If there is any concern that the patient poses a significant risk for surgery or may be challenging to manage intraoperatively, it is recommended that the otolaryngologists obtain an official preoperative anesthesiology consult.

Anesthesiologist's Perspective

Perhaps the single most important consideration guiding appropriate preoperative evaluation of the patient for endoscopic skull base surgery is an understanding of the surgical procedure and tumor type. The surgical procedures vary in terms of neurovascular structures at risk, operative time, and potential for conversion to open surgery. Once the anesthesiologist understands the nature of the tumor and the planned procedure, preoperative evaluation can be focused on patient risk factors. We will examine these in a systems-focused manner.

General

Skull base tumors are generally benign tumors that cause compression of surrounding structures or invasion of adjacent tissue. Due to the proximity of these tumors to cranial nerves, optic chiasm, and pituitary gland, a variety of symptoms may be reported. In addition secondary brain edema and hydrocephalus may be present.

Table 18.1 Possible symptoms of skull base tumors

- Headache, usually worse in the morning
- Vomiting
- Drowsiness
- First-time seizures
- Loss of sensation in an arm or leg
- Difficulty with balance and coordination
- Dizziness
- Vision loss or double vision
- Hearing loss
- Tinnitus (ringing in the ears)
- Speech difficulties
- Hoarseness
- Difficulty swallowing
- Facial pain, twitching, or paralysis and
- Change in personality

All patients presenting with skull base tumor resections need a thorough history and physical to determine the presence of raised intracranial pressure. The presence of raised intracranial pressure will have major implications on the management of pre-sedation, induction, and intraoperative management. Worrisome symptoms include: headache (often worse in the morning), vomiting, drowsiness, seizures, focal deficit in an extremity, difficulty with balance, changes in vision, auditory loss, speech difficulties, bulbar symptoms, facial pain, facial paralysis, or personality changes. Pituitary tumors may cause symptoms both by mass effect on surrounding tissue and by secretion of hormones. A pituitary tumor may increase ICP either directly from the tumor itself or indirectly from its obstruction of the third ventricle resulting in hydrocephalus (Table 18.1) ^[17].

Endocrine/Laboratory Evaluation

All patients should undergo preoperative laboratory evaluation before surgery. The benign skull base tumors may cause laboratory abnormalities when they are associated with compression of other structures. Pituitary adenomas may also be "functioning" tumors of a single cell type, producing hormones (Table 18.2). Generally these tumors present with the symptoms of anterior pituitary hormone excess^[17].

A complete blood count should be obtained. This is particularly relevant for men with pituitary tumors and low testosterone, which is associated with an increased risk of anemia ^[18]. Coagulation studies are indicated in patients with other medical or pharmacologic reasons for coagulopathy. Metabolic panels are an inexpensive, efficient way to evaluate possible posterior pituitary dysfunction and the presence of diabetes insipidus secondary to ADH deficiency. Hypercalcemia would alert the clinician to the possible

Table 18.2	Pituitary tumor types and incidences
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Hormone secreted	Percentage of all tumors
Prolactin	40-45
ACTH	10–12
GH	20
Gonadotropins	15
Null cell	5–10
TSH	1–2

diagnosis of multiple endocrine neoplasia, type I (MEN I). The endocrine evaluation of each patient should include an evaluation for anterior and posterior pituitary dysfunction: thyroid panel [thyroxine, thyroid-stimulating hormone (TSH)], and serum levels of cortisol, adrenocorticotropic hormone, insulin-like growth factor-1, testosterone, luteinizing hormone, follicle-stimulating hormone, [alpha] subunit, and prolactin.

However, of note the most common cause of secondary amenorrhea should always be investigated with a pregnancy test. Compression of the pituitary may result in panhypopituitarism. These patients should receive hormone replacement therapy with hydrocortisone and/or thyroxine, and evaluated for efficacy by laboratory studies. Additional stress doses of steroids (i.e., 100 mg hydrocortisone IV) may be necessary in the perioperative period; however, not all patients with skull base tumors should receive steroids since a subgroup of patients with functional adenomas will have excessive cortisol production.

Cardiac Evaluation

Cardiovascular evaluation should be based on the patient's symptoms, exercise tolerance, medical history, and tumor type. In general, the 2007 ACC/AHA algorithm for perioperative evaluation of the patient for noncardiac surgery is an appropriate guide ^[19]. A complete discussion of the approach to cardiovascular disorders can be found in the preoperative care chapter (Chap. 7). According to the algorithm most patients will be low to moderate risk, and most endoscopic skull base procedures are intermediate risk except in certain cases where there is risk to the carotid artery or cerebral vasculature. Further consideration may be warranted in patients with functional adenomas resulting in acromegaly (Fig. 18.5) or Cushing's disease (Fig. 18.6). Both of these diseases predispose toward structural and/or electrical cardiac pathophysiology.

Cardiac disease is the most frequent cause of death in patients with acromegaly, with 50% of patients dying before age 50^[20]. Acromegaly is associated with systemic hypertension, left ventricular hypertrophy, and diastolic dysfunction^[21].



Fig. 18.5 Patient with acromegalic facial features

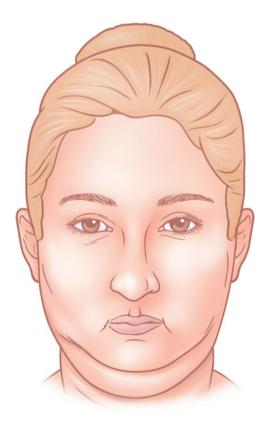


Fig. 18.6 Patient with Cushing's syndrome facial features

The latter can be one of the earlier signs of acromegalic cardiomyopathy ^[22]. While the ventricular hypertrophy may improve with treatment of the acromegaly, the diastolic dysfunction is related to myocardial fibrosis and does not resolve ^[23, 24]. Small vessel coronary artery disease may be present, and any signs of angina even in younger patients should be thoroughly evaluated ^[25]. Conduction defects are seen in approximately 50% of patients and may lead to cardiac rhythm disturbances, bundle branch blocks, and ECG changes including T wave abnormalities ^[26].

The majority of patients with Cushing's disease have systolic and/or diastolic hypertension [27]. The etiology of this hypertension is increased endogenous corticosteroids which can increase cardiac output as well as the hepatic production of angiotensinogen resulting in an increase in plasma volume. Additionally, corticosteroids increase the influx of sodium in vascular smooth muscle. Glucocorticoid inhibition of phospholipase A2 causes a reduction in the synthesis of vasodilator prostaglandins increasing the tone of vascular smooth muscle ^[28]. Inositol triphosphate production in vascular smooth muscle leads to increased sensitivity to vasoconstrictors (e.g., angiotensin I). The increased vasoconstriction and decreased modulation of vasodilation may result in septal and/or left ventricular hypertrophy. ECG abnormalities are common in patients with Cushing's disease including those indicative of left ventricular hypertrophy and left ventricular strain. Both electrical and functional changes tend to regress after tumor resection ^[29].

Certainly all patients with acromegaly and Cushing's disease should have preoperative electrocardiograms. Acromegalic patients should have an echocardiogram, and if they have symptoms of dysrhythmias or chest pain, further evaluation with Holter monitoring and a stress test may be warranted.

Respiratory Evaluation

Similar to cardiac considerations above, the evaluation of the respiratory system of the patient for skull base tumor surgery is identical to the evaluation of a patient for general surgery, except in the case of patients with functional adenomas resulting in acromegaly or Cushing's disease.

The second most common cause of death in acromegalic patients is respiratory disease ^[30]. Respiratory tract changes include: hypertrophy of the soft tissue of the nose, mouth, tongue, lips, pharynx, and larynx causing reduction in the size of the glottic opening, hypertrophy of the periepiglottic folds, calcinosis of the larynx, and recurrent laryngeal nerve injury. Patients with acromegaly or Cushing's disease may be difficult to bag-mask ventilate and intubate. Mallampati classification may yield a significant number of

false negatives in acromegalic patients ^[31]; therefore, clinical suspicion should be high and the threshold to perform awake intubation, low.

A recent study found that more than 80% of acromegalic patients have sleep apnea ^[32]. In the presence of diagnosed obstructive sleep apnea plans should be made for appropriate postoperative monitoring. Hoarseness should alert the physician to the possibility of laryngeal stenosis or recurrent laryngeal nerve injury ^[33]. Patients with laryngeal nerve injury may be predisposed to aspiration. Rapid sequence induction and extubation after the patient fully regains consciousness is prudent. Any combination of these issues may contribute to perioperative respiratory complications.

Intraoperative Considerations

Otolaryngologist's Perspective

The importance of the surgical field achieved through the appropriate anesthetic technique and adequate control of intraoperative blood pressure and heart rate cannot be overemphasized in the setting of endoscopic skull base surgery. The surgeon is operating in an area where visualization is of paramount importance. In open surgery, palpation of the tissue and depth perception are added surgical tools to enhance safety during the procedure. These two valuable adjuncts, however, are not available in endoscopic procedures. A surgeon must rely on his vision as well as an acquired skill of palpation via surgical instruments to determine safety and proximity to critical structures.

There are three main procedures that exemplify intraoperative management of skull base surgery: CSF leak/encephalocele repair, pituitary surgery and extended parasellar approaches, and anterior cranial fossa tumor resection. These three areas demonstrate the key concepts in skull base surgery: (1) approach and resection of the pathology, whether it be a tumor or an encephalocele; (2) reconstruction of the skull base defect with a free graft or vascularized tissue; and lastly (3) return to a normal functioning sinonasal cavity.

Paranasal sinus CSF leaks or encephaloceles most commonly involve the sphenoid and ethmoid regions ^[34]. The endoscopic approach for repair of these leaks has now evolved to become the standard of care for the initial attempt at closure ^[34]. The approach to this area is similar to a functional endoscopic sinus procedure. The anterior cranial fossa lies above the frontal and ethmoid sinuses, thus these areas must be resected completely in order to expose the skull base and location of the defect. We incorporate the use of dilute intrathecal fluorescein to help localize the site of the leak.

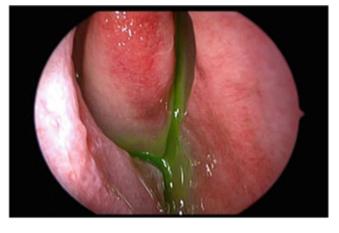


Fig. 18.7 Fluorescein can be seen draining from a cribriform plate cerebrospinal fluid leak into the nasal cavity. The fluorescein is used to localize the site of leak and also to confirm adequate closure

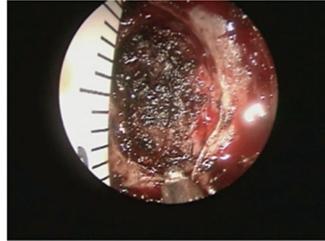


Fig. 18.8 An encephalocele has been reduced to the skull base defect and the mucosa around the edges of the defect has been removed to prepare the site for graft placement

It should be noted that fluorescein is not FDA approved for intrathecal use and the patient must be informed of this fact. The safety profile of the present recommended dilution, however, has been demonstrated in the literature [35, 36]. A lumbar drain is placed at the beginning of the procedure; 0.1 mL of 10% fluorescein solution is diluted in 10 mL of the patient's own CSF obtained during lumber drain placement. This dilute fluorescein is injected through the drain slowly over 30 min with the patient in a head down position. It is critical to place topical pledgets with decongestant in the nasal cavity in order to limit vascular engorgement of the sinonasal tissue. The fluorescein is used to help localize the site of leak during the endoscopic procedure as well as to confirm an adequate closure of the defect (Fig. 18.7). One will continue to see fluorescein-stained rhinorrhea around the repair if complete closure is not achieved.

If an encephalocele is present, it is reduced with bipolar cautery flush with the skull base defect (Fig. 18.8). This is critical in order to allow placement of an underlay and overlay graft. In addition the mucosa around the perimeter of the defect is removed in order to expose the underlying bone. If feasible, an underlay graft is placed in defects larger than 5 mm in size to avoid risk of herniation of intracranial contents. An overlay graft is then placed over the defect and stabilized with fibrin glue and nasal packing. There are multiple options for graft placement ranging from autologous tissues such as, septal or turbinate mucosa and bone, fascia lata, and temporalis fascia to allografts such as Alloderm® (LifeCell Corporation, Branchburg, NJ) or Duragen[®] (Integra LifeSciences Corporation, Plainsboro, NJ). Mucosa should never be placed as an underlay graft as this creates the risk of intracranial mucocele formation [37].

Pituitary surgery has undergone multiple evolutions over the past century due to advances in intraoperative technology ^[38].

It has evolved from its initial descriptions by Cushing with the use of a headlight to the present purely endoscopic or endoscopic-assisted technique [38-40]. The use of a microscope provides the surgeon with valuable depth perception in an area with little margin for error; however, there are limitations with respect to visualization around the perimeter of the sella that the endoscope is able to address. The use of an endoscope provides a panoramic view as well as large visual angles ranging from 0° to 70°. This has created the ability to examine around corners and access extra-sellar lesions. Endoscopes are not without their limitations. Most notable is the absence of depth perception and 3D imaging. During tumor removal and while working in close proximity to critical structures such as the carotid artery, it is imperative to work in a two-surgeon four-handed technique, using a purely endoscopic technique.

For lesions limited to the sella, a microscopic or endoscopic approach will result in equal success. In this setting, with limited tumor extension, the microscope provides an adequate field of vision and in some cases may be superior to the endoscope since it provides the added benefit of depth perception. When using a microscope, a posterior vertical incision is made at the bony-cartilaginous junction of the nasal septum and resection of the bony septum to the face of the sphenoid sinus. The septum between both sphenoid sinuses is resected which results in exposure of the sella turcica. A speculum is used to expand the field and allow a bimanual technique and the passage of instruments into and out of the nose. In contrast, the endoscopic technique requires more dissection; however, this results in a wider field of vision and greater range of motion for instrument dissection that is critical for larger, more extensive tumors.

The reconstruction of the skull base after pituitary and extended transsphenoidal surgery is performed in multiple ways.

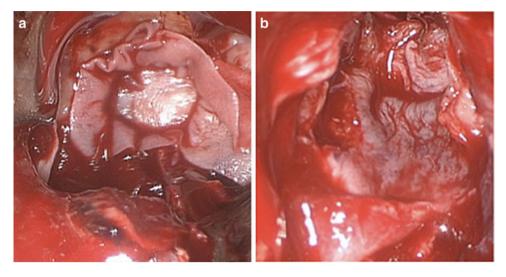


Fig. 18.9 (a) The first layer of closure is a combination bone and Alloderm graft that seals the skull base defect. (b) A nasoseptal flap that was harvested as part of the approach is rotated into the sphenoid sinus

and covers the initial layer of closure. Packing is placed to support the graft and is left in place for 5 days

A CSF leak is not always present in these cases; however, the concepts of reconstruction are the same. We favor a combination underlay–overlay technique with Alloderm[®] and septal bone, and if there is a CSF leak present, a posteriorly based nasoseptal flap is placed over this repair (Fig. 18.9). The nasoseptal flap is a vascularized flap within the nasal cavity that is based on the blood supply of the posterior septal branch of the sphenopalatine artery.

Anterior cranial fossa surgery for resection of benign and malignant lesions requires complete clearance and opening of the maxillary, frontal, sphenoid, and ethmoid sinuses. Once this is done, one or two nasoseptal flaps may be raised from the nasal septum with resection of the bony septum up to the skull base. Once this has been done the roof of the nasal cavity is removed exposing the underlying dura and tumor. The anterior and posterior ethmoid arteries will need to be controlled and cauterized during this approach in order to avoid the risk of intra-orbital bleeding. Reconstruction of a defect here is a challenge. If dural edges are present a sutured transnasal graft can be used (Fig. 18.10). In general, a combination of underlay and overlay techniques is used with fascia lata or Alloderm as the underlay graft and a large nasoseptal flap as the ideal overlay choice. After the repair is completed, nasal packing is placed to support the repair and kept in place for 5–7 days. This is usually removed in the office.

Maintenance of a functional sinonasal cavity after limited or extensive skull base resections is paramount. Inadequate attention to the sinuses during the resection will result in postoperative infection, compromise to the reconstruction, and long-term chronic sinusitis for the patient. The sinuses surrounding the area of resection are opened and stents placed to maintain patency especially in the case of the



Fig. 18.10 An Alloderm graft is being sutured in place with U clip sutures. A nasoseptal flap is placed over this closure and secured with tissue sealant

frontal sinus where long-term frontal recess stenosis and mucocele formation is a risk. In the case of pituitary surgery, the sphenoid sinus is the only sinus traversed and an adequate sinusotomy is created during the procedure to avoid longterm sphenoid sinus obstruction. Lastly, in addition to the sinuses, intranasal synechiae may develop and for this reason silastic splints are sometimes placed to avoid synechiae between the lateral nasal wall and septum. These are also removed in the outpatient setting along with the nasal packing.

Although rare (i.e., approximately 1% of cases), internal carotid artery (ICA) injury can be a life-threatening intraoperative complication. Primary management is surgical and

involves the placement of packing, possible endovascular embolization or ligation of the ICA while a primary repair or graft is placed. It is critical once recognized that this complication is communicated to the anesthesiology team as they may choose to manipulate the hemodynamics to either decrease hemorrhage (i.e., controlled hypotensive techniques) or maintain adequate intracranial perfusion (i.e., raise the blood pressure) once the bleeding is under control. Further, they can assure adequate intravenous access, invasive blood pressure monitoring, blood product administration, and potentially prepare for transport to an interventional radiologic suite for fluoroscopically guided embolization where appropriate.

Anesthesiologist's Perspective

Induction

In the operating room the patient is positioned for intubation in the supine position on the operating table. In addition to standard ASA monitors, patients with increased intracranial pressure or significant heart disease secondary to their tumor or underlying comorbidities may require monitoring of arterial blood pressure prior to induction. Often, the final position of the patient's airway will be away from the machine, so an expandable breathing circuit, extensions on the intravenous lines, and sufficiently long cables for the monitors may be required. While two experienced anesthesia providers may choose to induce the patient with the airway at a distance from the machine, a single provider or patient with a difficult airway should be induced within reach of the anesthesia machine and equipment cart.

If a difficult airway is anticipated, then consideration should be given to an awake intubation, especially if the patient may be difficult to ventilate. Certainly in the case of the patient with acromegaly, appropriately large oral airways, LMAs, a second provider, and backup intubation devices (e.g., fiberoptic bronchoscope) should be readily available ^[41]. In anticipation of subglottic stenosis, a range of endotracheal tube sizes should also be present. Standard endotracheal tubes are generally adequate, although some providers prefer oral RAE tubes for their low profile or armored tubes because of their resistance to kinking.

The choice of induction agent is determined by the presence of cardiac disease, increased intracranial pressure, likelihood of difficult airway and whether the patient has a full stomach. With an understanding of the patient's issues the anesthesiologist performs a risk/benefit analysis and chooses the most appropriate plan. Maintenance of the airway is always the most important of these considerations. As such, awake techniques may be indicated. Topicalization of the airway in a patient with a full stomach may be risky, however, as coughing and straining could worsen intracranial pressure.

Propofol may be the appropriate induction agent for patients with a preserved ejection fraction. Etomidate may be chosen for patients with poor cardiac reserve. Ketamine should generally be avoided as it may raise intracranial pressure.

The selection of a paralytic drug is dependent on the patient's risk for aspiration, whether they have increased intracranial pressure, and plans for intraoperative neuromonitoring. Administration of succinvlcholine may worsen increased intracranial pressure and defasiculating doses of nondepolarizing agents do not block this phenomenon ^[42]. However, it has not been shown that the use of succinvlcholine has resulted in brain herniation [43]. Therefore, the anesthesiologist must weigh the indication for rapid sequence against the theoretical risk of aspiration and choose the most prudent plan. Prior to the use of nondepolarizing neuromuscular blockade the anesthesiologist should confirm the intraoperative neuromonitoring plan. The use of facial nerve monitoring, EMG, or motor evoked potentials is not compatible with intraoperative paralysis. However, many procedures may have an initial prolonged phase of preparation and surgical start prior to monitoring. If this is the case, then an initial dose of intermediate acting paralytic may be appropriate so long as it is not re-dosed.

If intracranial hypertension is suspected or a real risk for the patient, premedication should be approached cautiously as it may result in hypoventilation and hypercarbia leading to increased intracranial pressure.

Maintenance

The intraoperative goal of the anesthesiologist is to provide an amnestic, immobile patient with stable hemodynamics. Surgical considerations include facilitation of a bloodless field (discussed in detail in Chap. 13) by a combination of lower blood pressure, avoidance of vasodilators, and the use of vasoconstrictors and nerve blocks.

Maintenance of anesthesia may be achieved with inhalational agents, intravenous agents, or both. Inhalational agents cause vasodilation and may contribute to venous oozing. While inhalational agents are not contraindicated, supplementation with intravenous agents including propofol, narcotics, or dexmedetomidine may be helpful. Whichever technique is chosen, the end goal is rapid emergence, extubation, and early neurologic evaluation. Therefore, the duration of the procedure and kinetics of the drugs must be kept in mind.

If the patient is to awaken in a timely fashion at the end of the procedure it is extremely important that the anesthesiologist is aware of the context sensitive half-life of the drugs they are using to maintain amnesia. Context sensitive halflife is the time for the plasma concentration to decrease by

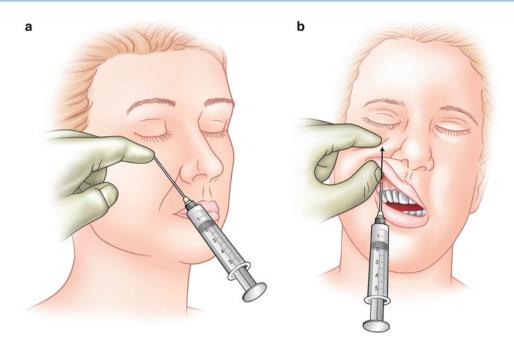


Fig. 18.11 Direct and transoral approach to infraorbital nerve. In both approaches, the anesthesiologist palpates the infraorbital foramen and directs the needle in that direction

50% from an infusion that maintains a constant concentration. Context refers to the duration of the infusion. Time to 50% decrease in plasma concentration was chosen because a 50% reduction in drug concentration roughly appears to be necessary for recovery after the administration of most intravenous hypnotics at the termination of surgery. During long surgeries this knowledge must be used to aggressively taper the intravenous anesthetic at the appropriate time, often 40 min prior to surgical finish in the case of propofol. During closure, inhalational gas can be added if necessary.

In general postoperative pain is mild to moderate following skull base surgery. Although counterintuitive, at least one study has shown that the presence of nasal packing is not associated with postoperative discomfort ^[44]. Therefore, the anesthesiologist should avoid large doses of long-acting narcotics for postoperative pain or to achieve blood pressure control during surgical stimulation. Blood pressure may be controlled with short-acting narcotics (remifentanil, alfentanil) or with antihypertensives. In fact, lingering respiratory depression from long-acting narcotics may serve to increase intracranial pressure in the postoperative period.

Blood Pressure Management

A detailed discussion of blood pressure management for endoscopic surgery can be found in Chap. 13) and the same principles should be applied to endoscopic base of skull surgery. Acceptable intraoperative blood pressure is a function of the patient's preoperative blood pressure and relevant comorbidities. Obviously, patients with poorly controlled hypertension and known carotid or cerebrovascular disease are not appropriate candidates for deliberate hypotension. If these conditions are recognized preoperatively, then a discussion should be had with the surgeon regarding the ability to provide intraoperative hypotension to facilitate a bloodless field. In general, intra-arterial catheters are not mandatory. However, the real possibility for hemorrhage and potential rapid changes in blood pressure if the brain is violated lead many to place this monitoring modality in all patients.

Recent studies have suggested that nerve blocks may be useful adjuncts to inhalational agents during sinus surgery. Higashizawa et al. showed that infraorbital nerve blocks are effective in reducing halogenated agent consumption, the need for blood pressure control with antihypertensives, in patients undergoing endoscopic endonasal maxillary sinus surgery (ESS) under general anesthesia ^[45]. The block is performed by injecting a small (1.0 mL) amount of local anesthetic in the area of the infraorbital foramen (Fig. 18.11). The injection may be approached via the soft tissue that overlies the foramen, intraorally or via the nares.

Fluid Management

Skull-based tumors which are amenable to endoscopic resection generally are not associated with brisk blood loss. However, there is always the potential for blood loss secondary to surrounding vascular structures. An example is the potential for damage to the ICA during transphenoidal pituitary surgery. As mentioned above, although exceedingly rare (1%), trauma to the ICA during endoscopic base of skull surgery can be life threatening. The treatment of this complication is primarily surgical yet the anesthesiologist must be prepared to assist the surgical team in many ways. In high-risk cases (large tumors in close approximation to the artery) the anesthesiologist must be prepared to acutely lower the blood pressure (MAP of 40-60) with vasodilator agents (e.g., nitroglycerin or sodium nitroprusside) or anesthetic agents (e.g., remifentanil, propofol, inhaled anesthetics) to reduce the risk of exsanguination while providing the surgeon an operative field for possible detection and repair. If the carotid is to be ligated, clamped, or embolized and if the patient is at high risk for a cerebrovascular event, the blood pressure may in fact need to be raised. This can be accomplished by lightening the anesthetic or through appropriate vasoactive agents (e.g., phenylephrine, ephedrine).

More commonly, venous "oozing" observed, and for prolonged surgeries may be significant. However, intraoperative bleeding does tend to be more severe in patients with larger tumors and also with suprasellar extension ^[46]. In higher risk procedures, it is prudent to have access to an extremity for additional venous access. At least one 18 g IV should be placed to start these cases.

Maintenance fluids with an isotonic crystalloid are generally adequate for intraoperative fluid replacement. Generally, intraoperative osmotic diuresis is not necessary. Awareness of previously existing or developing diabetes insipidus is important and may be signaled by large volumes of dilute urine without other inciting factors. This would prompt evaluation of an electrolyte panel, which would show hypernatremia and dilute urine (specific gravity <1.005). Acute management of diabetes insipidus would include conservative treatment, or in the case of rising serum sodium and large discrepancy between fluid inputs and outputs, DDAVP ^[47]. However, endoscopic approaches are less likely to cause diabetes insipidus than traditional open craniotomy ^[17].

Emergence

While rapid emergence is desirable to facilitate an early neurologic exam, avoidance of coughing and straining is extremely important to avoid contributing to a CSF leak or worsened venous oozing. The anesthesiologist must produce a calm, but awake patient who breathes reliably after extubation. Unfortunately, deep extubation is generally not an option as access to the airway is limited, patients may have an additional tendency to obstruct and it is undesirable to administer positive pressure with a mask. Awake extubation may be accomplished in a variety of ways. Short-acting narcotics (remifentanil or alfentanil) may be given by low-level infusion (e.g., 0.02–0.08 mcg/kg/min for remifentanil) and then supplemented with judicious use of longer acting narcotics while the amnestic agents are allowed to dissipate. Dexmedetomidine infusion may be used for its ability to produce a calm patient with reliable respiratory drive. Notably, dexmedetomidine does not obviate the need for postoperative narcotics although it may reduce total requirements ^[48].

Postoperative nausea and vomiting are very common (23% of patients) in patients undergoing neurosurgical procedures ^[49]. In addition to patient discomfort, vomiting has detrimental effects on ICP and could potentially contribute to a CSF leak. Given this risk, routine prophylaxis seems reasonable. Common sense dictates that when rapid emergence and early neurologic examination of the patient is desirable, antiemetics associated with sedation (e.g., droperidol, scopolamine) should be avoided. Using the Consensus Guidelines for Postoperative Nausea and vomiting, neurosurgical patients should be considered high risk ^[50]. With consideration of their underlying medical problems treatment with two or more of the antiemetic agents with IIA evidence should be considered. In this author's practice, dexamethasone 8 mg and ondansetron 4 mg are commonly administered prior to incision.

Postoperative Considerations

Otolaryngologist's and Anesthesiologist's Perspectives

The level of postoperative care is dictated by the patient's potential for complications, which may warrant monitoring in the intensive care unit. Major surgical complications (1.5%) include CSF leak, meningitis, ischemic stroke, and vascular injury. Minor complications (6.5%) include sinus disease, septal perforation, epistaxis, wound infections, and hematomas. Fluid and electrolyte disturbances occur in the majority of patients undergoing transsphenoidal resection of pituitary adenomas and are usually transient. In a few patients, DI may persist and require therapy with ADH analogs. Most commonly, hyponatremia occurs 1 week postoperatively and resolves within 5 days, but may occur up to 10 days later ^[47]. For a full treatment of the medical management of disorders of water balance, the reader is referred to any of the recent excellent reviews including Nemergut et al. ^[17].

From a surgical standpoint, some patients with spontaneous CSF leaks may require postoperative lumbar drainage ^[10, 15, 16]. This is usually done for 24–48 h in order to address the likely elevation in intracranial pressure seen in this patient population. In addition, long-term acetazolamide is administered as a means of prevention of recurrent or de novo CSF leaks. Antibiotic prophylaxis is given to patients while nasal packing is in place in order to prevent a secondary sinusitis. After nasal packing is removed patients undergo endoscopic debridement of the nasal cavity to remove retained mucus and crusting. It is important to avoid the area of reconstruction in the early postoperative period. After 4 weeks, more aggressive debridement can be safely done to avoid synechiae development in the sinonasal cavity.

Conclusion

The endoscopic approach to skull-based tumor surgery has been a major advance in otolaryngology and neurosurgery. While catastrophic intraoperative problems like hemorrhage and venous air embolism have decreased, there remain significant anesthetic considerations. The anesthesiologist and surgeon should communicate regarding the patient's tumor, planned procedure, and intraoperative expectations. With this knowledge both physicians can create a care plan to achieve a smooth intra and postoperative course.

Clinical Insights

For the Otolaryngologist (from the Anesthesiologist)

- Communication between the surgeon and the anesthesiologist is critical for patient safety during endoscopic skull base tumor surgery. Prior to the procedure communication regarding the planned procedure, type, location, and size of the tumor is critical. This allows the anesthesiologist to obtain appropriate monitoring and venous access prior to surgical draping and arrange for availability of blood and blood products.
- The need for intraoperative hypotension should be communicated ahead of time. Agreement of the risk-benefit ratio of this technique is important prior to surgical start. Plans for intraoperative neuromonitoring will affect anesthetic choice as well.
- Finally, plans for postoperative neurologic evaluation and need for intensive care monitoring allows the anesthesiologist to time their anesthetic regimen and consult with the intensive care team regarding planned transfer of care.

For the Anesthesiologist (from the Otolaryngologist)

- The value of a clear surgical field is a critical component to the success of endoscopic skull base surgery. Adequate visualization permits safer and more efficient dissection during an endoscopic procedure.
- The sphenopalatine and infraorbital blocks are useful in endoscopic skull base surgery as they are in sinus surgery. The ability to offer these regional blocks is an asset.
- These patients will often have nasal packing in place and in light of the surgery having intracranial communication, nasal cannula oxygen should be contraindicated to avoid the risk of peumocephalus.
- Although it is ideal to have a deep extubation to avoid the risk of compromise to the skull base reconstruction, this benefit must be weighed against the risk of an oversedated patient that will require mask ventilation with subsequent positive pressure being applied to the reconstruction.

Clinical Pearls

For the Otolaryngologist (from the Otolaryngologist)

- Discuss with the anesthesiologist the possibility of a total intravenous anesthetic to improve surgical conditions. If there is no medical contraindication, this technique will facilitate surgery through hemodynamic means.
- A separate consent is needed for the use of intrathecal fluorescein and the surgeon must inform the patient that it is not FDA approved for intrathecal use.
- The use of an extended needle tip bovie is an ideal instrument to elevate the posterior nasoseptal flap. It provides a dry field and more precise incisions for the flap.
- The placement of pledgets in the nasal cavity while the patient is in a head down position during intrathecal fluorescein injection will decrease the vascular engorgement in the nose and bleeding once the surgery is begun.

For the Anesthesiologist (from the Anesthesiologist)

- Understanding the tumor type, location, and planned procedure is critical when developing an anesthetic plan. Ideally the information will be obtained by history and documented in the patient's medical record; however, a discussion with the surgeon is critical.
- Lowering the cardiac output instead of lowering the systemic vascular resistance improves surgical visualization by minimizing nasal and sinus mucosal bleeding.
- Increased intracranial pressure, anticipation of the need for venous access, invasive monitoring, and neuromonitoring will affect the plan substantially.
- Like endoscopic sinus surgery, the placement of sphenopalatine and infraorbital blocks will improve intraoperative conditions and reduce postoperative pain.
- In the case of longer surgeries and patients with endocrine manifestations the ability to obtain intraoperative blood sampling is important so the placement of an arterial line should be considered even if blood loss is likely to be minimal.
- Positioning and draping may preclude easy access to the patient once surgery is underway; therefore, preoperative planning facilitates a smooth intraoperative course.

References

- 1. Lee SC, Senior BA. Endoscopic skull base surgery. Clin Exp Otorhinolaryngol. 2008;1:53–62.
- Castelnuovo P, Dallan I, Battaglia P, Bignami M. Endoscopic endonasal skull base surgery: past, present and future. Eur Arch Otorhinolaryngol. 2010;267:649–63.
- Williamson RA, Paueksakon P, Coker NJ. Inflammatory pseudotumor of the temporal bone. Otol Neurotol. 2003;24:818–22.
- Garg V, Temin N, Hildenbrand P, Silverman M, Catalano PJ. Inflammatory pseudotumor of the skull base. Otolaryngol Head Neck Surg. 2010;142:129–31.
- Swift AC, Denning DW. Skull base osteitis following fungal sinusitis. J Laryngol Otol. 1998;112:92–7.
- Rassekh CH, Kinsella JB, Calhoun KH, Maggio WW, Chaljub G, Gourley WK. Skull base allergic fungal sinusitis with abducens palsy in the third trimester. Skull Base Surg. 1996;6:253–8.
- Lanza DC, O'Brien DA, Kennedy DW. Endoscopic repair of cerebrospinal fluid fistulae and encephaloceles. Laryngoscope. 1996; 106:1119–25.
- Mattox DE, Kennedy DW. Endoscopic management of cerebrospinal fluid leaks and cephaloceles. Laryngoscope. 1990;100: 857–62.

- Purkey MT, Woodworth BA, Hahn S, Palmer JN, Chiu AG. Endoscopic repair of supraorbital ethmoid cerebrospinal fluid leaks. ORL J Otorhinolaryngol Relat Spec. 2009;71:93–8.
- Schlosser RJ, Woodworth BA, Wilensky EM, Grady MS, Bolger WE. Spontaneous cerebrospinal fluid leaks: a variant of benign intracranial hypertension. Ann Otol Rhinol Laryngol. 2006;115:495–500.
- Woodworth BA, Palmer JN. Spontaneous cerebrospinal fluid leaks. Curr Opin Otolaryngol Head Neck Surg. 2009;17:59–65.
- Woodworth BA, Schlosser RJ, Palmer JN. Endoscopic repair of frontal sinus cerebrospinal fluid leaks. J Laryngol Otol. 2005;119:709–13.
- Bernal-Sprekelsen M, Alobid I, Mullol J, Trobat F, Tomas-Barberan M. Closure of cerebrospinal fluid leaks prevents ascending bacterial meningitis. Rhinology. 2005;43:277–81.
- Bernal-Sprekelsen M, Bleda-Vazquez C, Carrau RL. Ascending meningitis secondary to traumatic cerebrospinal fluid leaks. Am J Rhinol. 2000;14:257–9.
- Schlosser RJ, Wilensky EM, Grady MS, Bolger WE. Elevated intracranial pressures in spontaneous cerebrospinal fluid leaks. Am J Rhinol. 2003;17:191–5.
- Schlosser RJ, Bolger WE. Spontaneous nasal cerebrospinal fluid leaks and empty sella syndrome: a clinical association. Am J Rhinol. 2003;17:91–6.
- Nemergut EC, Dumont AS, Barry UT, Laws ER. Perioperative management of patients undergoing transsphenoidal pituitary surgery. Anesth Analg. 2005;101:1170–81.
- Ellegala DB, Alden TD, Couture DE, Vance ML, Maartens NF, Laws Jr ER. Anemia, testosterone, and pituitary adenoma in men. J Neurosurg. 2003;98:974–7.
- Fleisher LA, Beckman JA, Brown KA, et al. 2009 ACCF/AHA focused update on perioperative beta blockade incorporated into the ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: a report of the American college of cardiology foundation/American heart association task force on practice guidelines. Circulation. 2009;120: e169–276.
- Wrightson P, Rajasoorya C, Holdaway IM, Scott DJ. Acromegaly: factors affecting the long term outcome after surgical treatment. J Clin Neurosci. 1994;1:164–72.
- Lopez-Velasco R, Escobar-Morreale HF, Vega B, et al. Cardiac involvement in acromegaly: specific myocardiopathy or consequence of systemic hypertension? J Clin Endocrinol Metab. 1997;82:1047–53.
- Herrmann BL, Bruch C, Saller B, et al. Acromegaly: evidence for a direct relation between disease activity and cardiac dysfunction in patients without ventricular hypertrophy. Clin Endocrinol (Oxf). 2002;56:595–602.
- Lombardi G, Colao A, Marzullo P, Biondi B, Palmieri E, Fazio S. Improvement of left ventricular hypertrophy and arrhythmias after lanreotide-induced GH and IGF-I decrease in acromegaly. A prospective multi-center study. J Endocrinol Invest. 2002;25:971–6.
- Colao A, Cuocolo A, Marzullo P, et al. Is the acromegalic cardiomyopathy reversible? Effect of 5-year normalization of growth hormone and insulin-like growth factor I levels on cardiac performance. J Clin Endocrinol Metab. 2001;86:1551–7.
- 25. Lie JT. Pathology of the heart in acromegaly: anatomic findings in 27 autopsied patients. Am Heart J. 1980;100:41–52.
- 26. Colao A. The GH-IGF-I axis and the cardiovascular system: clinical implications. Clin Endocrinol (Oxf). 2008;69:347–58.
- Whitworth JA, Williamson PM, Mangos G, Kelly JJ. Cardiovascular consequences of cortisol excess. Vasc Health Risk Manag. 2005;1:291–9.
- Magiakou MA, Smyrnaki P, Chrousos GP. Hypertension in Cushing's syndrome. Best Pract Res Clin Endocrinol Metab. 2006;20:467–82.

- Pereira AM, Delgado V, Romijn JA, Smit JW, Bax JJ, Feelders RA. Cardiac dysfunction is reversed upon successful treatment of Cushing's syndrome. Eur J Endocrinol. 2010;162:331–40.
- Seidman PA, Kofke WA, Policare R, Young M. Anaesthetic complications of acromegaly. Br J Anaesth. 2000;84:179–82.
- Schmitt H, Buchfelder M, Radespiel-Troger M, Fahlbusch R. Difficult intubation in acromegalic patients: incidence and predictability. Anesthesiology. 2000;93:110–4.
- 32. van Haute FR, Taboada GF, Correa LL, et al. Prevalence of sleep apnea and metabolic abnormalities in patients with acromegaly and analysis of cephalometric parameters by magnetic resonance imaging. Eur J Endocrinol. 2008;158:459–65.
- Williams RG, Richards SH, Mills RG, Eccles R. Voice changes in acromegaly. Laryngoscope. 1994;104:484–7.
- Banks CA, Palmer JN, Chiu AG, O'Malley Jr BW, Woodworth BA, Kennedy DW. Endoscopic closure of CSF rhinorrhea: 193 cases over 21 years. Otolaryngol Head Neck Surg. 2009;140: 826–33.
- Placantonakis DG, Tabaee A, Anand VK, Hiltzik D, Schwartz TH. Safety of low-dose intrathecal fluorescein in endoscopic cranial base surgery. Neurosurgery. 2007;61:161–5 [discussion 5–6].
- Tabaee A, Placantonakis DG, Schwartz TH, Anand VK. Intrathecal fluorescein in endoscopic skull base surgery. Otolaryngol Head Neck Surg. 2007;137:316–20.
- Eloy JA, Fatterpekar GM, Bederson JB, Shohet MR. Intracranial mucocele: an unusual complication of cerebrospinal fluid leakage repair with middle turbinate mucosal graft. Otolaryngol Head Neck Surg. 2007;137:350–2.
- Gandhi CD, Christiano LD, Eloy JA, Prestigiacomo CJ, Post KD. The historical evolution of transsphenoidal surgery: facilitation by technological advances. Neurosurg Focus. 2009;27:E8.
- Landolt AM. History of pituitary surgery from the technical aspect. Neurosurg Clin N Am. 2001;12:37–44.
- Jankowski R, Auque J, Simon C, Marchal JC, Hepner H, Wayoff M. Endoscopic pituitary tumor surgery. Laryngoscope. 1992;102: 198–202.

- Nemergut EC, Zuo Z. Airway management in patients with pituitary disease: a review of 746 patients. J Neurosurg Anesthesiol. 2006;18:73–7.
- Bozeman WP, Idris AH. Intracranial pressure changes during rapid sequence intubation: a swine model. J Trauma. 2005;58: 278–83.
- 43. Clancy M, Halford S, Walls R, Murphy M. In patients with head injuries who undergo rapid sequence intubation using succinylcholine, does pretreatment with a competitive neuromuscular blocking agent improve outcome? A literature review. Emerg Med J. 2001;18:373–5.
- Friedman M, Venkatesan TK, Lang D, Caldarelli DD. Bupivacaine for postoperative analgesia following endoscopic sinus surgery. Laryngoscope. 1996;106:1382–5.
- 45. Higashizawa T, Koga Y. Effect of infraorbital nerve block under general anesthesia on consumption of isoflurane and postoperative pain in endoscopic endonasal maxillary sinus surgery. J Anesth. 2001;15:136–8.
- 46. Lee HW, Caldwell JE, Wilson CB, Dodson B, Howley J. Venous bleeding during transsphenoidal surgery: its association with preand intraoperative factors and with cavernous sinus and central venous pressures. Anesth Analg. 1997;84:545–50.
- 47. Kristof RA, Rother M, Neuloh G, Klingmuller D. Incidence, clinical manifestations, and course of water and electrolyte metabolism disturbances following transsphenoidal pituitary adenoma surgery: a prospective observational study. J Neurosurg. 2009;111:555–62.
- 48. Pascoe PJ, Raekallio M, Kuusela E, McKusick B, Granholm M. Changes in the minimum alveolar concentration of isoflurane and some cardiopulmonary measurements during three continuous infusion rates of dexmedetomidine in dogs. Vet Anaesth Analg. 2006;33:97–103.
- Manninen PH, Tan TK. Postoperative nausea and vomiting after craniotomy for tumor surgery: a comparison between awake craniotomy and general anesthesia. J Clin Anesth. 2002;14:279–83.
- Gan TJ, Meyer T, Apfel CC, et al. Consensus guidelines for managing postoperative nausea and vomiting. Anesth Analg. 2003;97: 62–71 [Table of contents].

Tracheotomy

Sharmin Haque, Adam I. Levine, Chan W. Park, and Kenneth W. Altman

Introduction

Tracheotomy is commonly performed electively, urgently, or emergently in order to provide a definitive and secure airway. Patients requiring tracheotomy or those with a preexisting tracheotomy generally have significant comorbidities, specifically of the cardiovascular and pulmonary systems, that place them at increased risk in the perioperative period compared to the general population. In addition, despite having a "definitive airway," patients with tracheotomies are still at risk of airway loss from device dislodgement or occlusion, compounded by a lack of familiarity with the postsurgical anatomy and/or the devices used for tracheotomy patency. Patients scheduled for tracheotomy placement and those with in situ tracheotomies present to the operating room frequently. Anesthesiologists must be knowledgeable of the tracheotomy procedure and the care of patients with tracheotomies and otolaryngologists must be knowledgeable of the impact of significant comorbidities on anesthetic management to assure optimal patient care and decrease perioperative morbidity and mortality. In this chapter we will review the surgical and anesthetic considerations for patients

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presenting for tracheotomy placement and those presenting to the operating room who already have a tracheotomy. A special section will review in detail tracheotomy appliances and furnish the anesthesia caregiver with a working knowledge of these devises. As noted throughout this text, consistent planning and good communication between the otolaryngologist and anesthesiologist is vital to successful outcomes.

Although the surgical care of the patient with tracheal stenosis will be covered in this chapter as well, the anesthetic management for patients presenting for evaluation and management of tracheal stenosis is covered in detail in Chap. 10.

Overview

Whether it is airway obstruction, trauma, or elective surgery, the establishment of a secure airway is of paramount importance and a critical component prior to the commencement of any surgical procedure. The otolaryngologist plays the primary role in clinical scenarios when a surgical airway such as a tracheotomy is necessary. The role of a surgical airway has been discussed for many years. The primary goal of a tracheotomy is to create and secure an artificial airway for the patient. The reasons for performing this procedure are several fold, but the major indications for tracheotomy are as follows: (1) bypass upper airway obstruction, (2) assist respirations over a prolonged period, (3) improved pulmonary toilet, (4) help manage aspiration, (5) promote weaning from the ventilator by reducing ventilator circuit airway resistance, and (6) reduce the likelihood of endotracheal tube complications such as subglottic stenosis [1, 2]. The decision to perform a tracheotomy can be at times very complex and the benefits of performing this procedure must be balanced by the risks (Table 19.1).

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Table 19.1 Indications for tracheotomy placement

- Inability to intubate
- · Inability to manage secretions
- Facilitation of ventilation support and airway protection (e.g., patients with laryngeal and bulbar dysfunction from severe brain or spinal cord injury or cerebrovascular accidents ^[2])
- Prolonged or expected prolonged intubation (e.g., patients with failed trials of extubation or weaning attempts)
- Adjunct to manage head and neck surgery (e.g., patients with free tissue transfer reconstruction of the oral cavity, larynx or pharynx for malignancy ^[1])
- Adjunct to manage head and neck trauma and to bypass upper airway obstruction (e.g., patients with laryngeal or subglottic stenosis, laryngeal nerve palsy, severe obstructive sleep apnea, angioedema^[2])

American Academy of Otolaryngology-Head and Neck Surgery in the Clinical Indicators Compendium^[3]

Absolute contraindications for tracheotomy are rare and include soft tissue infections of the neck and distorted anatomy, e.g., from prior major neck surgery. Relative contraindications include severe respiratory distress with refractory hypoxemia and hypercapnia ^[4]. Hematologic and coagulation disorders are sometimes considered as contraindications, but prior studies have shown that tracheostomies can be safely performed in thrombocytopenic and neutropenic patients after necessary transfusions ^[5–7].

Under most circumstances, the tracheotomy is performed electively. However, when an emergency surgical airway is required as in the "cannot intubate/cannot ventilate" scenario, a cricothyroidotomy or tracheotomy may be necessary (as seen in Fig. 19.1)^[8].

With every potential difficult airway, one must weigh the risks and benefits of performing such an invasive surgical airway access in lieu of intubation or to attempt either an awake or asleep intubation first. The management of the difficult airway is discussed in detail in Chap. 8.

The Timing of Elective Tracheotomy Placement

There is ongoing debate regarding the optimal time to perform an elective tracheotomy (early versus late) in the intubated patient. The difficulty in resolving this issue arises from the lack of a standard definition of "early" tracheotomy in the literature ^[9]. To add to the controversy, meta-analyses on this topic are limited by different tracheotomy techniques (open surgical procedure versus percutaneous placement), different definitions of early versus late tracheotomy, inconsistent use of weaning protocols, and differences in blinding of clinicians to group assignment ^[4, 10]. Moreover, many of the randomized controlled trials on tracheotomy timings have been under-powered to detect statistically significant differences ^[9].

One of the primary reasons to perform a tracheotomy is to minimize the complications of prolonged endotracheal intubation^[11]. Consequently, tracheotomy as early as 2–10 days post-intubation has been recommended in the otolaryngologic literature [12]. The advantages and disadvantages of both tracheotomy and endotracheal intubation are listed in Tables 19.2 and 19.3, respectively [3, 4, 9, 13, 14]. The current medical practice favors "early" tracheotomy within the first 7-10 days of intubation when extubation appears unlikely and the patient is stable ^[3, 5, 9, 12, 15]. Otherwise, endotracheal intubation is continued and reassessed daily to determine when tracheotomy is warranted. In general, the timing of the tracheotomy should be individualized, but the candidate for elective tracheotomy must be hemodynamically stable without vasoactive substances, tolerate transport to the operating room, and have sufficient pulmonary reserve for weaning to spontaneous ventilation.

Types of Tracheotomy Tubes

There are several types of tracheotomy tubes. In general they can be divided into plastic (Shiley, Fig. 19.2) and metal (Jackson, Fig. 19.3) tubes. The plastic tubes may have a cuff or inflatable balloon that occludes the airway to allow mechanical positive pressure ventilation without loss of tidal volumes and to potentially decrease the risk of aspiration. This type of tube will have a port and pilot balloon for cuff inflation and cuff pressure monitoring respectfully. Although modern tracheotomy tubes have low-pressure cuffs to decrease the risk of mucosal ischemia and secondary tracheal stenosis, the cuff should be deflated once mechanical ventilation is no longer necessary and the patient's mental status allows for airway protection and management of secretions. In patients who are tracheotomy dependent, but no longer require mechanical ventilation, tracheotomy tubes without a cuff (plastic or metal) are an option. Plastic tubes also may come with fenestrations that permit speech during tracheotomy lumen occlusion.

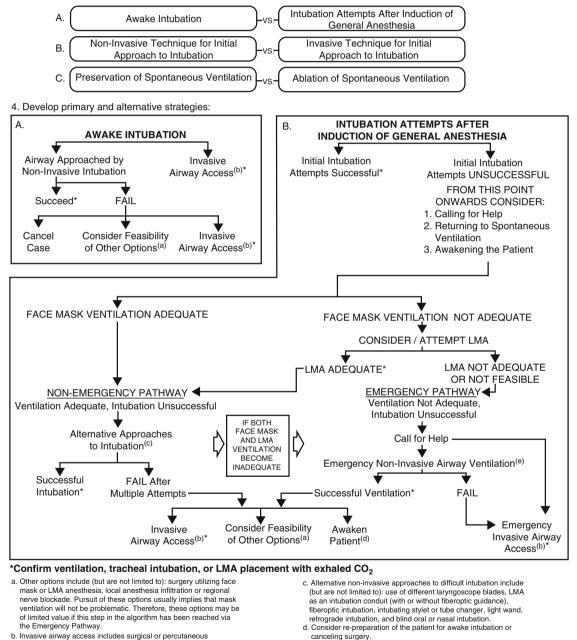
Metal (Jackson) tubes are commonly used most often for patients who are progressing towards decannulation. These tubes have narrow lumens and can be easily downsized as tolerated. In general, the adult patient who tolerates a number 5 Jackson (5 mm lumen) with a cork in place is a good candidate for decannulation. Tracheotomy-dependent patients should be evaluated via a flexible laryngoscopy or direct laryngoscopy under general anesthesia to rule out upper airway obstruction

AMERICAN SOCIETY OF ANESTHESIOLOGISTS **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:



 Invasive airway access includes surgical or percutaneous tracheotomy or cricothyrotomy

e. Options for emergency non-invasive airway ventilation include (but are not limited to): rigid bronchoscope, esophageal-tracheal combitube ventilation, or transtracheal iet ventilation.

Fig. 19.1 ASA 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 May;98(5):1269-77.

Tracheotomy^(b) (*asterisk*) may be needed after the following scenarios: failed awake intubation, failed intubation/adequate mask ventilation, or failed intubation/inadequate mask ventilation

Table 19.2 Potential advantages and disadvantages of tracheotomy

Advantages

- · Improved weaning from mechanical ventilation
 - Smaller dead space, reduced work of breathing, and decreased airway resistance
- Ease of pulmonary toilet and oral hygiene
- Better patient comfort
- Reduced sedation needs
- Better communication and swallowing ability Deflating cuff or using fenestrated tracheotomy tubes/speaking valves allow for phonation
- Increased and earlier patient mobility
- Ease of tube replacement (after stoma matures) Transfer spontaneously breathing patients to step down units or wards
- Reduced laryngeal damage
- More stable than an endotracheal tube

Disadvantages

- · More invasive and complicated operative procedure
- Stomal scar
- Stoma site bleeding or infection
- · May be unnecessary if patient improves quickly
- · Procedural and operating room costs
- Possible long-term complications including tracheal stenosis, tracheomalacia, tracheo-innominate artery fistula (rare), and tracheoesophageal fistula (rare)

 Table 19.3
 Potential advantages and disadvantages of endotracheal intubation

Advantages

- · Easier and quicker to place compared to tracheotomy
- Lower initial cost and resource use
- Prevents aspiration of secretion
- Route for medication (e.g., epinephrine, atropine)
- Avoid surgical complications of tracheotomy (e.g., nerve/tracheal injury, bleeding, stomal complications)

Disadvantages

- Sedation required
- Weaning more difficulty after long-term placement
- Long-term complications (laryngeal damage, subglottic stenosis, voice damage)
- · Higher risk of ventilator-associated pneumonia and sinusitis
- Possible improper placement (esophageal, endobronchial, self- or accidental extubation)
- Replacement may be technically difficult and require skilled operators

With their standardized 15 mm adapters, plastic tracheotomy tubes are advantageous because they allow the attachment of manual self-inflating ventilation devices and circuits from ventilators and anesthesia machines (Chap. 6) affording assisted ventilation in the setting of respiratory distress or failure. Pediatric tracheotomy tubes are predominantly plastic.

T tubes (Fig. 19.4) are a type of plastic tracheotomy tube that have a long arm that extends distally into the trachea and a proximal arm extending to the subglottis to maintain airway patency (Fig. 19.5). This allows the patient with a proximal airway obstruction to breathe orally as well as through

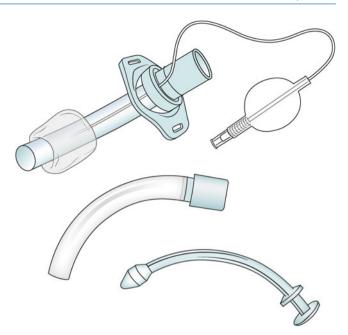


Fig. 19.2 Plastic tracheotomy tubes

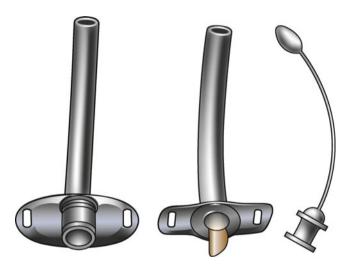
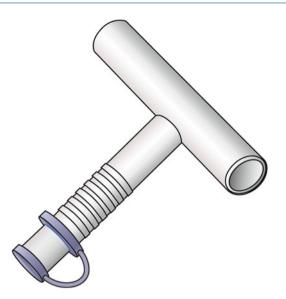


Fig. 19.3 Jackson metal tracheotomy tubes

the neck. These are used in non-ventilator-dependent patients with longstanding stable airways, but who have significant upper airway obstruction. The presence of an otolaryngologist is absolutely required should these patients require airway management or present to the operating room for elective general anesthetics.

In special situations where there are anatomic challenges either from patient body habitus or an area of tracheal stenosis that needs to be bypassed, the Shiley tracheotomy tubes are available in extended length forms. Proximal extension may be necessary in the patient with a large amount soft tissue between the skin and tracheal lumen, and distal





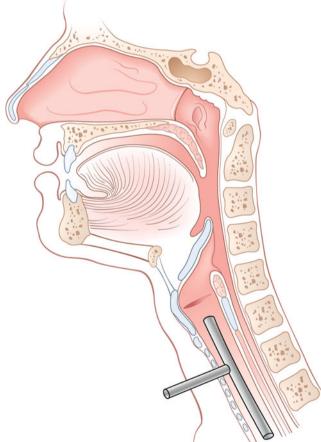


Fig. 19.5 T-tube placement in the airway

extension may be needed in the patient with an area of obstruction or granulation tissue that needs to be bypassed in the tracheal lumen. Prior to the development of these tubes, endotracheal tubes were commonly used to maintain airway control in these patients.

Tracheal Stenosis

Many times patients require ventilatory assistance and therefore should undergo conversion from endotracheal intubation to tracheotomy. A wide variety of patients can fall into this demographic but some examples include those with CNS disorders, those who have suffered a stroke, and those with respiratory failure of various etiologies. Leaving an endotracheal tube in a patient's trachea for a prolonged time can cause laryngeal stenosis and has been well described especially in the pediatric literature [16-18]. Prolonged intubation can lead to other complications and the use of a tracheotomy can be useful in these scenarios.

Airway stenosis is most commonly the result of airway intervention with endotracheal intubation. Although some may argue that the incidence of post-intubation tracheal stenosis has decreased slightly over the years with recognition of its etiology and modifications in design and management of endotracheal tubes, it still remains a challenging problem for the otolaryngologist ^[19, 20]. The stenosis is most prone to occur at the subglottis, corresponding to the level of the cricoid ring. This area is particularly susceptible to injury and subsequent stenosis because (1) the anterior aspect of the cricoid ring lies posterior to the anterior commissure of the membranous vocal folds (cricoid shelf), predisposing to injury at the time of intubation, (2) the endotracheal tube cuff is often situated at this level, (3) additional injury occurs if the cuff pressure is greater than 25 cm H₂O (capillary perfusion pressure within the perichondrium), leading to potential necrosis and inflammatory response in the cricoid cartilage, (4) the cricoid cartilage is the narrowest lumen at the apex of an inverted funnel shape formed with the thyroid cartilage, (5) the cricoid is a circumferential ring (compared to the U-shaped rings of the tracheal cartilage), predisposing to cicatrix formation, and (6) the proximity of the subglottis to the esophageal inlet makes this area particularly prone to secondary effects of gastroesophageal reflux, particularly in the sedated patient ^[21-24]. Rehabilitation of the patient requiring intubation therefore begins with proper initial airway management, conversion to tracheotomy early with the proper surgical technique, and avoidance of complications by providing vigilant care.

Preoperative Considerations

Otolaryngologist's Perspective

As with all surgery, every effort should be made to optimize the patient prior to performing a tracheotomy. Laboratory values including coagulation studies should preferably be within normal limits prior to the surgery. Typically, an INR of less than 1.5, and platelet counts exceeding 50,000 (without uremia) are acceptable. It is also important that aspirin, nonsteroidal anti-inflammatory agents, clopidogrel, coumadin, and other anticoagulation medications be stopped several days prior to the procedure when feasible. Type and cross is not necessary unless the hematocrit is very low prior to the procedure.

When a patient already has a controlled airway with endotracheal intubation, tracheostomies may be performed either in the operating room or in an intensive care unit (ICU) setting at the bedside. Although bedside tracheostomies are safe and complications are generally similar to those performed in the OR ^[25–29], technically challenging (obese, short neck, limited cervical neck range of motion, prior head and neck surgery or radiation therapy) or medically complex (significant cardiovascular, pulmonary or hematologic comorbidities) patients will likely be taken to the operating room to improve patient access and care from both the otolaryngologist and the anesthesiologist. There are certain situations that require an emergent surgical airway and the otolaryngologist should understand the proper preoperative as well as intraoperative considerations that go along with this emergency procedure (Chap. 8)^[30].

Anesthesiologist's Perspective

The most important anesthetic goal is to maintain a secure airway. Usually those patients presenting for elective tracheotomy fall into the following categories: (1) patients already intubated secondary to chronic processes (e.g., intracranial process and depressed mental status, respiratory failure, neuromuscular disease), (2) patients status post complex trauma, (3) patients scheduled for elective scheduled tracheotomy as a component of a more complex surgery (e.g., radical neck dissection) ^[3]. In most of these cases the patients are initially intubated and then the tracheotomy is performed. Although timing of tracheotomy placement is important, the cases from categories 1 and 2 are 100% elective since these patients have a secure, reliable airway.

A standard preoperative evaluation with an emphasis on the patient's pulmonary and cardiovascular status should be performed. If the patient is already intubated, it is important to note the most recent arterial blood gas and maintain mechanical ventilatory settings including PEEP in the OR and during transport. In addition, the extent of exercise tolerance, and cardiac reserve should be ascertained and if available cardiac testing such as stress test, echocardiogram, or coronary angiography should be reviewed or obtained. Preoperative lab values including complete blood count, coagulation studies, and platelet count should be noted. The patient should be hemodynamically stable to be transferred from the ICU to the operating room as well. Otherwise, it may be prudent to postpone the procedure or discuss the option of performing a bedside tracheotomy in the ICU with the otolaryngologist. NPO guidelines should also be followed, and enteral feeds discontinued 6–8 h prior to the tracheotomy ^[3]. Heparin infusions should also be suspended prior to the procedure.

For patients coming from an intensive care unit (ICU) and who have had associated multisystem organ failure it is critical to confirm medical stability and optimization of the cardiovascular, pulmonary, hepatic, renal, and hematologic systems. Since these patients are already intubated, accidental extubation during transport must be avoided since bag mask ventilation and re-intubation may prove exceedingly difficult. In our practice we assist with patient transport from the ICU to the operating room. Prior to transport correct placement of the endotracheal tube and cuff integrity are confirmed and sedation and neuromuscular blockade is initiated when appropriate. The patient is then transported in their monitored ICU bed. To minimize patient movement, which can compromise cardiovascular stability and disrupt endotracheal tube, intravenous and invasive monitoring positioning, the patient is left in the ICU bed for the procedure when possible.

For those patients who are not already intubated (category 3), the anesthesiologist needs to determine whether or not the patient may be a difficult airway and decide on the appropriate anesthetic plan. This is discussed in great detail in Chap. 8, but briefly an extensive history and physical exam should be performed, and radiological imaging should be obtained where feasible. The strongest predictor of a difficult intubation is a history of previous difficult intubation or mask ventilation^[31]. Conditions associated with a possible difficult intubation are numerous. Common factors include certain anatomical indices (e.g., limited mouth opening, large tongue, short neck, small interincisor gap, decreased thyromental distance, micrognathia, obesity, and increased Mallampati score), laryngeal/pharyngeal abnormality (e.g., vocal cord pathology, subglottic stenosis, hypopharyngeal or epiglottic tumor/abscess), and limited cervical mobility (e.g., short neck/obesity, previous cervical spine surgery, postradiation changes) [3, 32, 33] Difficult mask ventilation is also associated with the presence of a beard, lack of teeth, obesity, and previous radiation ^[3, 29, 30]. Clinical signs and symptoms such as stridor, sternal retractions from airway narrowing, diaphoresis, tachypnea, and tachycardia from the increased work of breathing should be noted. Based on this assessment, the anesthesiologist and otolaryngologist must collaboratively decide that the best and safest approach would be to perform an awake fiberoptic intubation, an asleep intubation (via fiberoptic bronchoscopy or direct laryngoscopy), or perform the tracheotomy awake under local anesthesia ^[3, 30]. Since a large percentage of anesthetic morbidity

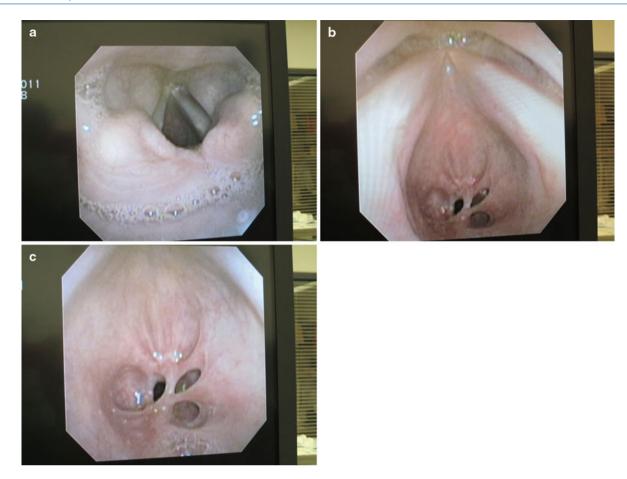


Fig 19.6 A sequence of fiberoptic views in an awake patient who was first examined bronchoscopically. Given the extent of airway obstruction from the tracheal web an awake tracheotomy was performed

and mortality involves airway mismanagement, anesthesiologists should familiarize themselves with a practice algorithm (such as Fig. 19.1) in case their first plan fails. Obviously the otolaryngologist will be immediately available to perform a tracheotomy in the case of failed intubation and/or impossible ventilation.

Ideally, the tracheotomy is performed under general anesthesia with an endotracheal intubation in place as this provides the surgeon with an immobile patient and optimal surgical conditions. In our practice we prefer to prepare most patients presenting for major head and neck surgery (Chap. 15) for an awake intubation and perform flexible laryngoscopy and bronchoscopy. If there is any concern about airway patency and the ability to pass an endotracheal tube, the otolaryngologist is present to witness the fiberoptic airway examination. Based on the examination the patient is intubated when appropriate. If there is any doubt or if the endotracheal tube does not easily pass into the airway the procedure is aborted and an awake tracheotomy is performed (Fig. 19.6).

Intraoperative Considerations

Otolaryngologist's Perspective

Elective Tracheotomy

After the patient has been evaluated and the need for a tracheotomy verified, the patient is placed on the operating table with a shoulder roll inserted underneath the shoulders to provide adequate extension of the neck. This should be performed in most patients, unless they have a known or suspected cervical spine injury or cervical disease that could cause spinal cord compression. Trauma patients with possible cervical spine injuries, patients with rheumatoid arthritis, and patients with Down's syndrome ^[34] are examples of patients where extreme neck extension is contraindicated.

Landmarks of the neck, such as the thyroid notch, the sternal notch, and the cricoid cartilage are palpated and marked with a marking pen. A high riding innominate artery can sometimes be palpated and its presence should be taken into consideration during the operation. The planned incision should also be marked and is usually placed 1-2 cm above the sternal notch or 1-2 cm below the level of the cricoid cartilage. Even if the patient is under general anesthesia, local tissue hemostasis is critical for effective surgical exposure. Therefore lidocaine 1% with 1:100,000 epinephrine should be injected into the skin and subcutaneous tissue in the area where the planned skin incision will take place. Before injecting, the anesthesiologist should be informed to confirm that the injection of epinephrine is not contraindicated based on the patient's comorbidities. After injection, the patient's neck and chest area are prepped and draped in the usual sterile fashion. Depending on the surgeon's preference, a vertical or horizontal incision is performed through the skin. Blunt dissection is carried through the subcutaneous tissue using curved hemostats and electrocautery. Some patients will require the removal of subcutaneous fat in order to provide better exposure and this can be done using electrocautery. The dissection is carried down through the platysma layer and this fascial layer is divided and lateralized using rake retractors. Any anterior jugular veins encountered should be completely identified and retracted laterally. Ligating these vessels is not necessary unless they are transected accidentally. There is a subset of patients who may have increased venous congestion (e.g., cirrhosis, prior thoracic surgery, thoracic outlet syndrome, congestive heart failure) where bridging vessels between the anterior jugular veins may need to be ligated in order to maintain a midline dissection. The median raphe is then identified and then divided and the strap muscles are lateralized with retractors. The thyroid isthmus should then be visualized as well as the anterior tracheal wall. In most instances the thyroid isthmus is small and can be retracted superiorly and the tracheal rings identified. However, sometimes the isthmus is too large and must be divided. This is performed while undermining the isthmus and reflecting it off the trachea using curved hemostats. The isthmus can then be divided using bovie cautery and then ligated using 3-0 vicryl or silk sutures on a tapered needle.

Care must be taken when using an electrocautery because of the risk of an airway fire. For this reason communication amongst the otolaryngologists and anesthesiologists is critical, patients should be ventilated on the lowest oxygen concentration allowable, nitrous oxide should be avoided and the tracheal stoma should never be created with an electrocautery. The thyroid gland is then swept laterally to expose the anterior tracheal wall as well as the cricoid cartilage. At this time a cricoid hook is placed just underneath the cricoid cartilage by an assistant and gentle upward pressure is used to elevate and stabilize the airway. The pretracheal fascia is exposed and gently swept away with a Kittner. Before entering the airway, the anesthesiologist will deflate the cuff on the endotracheal tube to prevent cuff trauma. Using a 15 blade two parallel horizontal incisions are made above and below the second or third tracheal ring. Using Metzenbaum scissors, vertical cuts are made to connect these two horizontal incisions. The anterior portion of the tracheal ring is removed in order to create a small window. Besides creating a tracheal window, a vertical incision may be used, as well as a Bjork flap ^[35]. This is done by creating an inferiorly based tracheal ring flap and suturing this flap to the inferior skin margin. This technique reduces the likelihood of accidental decannulation and can make reinsertion of a dislodged tracheotomy tube easier ^[36]. Bjork flaps should only be used in patients where prolonged tracheotomy is anticipated and not for patients that only need a temporary tracheotomy. A Bjork flap should not be performed in the pediatric population, as stenosis after this type of incision is very high. Therefore, a vertical incision is usually performed in a pediatric tracheotomy, with stay sutures tied to the lateral tracheal cartilage that can be retrieved in the postoperative period in the case of accidental decannulation. Another incision option is the cruciate incision, where intersecting horizontal and vertical incisions are placed on the anterior surface of the trachea.

Once the desired incision is made, the endotracheal tube can then slowly be withdrawn from the patient to a level just above the tracheotomy incision created. The tracheotomy can be dilated with a tracheal dilator if needed. The endotracheal tube should remain in the patient's glottis ideally with the cuff below the level of the vocal folds while the tracheotomy tube is inserted. It should also remain until the (1) tracheotomy tube position confirmed to be in the airway and not in a false lumen. This is done via the presence of persistent end-tidal carbon dioxide (bilateral breath sounds will be challenging to ascertain given the surgical site and surgical draping). (2) The tracheotomy tube face plate is sewn to the skin and circumferential neck tie placed (so the tracheotomy tube is confirmed secure and not dislodged). In case the tracheotomy tube is in a false passage or becomes dislodged, then the existing endotracheal tube may be advanced through the proximal trachea to maintain effective ventilating and oxygenation.

At this time the tracheotomy tube may be introduced into the newly created tracheotomy. One should start the insertion of the tracheotomy tube at a right angle to the trachea in order to avoid insertion problems encountered by a patient's chest. As the tracheotomy tube is being inserted, it is turned $90-135^{\circ}$ in order to align with the patient's trachea. Slight resistance should be anticipated. However, difficult insertion should alert the surgeon to improper tracheotomy tube placement. Either way, the insertion process should always be done with an obturator. Once the tracheotomy tube is placed within the lumen of the trachea, the obturator can be removed and the inner cannula should be placed. This is then attached to the ventilator circuit. Once adequate ventilation is achieved through the tracheotomy tube, the endotracheal tube, cricoid hook, and retractors may be removed and the tracheotomy tube can be secured to the neck. This should be done with 2-0 silk sutures on a cutting needle to the underlying skin. A tracheotomy tie should also be placed around the patient's neck with only two fingerbreadths of space between the tie and the skin. This tie should be placed after the shoulder roll has been removed and with the patient's neck in a flexed position. Patients undergoing flap reconstruction or major head and neck procedures should only have sutures placed to secure the tracheotomy tube in order to prevent vascular compromise to local or free flaps.

Emergent Awake Tracheotomy

In coordination with anesthesiologists, emergency room physicians, and trauma surgeons, the otolaryngologist should play an integral role in emergency airway management. Performing the emergent surgical airway should be part of the otolaryngologist's armamentarium. If the patient's respiratory and mental status will allow, and an awake endotracheal intubation is not an option, then the patient should undergo an emergent awake tracheotomy in a controlled operating room setting. In order to achieve beneficial outcomes from an emergent awake tracheotomy procedure several steps should be followed. The patient should be in the upright/lounge chair position with a shoulder roll in place. No general or intravenous anesthetic, anxiolytic, or pain medication should be given that would suppress the patient's spontaneous respiratory drive. Subcutaneous injection with 1% lidocaine with 1:100,000 epinephrine should be placed. The use of an incision with wide exposure and cricoid hook should be performed as described above. Clear communication with the anesthesiologist and the nursing staff throughout the procedure is critical. Once the airway is established, general anesthetics should be administered quickly in order to prevent the combative patient from being a danger to themselves or others in the environment. A direct laryngoscopy to assess the etiology of the obstruction can be performed if indicated ^[28].

Percutaneous Dilational Tracheotomy

Percutaneous dilational tracheotomy has become popular in recent years and is common in certain intensive care units ^[37]. This procedure entails bronchoscopic-guided insertion of a needle into the trachea, followed by insertion of a guidewire into the lumen, and then serial dilation via Seldinger technique. The final insertion is the tracheotomy tube.

This procedure offers several advantages over the conventional open tracheotomy. Proponents state that this procedure is easier to perform, has a shorter operative time, lower stomal infection rate, can be performed at the bedside, is lower in cost, and avoids the need to transport the patient to the operation room. Those opposed to the use of this technique argue that blind entry into the trachea is dangerous and complications can be significant, including (1) high or lateral position of the tracheotomy tube, (2) trauma to the posterior tracheal wall, and (3) prolapse of anterior tracheal cartilage into the airway lumen, all leading to higher risk of high tracheal stenosis. There is also the risk of excessive bleeding if the site is dilated through a vascular thyroid gland, or if a high-riding innominate artery is not identified.

Perioperative complications in the reported literature, however, are at least comparable with those of surgical tracheotomy and most of them are minor when flexible bronchcoscopy is performed with the procedure to insure proper needle site insertion into the trachea and to limit trauma to the posterior tracheal wall during the insertion process ^[38]. This technique should be in the armamentarium of the otolaryngologist and can be a viable alternative for certain patients. Note that the best candidates will have palpable surface anatomy and a high cricoid cartilage. The surgeon must be prepared to convert to an open procedure if indicated, and the surgeon introducing the percutaneous tracheotomy is prepared to attend to the needs of these patients depending on the ease of decannulation.

Anesthesiologist's Perspective

Regardless of whether the tracheotomy is placed awake or under general anesthesia, prior to the procedure start, as with all cases, the anesthesia machine should be checked and airway equipment including a bag-valve mask resuscitation device for manual ventilation (i.e., Ambu bag) and suction must be available. American Society of Anesthesiologists (ASA) standard monitors—blood pressure measurements every 5 min at a minimum, capnography, electrocardiography, and pulse oximetry—are used. For patients with significant neurological or cardiovascular disease, an arterial catheter may be necessary for more accurate blood pressure monitoring. Peripheral intravenous access is required.

The patient will be placed supine with a bolster or roll placed transversely behind the shoulders to extend the head and neck to expose the neck and increase the distance from the cricoid cartilage to the sternal notch, thereby maximizing surgical exposure ^[12, 39]. This extension is limited in patients with unstable cervical spines. The head of bed may be

elevated $15-20^{\circ}$ to minimize venous engorgement ^[11]. Prophylactic antibiotics targeting skin microbes can be given 30 min prior to skin incision (at our institution, cefazolin 1-2 g IV is used) if desired ^[3].

The Awake Tracheotomy

Although ideally the tracheotomy is performed under general anesthesia, if there is any concern or difficulty establishing a secure airway prior to the procedure then an awake tracheotomy should be planned. Although the literature indicates that laryngeal mask airways (LMA) have been successfully used in tracheostomies particularly the percutaneous dilatational type as well [40, 41], at this point we would advocate that the tracheotomy be performed safely with local anesthesia. Bilateral superficial cervical plexus blocks can supplement local infiltration by the surgeons ^[30]. Patient cooperation is required, and is best achieved through verbal reassurance by both the anesthesiologist and surgeon. Heavy sedation should be avoided as the synergistic effects of benzodiazepines, opioids, and propofol may depress or abolish the patient's respiratory drive. Small amounts of premedication such as an anxiolytic (1-2 mg midazolam) or narcotic (10-25 mcg fentanyl titrated at a time) alone may be utilized only if the likelihood of ventilator depression is extremely low (e.g., young patient). Supplementation with low-dose dexmedetomidine (0.2-0.7 mcg/kg/h) has been successfully used ^[42-44].

Tracheostomies Under General Anesthesia

Induction

If intubated the patient is connected to the anesthesia machine circuit. Once positive end tidal CO₂ is obtained, general anesthesia can be induced via intravenous induction agents or volatile anesthetic gases. In the event of an anticipated difficult airway, an awake fiberoptic intubation followed with induction of anesthesia is recommended. If the patient is not intubated and there is no anticipated airway difficulty, standard induction (dose and agent dependent on the patient's comorbidities, age, and weight) followed with laryngoscopy may be performed. Muscle relaxant is used to optimize surgical immobility and opioids (fentanyl is common) are used for analgesia. Administration of fentanyl or lidocaine (1.5 mg/kg IV) approximately 2 min prior to tracheotomy incision may attenuate any increases in heart rate and blood pressure from surgical stimulation [30]. The surgeon will also infiltrate the skin and neck with local anesthesia with epinephrine to decrease bleeding and supply analgesia. The anesthesiologists must be prepared to discuss the risks and benefits of epinephrine infiltration in cases when the patient's

comorbidities make this injection too risky (patients with significant coronary artery disease, valvular disease including hypertrophic cardiomyopathy, aneurysm disease etc.).

Maintenance

Anesthetic maintenance usually consists of inhaled volatile gases or intravenous infusions (propofol at 50–150 mcg/kg/ min is commonly used), fentanyl infusion or boluses, and/or neuromuscular blockade. Blood loss is minimal, and fluid requirements encompass deficits and maintenance rates (on average 1–2 ml/kg/h of crystalloid for the entire tracheotomy) ^[3]. Although patients arriving from ICUs are medically "optimized" to undergo this elective procedure, they generally have limited cardiovascular reserve, questionable fluid balance, and have been receiving chronic sedation, therefore they generally tolerate and require very little additional anesthesia for the procedure.

During the open surgical tracheotomy technique, the trachea is incised and a dilator maximally exposes the tracheal lumen. Once the endotracheal tube is visualized within the lumen, the anesthesiologist slowly removes the tube until the posterior tracheal wall is seen [1]. At this point, the tracheotomy tube is placed into the tracheal lumen under direct visualization [3, 30, 36]. Correct tracheotomy placement must be verified by positive end tidal CO₂ on capnography before the endotracheal tube is removed [3, 36]. The trachea should be suctioned with a flexible catheter as well. Then the endotracheal tube can be removed and the tracheotomy tube secured. If there is no end tidal CO₂ seen or an inability to ventilate, the endotracheal tube must be reinserted into the trachea. It is likely that the tracheotomy tube is in a false passage, either anterior to the tracheal wall or posterior to the trachea into the esophagus^[3]. If there is doubt, bronchoscopic confirmation can be performed and a tracheal suction catheter can be inserted. If the tracheotomy is correctly placed, the suction catheter should easily pass through the entire tube length. Tube malposition can also be determined by the lack of a cough response in a lightly anesthetized patient or the lack of sputum or bloody tracheal secretions in the asleep patient ^[36]. In both the percutaneous and open surgical technique, it is crucial not to completely remove the endotracheal tube from the trachea until positive end tidal CO₂ confirmation and/or bilateral chest rise with bilateral equal breath sounds. In addition, when the tracheotomy is performed as part of a major head and neck procedure like a free flap reconstruction, a reinforced/anode tube can be placed through the tracheotomy and sutured. It is then replaced at the end of surgery by a cuffed tracheotomy tube ^[3].

Intraoperative complications can occur and are described in more detail in the Postoperative Immediate Complication section (see below).

 Table 19.4
 Tracheotomy complications

Immediate	Early	Late	
Bleeding	Pneumomediastinum/pneumothorax	Minor bleeding from local vessels	
Pneumothorax	Subcutaneous emphysema	Major bleeding (erosion into innominate blood vessel)	
Intraoperative airway fire	Airway obstruction	Infection (pneumonia, stomal)	
Venous air embolism	Dislodged tracheotomy tube	Tracheal stenosis	
False passage of tracheotomy tube	Stoma infection or erosion	Tracheoinnominate fistula	
	False passage of tracheotomy tube	Tracheoesophageal fistula	
	Pneumonia	Tracheocutaneous fistula	
	Bleeding	Tracheomalacia	

Emergence

Hemostasis of the tracheotomy site should be achieved prior to emergence. Allowing the patient to remain anesthetized, breathing spontaneously, can eliminate bucking and coughing during emergence and thus minimize bleeding and potential barotrauma. Patients can be transported breathing spontaneously with a supplemental oxygen source from either a tracheotomy collar or a T piece.

Postoperative Considerations

Otolaryngologist's and Anesthesiologist's Perspectives

Immediately postoperative, patients are placed with the head of bed in a 30-45° position to maximize deep breathing, facilitate suctioning, and to decrease pain associated with coughing. Nursing and post anesthesia care unit (PACU) care are critical to evaluate the patient's vitals as changes in vital signs may indicate a problem with the tracheotomy tube. Humidifying inspired air is necessary to prevent crusting and tracheitis. Suctioning should be performed frequently in the immediate postoperative period to clear any secretions and to prevent mucus plugging of the tracheotomy tube. The frequency of suctioning can slowly be decreased as the patient begins to recover. The tracheotomy site should be cleaned as often as necessary in order to prevent any wound infections. This cleaning frequency can also be decreased as the postoperative time increases. The inner cannula of the tracheotomy tube must be removed and cleaned frequently to prevent mucous plugs. Sutures used to keep the tracheotomy tube secure, stay sutures and Bjork sutures can be removed 3-5 days postoperatively. Tracheotomy tube changing can also be performed during this time interval as the stomal tract has matured by this time.

Decannulation should only be considered if the underlying disease process that resulted in the necessity of a tracheotomy has resolved. Patency of the airway should be assessed prior to decannulation via fiberoptic or mirror laryngeal exam. Another way to assess for patency is to gradually down size the tracheotomy tube over several days. This tube can then be corked or occluded and the patient's respiratory condition evaluated. The patient should be able to tolerate occlusion of the tube for over 24 h prior to decannulation. After decannulation has taken place, the stoma is covered with a light dressing and occlusive tape or steri-strips.

Complications of Tracheotomy and Airway Stenosis Surgery

Complications can occur during and after tracheotomy placement and are characterized as immediate, early, and late (Table 19.4)^[3,4,12,45]. Although these complications can overlap in time, immediate complications generally occur during placement and include bleeding, pneumothorax, fire, air embolus and the creation of a false passage. Early complications occur in the early postoperative period and include infection, hemorrhage, subcutaneous emphysema, pneumomediastinum, pneumothorax, tracheoesophageal fistula, recurrent laryngeal nerve injury, tube displacement, and intraoperative airway fire. Late complications include tracheal-innominate artery fistula, tracheal stenosis, delayed tracheoesophageal fistula, and tracheocutaneous fistula.

Intraoperative and Immediate Complications

Immediate complications occur during and immediately after the procedure, and can be seen in the PACU.

Bleeding

Post-tracheotomy bleeding is the most common complication. It is less likely with percutaneous dilatational tracheotomy since there is limited soft tissue dissection. If bleeding occurs intraoperatively, it is likely from the anterior jugular veins early in the dissection, highly vascular thyroid isthmus later in the dissection, and thyroid ima artery ^[3]. Tracheostomies done below the fourth tracheal ring have a higher incidence of damage to the innominate artery and bleeding is more common ^[3]. When dissection is performed inferiorly, high-riding innominate arteries can increase the risk of severe hemorrhage ^[3]. Therefore, after the tracheotomy tube is placed, the surgical site should be carefully inspected for any bleeding and hemostasis should be achieved prior to emergence. Good pulmonary toilet should also occur prior to emergence to prevent the aspiration and formation of significant blood clots in the airway. Tracheotomy cuff inflation and packing is usually adequate to controlled bleeding ^[12]. Major postoperative hemorrhage may require a reoperation and wound exploration.

Pneumothorax

Pneumothorax from pleural puncture can occur immediately as well as the early periods postoperatively. In adults, it is more likely following a difficult tracheotomy or one with excessive lateral dissection with posterior tracheal wall damage during tracheotomy tube placement or tracheal invision ^[3, 36]. With adults, this complication usually involves a pulmonary bleb rupture in a patient struggling to breathe during an awake emergency tracheotomy [3]. Pneumothorax overall occurs more commonly in children because of anatomy; the relatively elevated pulmonary apices are more exposed and hence, vulnerable in the neck to puncture ^[3, 46]. If pneumothorax occurs and is symptomatic, an emergent needle decompression in the second intercostal space midclavicular line maybe necessary followed by the insertion of a chest tube Some literature recommend postoperative portable chest radiographs on all tracheostomies; however, other studies suggest that it is unnecessary in routine cases ^[3]. In these cases, a chest radiograph should be done if there is a high suspicion of pneumothorax (e.g., in emergency or difficult tracheostomies, patients with signs and symptoms of pneumothorax)^[3, 36].

Airway Fire

Airway fires, although rare, can occur during tracheostomies ^[47–50]. Consequently, saline should be available on the surgical field at all times. The inspired oxygen concentration (FiO₂) should be as low as tolerated and used in air mixtures or helium. Nitrous oxide should be avoided as it can support combustion. The endotracheal cuff can be filled with saline to prevent ignition in case of cuff puncture. In order to ensure that the endotracheal cuff is not accidentally punctured, the endotracheal tube should be advanced close to the carina (24–25 cm from the teeth in the average male) ^[3, 44, 51]. Also, tenting surgical drapes should be avoided to prevent oxygen buildup, as well as alcohol-based and petroleum-based products ^[3]. The trachea should be incised with a scalpel, knife, or

scissor; electrocautery should never be used to enter the trachea ^[45, 46]. Diathermy increases the airway fire risk and is ineffective in cutting through calcified tracheal rings and in preventing mucosal bleeding ^[44].

In the case of an airway fire, immediately disconnect the patient from the ventilator, switch off the anesthetic gas, and ventilate with room air using a self-inflating bag ^[44]. Flush saline down the endotracheal tube and in the surgical field to extinguish the fire. Assess the benefits and risks of removing the tracheotomy tube. Be aware that the airway may be lost during tube exchange in edematous or prior difficult airway patients. If the tube is removed or changed, the spread of the fire into the tracheobronchial tree and possible inhalation injury are minimized ^[44]. Mask ventilate the patient with 100% oxygen and then re-intubate. Continue mechanical ventilation, administer high-dose steroids, and apply PEEP if necessary ^[3].

Venous Air Embolism

Another extremely rare but life-threatening intraoperative complication that can occur is an air embolism. This happens after air is entrained through an opening in the venous circulation. It is more likely to occur in an awake tracheotomy when inspiration produces negative venous pressure and distension in the neck veins ^[3]. To prevent an air embolism, venous hemorrhage should be adequately controlled. If a venous air embolism occurs, notify the surgeon immediately. Stop the tracheotomy and administer 100% oxygen. Place the patient in Trendelenburg and left lateral decubitus positions in order to trap the air in the ventricular apex so it does not travel into the pulmonary arterial tree ^[3]. Then provide hemodynamic support by fluid administration, vasopressors, and cardiopulmonary resuscitation as needed.

Early Complications

Early complications occur in the first postoperative week and include tracheotomy tube obstruction and dislodgement, subcutaneous emphysema, pneumomediastinum, pneumopericardium, and pneumonia.

Tracheotomy Tube Dislodgement and Decannulation

The first few days after tracheotomy placement are crucial and patients need to be monitored carefully. It takes about 7 days for the tract from the stoma to the trachea to mature. If the tracheotomy tube is removed prior to maturation, the tissue planes collapse upon each other and the airway can be lost. This inadvertent decannulation or displacement is a true airway emergency and can be life threatening. This primarily occurs in obese patients with thick necks in which

Risk factors	Prevention ^a		
Inadequately secured tube	Secure tube with sutures through neck flange and tracheotomy ties should be snug		
Altered mental status, agitation	Sedation and verbal reassurance as needed.		
Patient turning	Minimize transport in early postoperative period		
Increased pulmonary secretions	Aggressive pulmonary toilet		
Lack of restraints	Restrain as necessary. Relieve traction and weight off ventilator tubing		

Table 19.5 Risk factors and preventive measures for accidental decannulation [3, 13]

^aIdentify which patients were prior difficult airways, place extra tracheotomy tube by the bedside, and familiarize oneself with how to manage tracheotomy dislodgement

there is a progressive increase in the distance from the anterior tracheal wall to the neck skin (exacerbated by postoperative edema), resulting in a malpositioned tube ^[13, 36]. Other risk factors include adjustable-length tubes, excessive coughing, excessive movements of an agitated uncooperative patient, and a loosely tied tracheotomy tube (see Table 19.5) ^[3, 12, 13, 36]. Inability to pass a suction catheter into the trachea is ominous and may be a sign of malposition but may be a sign of a blood clot or mucous plug. The displacement may manifest as respiratory distress especially if the patient's airway is partially obstructed ^[3, 13]. The patient may present with stridor and complain of dyspnea. Increased airway resistance will likely be observed during positive pressure ventilation with a ventilator or manual resuscitation bag^[3]. Complete occlusion is a medical emergency and if not detected or corrected will result in respiratory arrest and death.

If the tracheotomy tube is dislodged, the corrective plan is based on the age of the tracheotomy and whether or not the patient is status post a total laryngectomy. (Patients with a tracheotomy who are status post a total laryngectomy can neither be ventilated nor intubated orally or nasally since they are left with a blind pharyngeal pouch.) If a dislodged tracheotomy tube is reinserted in a "fresh" tracheotomy (within 7 days of tracheotomy placement), the tube may be placed in a false passage, ending up in the anterior mediastinum. This is more likely to occur in patients with thick, obese, or short necks ^[13]. In this case, the tracheotomy tube should not be reinserted, but should be removed and bag mask ventilation initiated followed by oral endotracheal intubation. Once the airway is secured, the tracheotomy should be formally revised in the operating room. If the tracheotomy tube is accidentally decannulated after postoperative day 7, then the tracheotomy tract is likely to be mature. (Fig. 19.7)^[52]. In this case, the tracheotomy tube is reinserted

and the position is confirmed with fiberoptic bronchoscopy ^[3]. If at any point the patient is unstable or has no relief of respiratory distress, the tracheotomy tube should be removed, bag mask ventilation initiated and endotracheal tube placed. The stoma is then directly suctioned and the patient intubated (Fig. 19.8).

Tube Obstruction

Tracheotomy tubes can become obstructed by mucous plugs and blood clots in addition to malposition. Since the tracheotomy bypasses the upper airway where gas humidification occurs, patients are prone to drying of secretions which then produces mucous plugs^[3]. Obstruction can be minimized by frequent nursing care involving suctioning, hydration, and humidified oxygen ^[3]. When the tube is occluded, immediately check and clean the inner cannula. Deep suctioning may be needed which can be assisted with aggressive use of a manual resuscitation bag to promote coughing and oxygenation^[3]. If the obstruction is still unrelieved, the tracheotomy tube should be changed or removed and the stoma should be directly suctioned. If needed, flexible bronchoscopy can be utilized to remove distal deeper clots or plugs [3, 13, 36]. Literature also recommends the Heimlich maneuver to "bring a mucus plug into reach" in the patient for whom it is not contraindicated ^[3].

Subcutaneous Emphysema

Subcutaneous emphysema can occur from coughing or bucking against the packed and sutured tracheotomy tube or from positive pressure ventilation ^[3, 12, 56]. This presents as crepitus around the tracheotomy site, and rarely, can encompass more of the body with forceful use of the manual resuscitation bag with an immature stoma ^[3, 50]. Subcutaneous emphysema can be prevented by several surgical measures including avoiding excessive dissection lateral to the trachea and avoiding skin incision closure when the tracheotomy site is packed (thus, allowing air to escape) ^[3]. In terms of anesthesia, the patient should be deep to minimize coughing or bucking prior to tube insertion. Subcutaneous emphysema will subside spontaneously but pneumothorax should be ruled out. If the site is sutured closed or packed, it should be opened as well.

Pneumomediastinum and Pneumopericardium

Pneumomediastinum and pneumopericardium occur when air tracks from skin incision into the chest cavity and then accumulates, possibly causing difficulty with ventilation, decreased cardiac output, and cardiovascular collapse ^[3, 50]. The patient may complain of dyspnea and pleuritic chest pain spreading to the shoulders, back, and neck. As with pneumothorax

Obturator

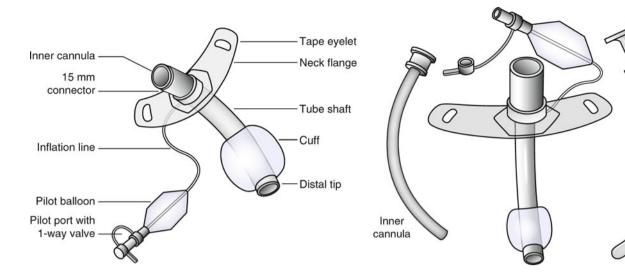


Fig 19.7 Standard tracheotomy tube components ^[49]

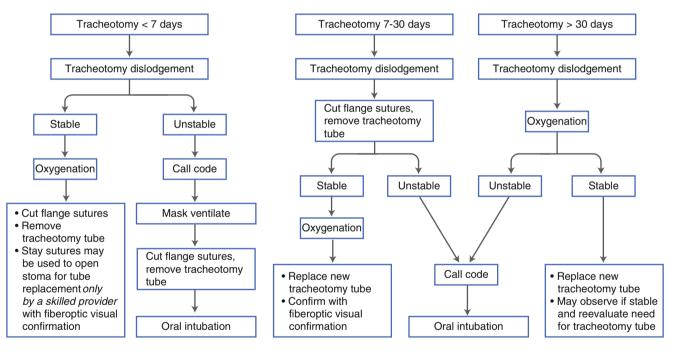


Fig 19.8 Algorithm for management of tracheotomy tube dislodgement ^[13] (From O'Connor HH MD, Resp Care, Aug 2010 Vol 55 No 8. Adapted from illustrations courtesy of Stanley Nasraway MD, Tufts Medical Center, Boston, Massachusetts)

and subcutaneous emphysema, pneumomediastinum occurs when there is extensive peritracheal dissection and increased negative intrathoracic pressures develop with forceful inspiration ^[3]. Consequently, prevention and management is similar. An airway should ideally be placed prior to tracheotomy start and for the symptomatic patient a chest tube is inserted ^[3]. Chest radiographs should be ordered as well.

Late Complications

Few rare, but clinically relevant late postoperative complications can occur beyond one week of tracheotomy placement. These include granulation, tracheoinnominate fistula (TIF), tracheoesophageal fistula, tracheocutaneous fistula, tracheomalacia, and tracheal stenosis.

Stomal Granulomas

The most frequent is granulation tissue formation. This can be asymptomatic or present as inability to decannulate, inability to wean from the ventilator, or upper airway obstruction/respiratory failure after decannulation ^[42]. Granulomas can occur from foreign body reaction to the tracheotomy tube or certain parts of it and are more common with fenestrated tubes ^[11, 12, 36]. They are treated with topical silver nitrate cauterization, inhaled beclomethasone, CO₂/YAG laser or surgical excision ^[3, 12].

Tracheoinnominate Fistulas and Hemorrhage

This fistula develops when the tracheotomy tube erodes into the anterior tracheal wall, resulting in a communication between the trachea and the posterior wall of the innominate artery as it crosses the trachea in the mediastinum ^[36]. This complication is extremely rare with a reported incidence of 0.7%. However, when hemorrhage from the fistula occurs, there is a mortality rate ranging 80–100% even with surgery ^[3,42]. The majority of these cases (approximately 75%) occur 1–4 weeks after the initial tracheotomy placement. Risk factors include tracheotomy below the fourth tracheal ring, overinflated cuff, excessive tube movement, hypotension resulting in decreased mucosal capillary pressure, sepsis, corticosteroids, diabetes, and malnutrition ^[3,42].

Due to the high mortality associated with TIF, it is imperative to minimize risk factors. Therefore, low surgical stomal incision should be avoided, cuff pressures regularly checked, and hypotension treated. The hallmark of such a hemorrhage is an initial sentinel bleed (self-limited episode of bright red blood from the stoma) and pulsation of the tracheotomy tube coinciding with the heartbeat followed by a fatal massive hemorrhage ^[3, 36, 42, 53]. When seeing bleeding around the tracheotomy site 72 h after insertion, the clinician should have a high suspicion of TIF until proven otherwise ^[51].

When TIF is suspected, the patient should be emergently sent to the operating room for direct exploration and surgical repair under general anesthesia. Prior large bore intravenous access is required as is the assurance that a blood specimen was sent to the blood bank for an active type and cross since massive blood transfusions is likely. If massive hemorrhage occurs, the tracheotomy cuff should be overinflated to aid in tamponade ^[3,42,51]. This may require re-intubation with an endotracheal tube and positioning the cuff over the fistula ^[3].

Tracheoesophageal Fistula

This type of fistula is rare and occurs less than 1% of patients ^[12]. It usually develops after the tube erodes into the posterior tracheal wall especially with an in-situ nasogastric tube ^[3, 12]. One can suspect TEF when the patient has excessive secretions, continuous cuff leak, recurrent aspiration, and/or gastric contents presenting through the tracheotomy ^[3, 42]. TEF is

diagnosed with bronchoscopy esophagoscopy or esophagography, and then surgically repaired.

Tracheocutaneous Fistula

This fistula occurs when the stoma does not close after decannulation. It presents as consistent mucus draining onto the anterior chest wall and cough, altogether promoting recurrent aspiration and impaired swallowing ^[3]. TCF is closed with electrocautery of the fistula walls or a more formal surgical excision and closure ^[3,36].

Tracheomalacia

Tracheomalacia—weakening of the tracheal wall—develops after ischemic injury (i.e., direct pressure from tube or cuff), followed by chondritis and subsequent necrosis of supporting tracheal cartilage ^[51]. Consequently, the trachea collapses during expiration and may be compressed by adjacent structures. This presents as dyspnea in patients with a past history of tracheotomy or failure to wean from mechanical ventilation ^[3, 42]. Flow-volume loops of spontaneously breathing patients will reveal variable intrathoracic obstruction. Prevention involves the maintenance of cuff pressures below 20 mmHg ^[3]. Treatment reflects severity of the expiratory obstruction and ranges from conservative measures to tracheal stenting and tracheoplasty.

Tracheal Stenosis

It is difficult to ascertain whether tracheal stenosis is a complication of tracheotomy alone or in addition to prolonged endotracheal intubation prior to tracheotomy ^[36]. Narrowing commonly occurs at the stoma; above the stoma (suprastomal) but below the vocal cords (subglottic); at the tracheotomy tube cuff (infrastomal); or at the distal tip of the tube [12, 42]. Stomal stenosis develops after bacterial infection and granulation formation usually and has the same risk factors as tracheomalacia and granulation. Suprastomal stenosis has been seen as a complication of percutaneous dilation tracheotomy when there is guidewire-related injury to the posterior tracheal wall^[42]. Infrastomal stenosis is usually due to ischemia injury to the mucosa as seen with overinflated cuffs, and the incidence has decreased with the advent of high-volume lowpressure cuffs ^[3, 42]. With prolonged ischemia, chondritis, ulceration, and necrosis occur, leading to granulation and fibrous narrowing ^[3, 36]. Stenosis at the tip of the tracheotomy tube occurs when the tip rubs against either the anterior or posterior tracheal wall.

The clinician should have a high degree of suspicion when a patient is unable to be weaned off chronic ventilation or be decannulated, particularly when a patient has a past history of intubation and/or tracheotomy ^[42]. Tracheal stenosis may manifest as dyspnea months to years after decannulation. Patients are usually asymptomatic until the tracheal lumen has been reduced by 50–75% ^[42]. Once the tracheal lumen has been reduced to less than 10 mm, exertional dyspnea is seen ^[42]. When the lumen is less than 5 mm, stridor or dyspnea at rest is observed ^[3, 42]. Tracheal resection may be necessary.

In addition to stenosis, granulation tissue formation is another potential risk of tracheotomy. It can complicate the postoperative course of a tracheotomy for several reasons. Granulation tissue can cause bleeding and lead to aspiration of blood into the airway ^[53]. It can also delay attempts at decannulation ^[54]. Granulation tissue can also obstruct the tracheotomy tube, which can lead to fatal outcomes. Frequent tracheotomy tube changes every 3–4 weeks can dramatically reduce the incidence of this problem ^[52]. Also, controlling bacterial infections around the stoma and controlling gastroesophageal reflux are thought to decrease the incidence of granulation tissue formation.

Airway Stenosis Surgery

There is a wide spectrum of surgical procedures for airway stenosis, and the specific details of these procedures are beyond the scope of this text. Generally, procedures fall into multiple categories: (1) endoscopic versus open surgery, (2) patients entering a procedure with or without an existing tracheotomy, (3) the use of stents, keels and T-tubes, and (4) patients emerging from anesthesia with or without a secure airway lumen (tracheotomy or T-tube).

Endoscopic surgery for tracheal stenosis involves direct line of sight laryngoscopy with the use of a telescope or microscope to allow a magnified field of view. Flexible endoscopes are also frequently used to access the distal airway, and this equipment should be available during any airway stenosis procedure in case of loss of the airway or suspected mucus plugging of the distal airway. A video monitor in the operating room is essential for good communication between the surgeon and anesthesiologist, and joint confirmation of a secure airway.

Patients presenting for the procedure with an existing tracheotomy simplify the anesthesia induction and the initial risk of airway lost. However these procedures are more likely to use the CO_2 or other lasers, requiring strict laser safety to avoid complications such as an airway fire. If patients have some airway compromise before surgery and an awake tracheotomy is not planned, the scrub nurse should have an open tracheotomy set in the room with an open blade in preparedness for possible urgent tracheotomy during induction in case the airway is lost. Again, communication regarding airway and sedation management during induction is paramount to safety. If the patient can tolerate bagging through mask ventilation, then the surgeon may expose the larynx and jet ventilation may be used for oxygenation. However, in this situation ventilation and CO_2 egress is passive, thereby increasing the risk of CO_2 retention during tachypneic jetting.

Stents and keels are foreign bodies placed more commonly in open surgery procedures and are used to reduce scarring postoperatively. They must be removed at a subsequent visit to the operating room. T-tubes are similar to tracheotomy tubes in that they have access to the distal airway. However, they do not have a cuff and have a proximal limb tenting open a stenotic proximal trachea or subglottis. As such, ventilation through this circuit is often accompanied by periods of air-leak. In these situations, inspired and expired tidal volumes on the anesthesiology circuit will not be reliable. Similar to jet ventilation, a pressure mode should be used on the ventilator to insure adequate chest-wall rise and continued oxygenation. It is also preferable not to use inhaled anesthetics when a large leak to the operating room air is expected, as these may have an unfavorable effect on the surgeon.

Each of these permutations requires a specific strategy and backup plan for joint airway management between the otolaryngologist and the anesthesiologist. The general principles are the same regardless of the unique situation, including (1) a preference for more secure airways, (2) advanced planning by the surgeon-anesthesiology team, and (3) preparedness of surgical nursing especially during anesthesia induction and emergence.

The anesthetic evaluation and intraoperative management for tracheal stenosis surgery is discussed in detail in Chap. 10.

The Patient with a Preexisting Tracheotomy

The evaluation of the patient should be directed toward the tracheotomy site, the tracheotomy apparatus, and the indications for initial tracheotomy placement. If the tracheotomy is still sutured, the anesthesiologist must assume the tracheotomy site is not mature. Tracheotomy maturation occurs around 7 days. If less than 7 days have lapsed since placement, an otolaryngologist must be present before electively attempting to exchange a tracheotomy for another airway. If mature, airway exchange is generally an easy process. Patients with tracheotomies frequently present to the operating room breathing spontaneously. In this situation, the anesthesiologist must determine the type of tracheotomy appliance, the presence of a 15 mm adaptor for the attachment of a bag-valve manual ventilation or the circuit from a ventilator or anesthesia machine, and the presence and integrity of a cuff before inducing general anesthesia since the ability to deliver positive pressure ventilation maybe limited or impossible with certain devices.

Because Jackson tracheotomies neither possess a 15 mm adaptor or a cuff, the ability to deliver positive

pressure ventilation is generally not possible. Although preoxygenation can be facilitated via the tracheotomy site, the ability to ventilate and oxygenate once apneic can only be accomplished reliably by exchanging the Jackson for either a cuffed endotracheal tube or a cuffed Shiley tracheotomy. Depending on the patient's respiratory reserve, the decision to replace the Jackson before or after the anesthetic induction must be considered. Although some have advocated using the 15 mm adaptor from a 5.5 ID diameter pediatric endotracheal tube placed in the lumen of the Jackson to create a more reliable attachment for pre-oxygenation and possible ventilation, the authors recommend considering exchanging the device since the lack of a cuff will make positive ventilation unreliable regardless of this modification.

Although all Shiley tracheotomies have a 15 mm adaptor for mechanical ventilation, a cuff may or may not be present. Even if a cuff is present, it is usually deflated in spontaneously breathing patients to reduce tracheal trauma and patient discomfort. Prior to the induction of general anesthesia, it is critical to determine the presence or absence of a cuff and whether or not the cuff is or isn't inflated. Ideally cuff integrity should also be determined prior to induction, even if this inflation is mildly uncomfortable for the patient.

Depending on the original device and the need for surgical site avoidance, the tracheotomy site can be maintained with either a cuffed Shiley Tracheotomy or a cuffed, typically, wire reinforced anode endotracheal tube. Once asleep the tracheotomy site should be suctioned; 4% lidocaine maybe placed endotracheally to reduce airway irritation and an anode tube can be placed. Careful attention must be made to avoid a mainstem intubation and chest rise, bilateral breathe sounds, peak airway pressures, oxygen saturation, and end-tidal capnography should guide placement. Depending on the surgical case, the tube can be sutured to the patient's chest wall by the otolaryngologist. During the procedure, these tubes have a tendency to migrate into the main stem and increased airway pressures, desaturation, and changes of capnography morphology should guide management.

Special Considerations for the Patient with a Tracheotomy

Lung Isolation in Patients with Tracheotomy

There is limited literature on how to perform lung isolation and one lung ventilation for patients with tracheotomy. Double lumen tubes (DLT) have been successfully used in patients with tracheostomies. However, DLT inserted through the stoma is more likely to be malpositioned because the upper airway has been shortened and the typical DLT is too long ^[55].
 Table 19.6
 Lung isolation options for patients with tracheostomics

 ^[55, 57]

- · Insert BB or DLT through tracheotomy stoma
- Place a single lumen endotracheal tube (SLT) through the stoma followed by BB
- · Place a SLT through the stoma followed by a coaxial BB
- Replace tracheotomy cannula with a disposable-cuff cannula followed by a coaxial BB
- Replace cannula with short DLT (i.e., Naruke DLT that is specifically for patients with tracheostomies)
- · (If possible) perform oral endotracheal intubation followed by BB
- (If possible) perform DLT orally

Moreover, the DLT may require tracheotomy tube removal, which is not an option in fresh tracheostomies with immature tracts [56, 57]. Bronchial blockers (BB) can also be used and be placed through or alongside the endotracheal tube. Bronchial blockers consist of the Arndt wire-guided endobronchial blocker (Cook Critical Care, Inc, Bloomington, IN), Cohen Flextip Plus endobronchial blocker (Cook Critical Care, Inc), Fogarty vascular embolectomy catheter (Edwards Lifesciences, Irvine, CA), Fuji Univent tube (Fuji Systems Corporation, Tokyo, Japan), Univent torque control blocker (Vitaid Ltd, Lewiston, NY), and the Mallinckrodt double-lumen endobronchial tube (Tyco Healthcare group LP, Pleasanton, CA)^[55, 57]. Case reports have also shown the combination of a laryngeal mask airway (LMA) and bronchial blocker to be successful. In these cases, the aperture bars of the LMA were removed to allow simultaneous fiberoptic bronchoscope and bronchial blocker access through the LMA. The lung isolation options for these patients are listed in Table 19.6 [55, 57].

Conclusion

Patients present to the operating room electively, urgently, and emergently with tracheostomies and for tracheotomy placement. Despite having a secure and definitive airway, patients for tracheotomy placement and those with tracheostomies in situ are medically challenging for both otolaryngologists and anesthesiologists. It is critical that the otolaryngologists and anesthesiologist worked collaboratively to safely and efficiently care for these patients to optimize patient safety and minimize morbidity and mortality. In order to achieve these goals each specialist must have a working knowledge of the tracheotomy procedure including the indication, contraindications, and complications. In addition and perhaps equally important, each specialists must possess an insight and understanding of the unique roll the other specialist brings to the operating room to optimize outcome for patients with and those who need tracheostomies.

Clinical Insights

For the Otolaryngologist (from the Anesthesiologist)

- Thoroughly discuss with the anesthesiologist about the airway management of patients requiring tracheotomy (i.e., endotracheal intubation versus tracheotomy under local anesthesia).
- Patient must be NPO at least 6–8 h prior to elective tracheostomies. The patient should also be hemody-namically stable and have a recent complete blood count.
- Do not use electrocautery when entering the airway, if electrocautery is necessary for intratracheal hemostasis communication with the anesthesiologists must confirm an inspired oxygen less than 30% and that nitrous oxide is not being used. Be aware that airway fires can occur during tracheotomy placement and have safety measures in place.
- Alert the anesthesiologist prior to tracheal incision and tracheotomy tube placement to ensure that the patient is deep and bucking or coughing is minimized.
- Communicate with the anesthesiologist whether there were multiple attempts or difficulty in placing the tracheotomy tube so he/she is aware of potential PACU complications that may arise.
- Have a low threshold to suspecting pneumothorax, pneumomediastinum, and subcutaneous emphysema in susceptible patients. Obtain a portable chest radiograph postoperatively.
- Be vigilant about postoperative bleeding. Until proven otherwise, treat any hemorrhage after 72 h (usually at 1–4 weeks postoperative) as a possible TIF/bleed.

For the Anesthesiologist (from the Otolaryngologist)

- When feasible the anesthesiologist should be present for all tracheostomies in case of cardiopulmonary emergencies.
- Proper communication with the anesthesiologist, in addition to the nursing staff, throughout the procedure is essential and is especially crucial in an impending airway.
- During an emergent tracheotomy, minimal to no anesthetic agents should be used. If that is not possible, then the use of agents with minimal ventilation depression should be used

- The lowest possible oxygen concentration should be used to avoid airway fires from the electrocautery.
- During percutaneous tracheostomies, the anesthesiologist should manage the bronchoscope and endotracheal tube while the surgeon performs the procedure.

Clinical Pearls

For the Otolaryngologist (from the Otolaryngologist)

- Always perform a proper preoperative evaluation of the patient including whether or not the patient requires emergent surgical airway establishment.
- Constant palpation of tracheal landmarks throughout the procedure ensures staying within the midline.
- When feasible divide the thyroid isthmus.
- Use of the cricoid hook and a wide incision is essential for good exposure, especially in those who cannot have their neck flexed.
- A finder needle (a 10 mL syringe half-filled with saline, with an 18 or 19 gauge needle) can be useful in situations where the tracheal architecture is obscured or encased with tumor.
- Before entering the airway, make sure proper instruments and tracheostomy tube are readily available.
- Do not extubate endotracheal tube from patient until the tracheotomy tube is confirmed to be in the airway with adequate tidal volumes, peak pressures, and baseline or improved saturations.

For the Anesthesiologist (from the Anesthesiologist)

- Assess whether or not the patient is a surgical candidate and can tolerate transport to the operating room for an open elective tracheotomy placement.
- Do not remove the endotracheal tube entirely out of the trachea until there is confirmation that ventilation is occurring through the tracheotomy (i.e., positive end tidal CO₂ seen on capnography, easy bag ventilation, bilateral chest rise with bilateral equal breath sounds).
- Decrease FiO₂ as tolerated during the tracheotomy procedure. Know how to identify and manage

possible tracheotomy-related complications i.e., intraoperative airway fires, air embolism, pneumothorax, and hemorrhage.

- Maintain appropriate cuff pressures to minimize the risk of developing late complications such as tracheomalacia and tracheal stenosis.
- Be able to appropriately manage respiratory distress in a patient with tracheotomy. A dislodged tracheotomy tube can result in an airway emergency. Remove the tracheotomy tube and perform endotracheal intubation in patients with new (less than 7 days old) tracheostomies. Reinsert or replace the tracheotomy tube in patients with mature stomas.
- Be able to identify tracheotomy devices and determine whether or not the spontaneously breathing patient can be administered positive pressure ventilation via their in situ device once apneic.
- Have several strategies to convert a tracheotomy device that either has a cuff or a 15 mm adaptor in order to delivery positive pressure ventilation during the induction of anesthesia.

References

- Yu KCY. Airway management and tracheotomy. In: Lalwani AK, editor. Current diagnosis and treatment. 2nd ed. New York: McGraw-Hill; 2008. p. 518.
- Kost KM, Myers EN. Tracheostomy. In: Myers EN, editor. Operative otolaryngology head and neck surgery, vol. 1. 2nd ed. Philadelphia: Saunders Elsevier; 2008. p. 578.
- Morris LL, Sherif Afifi M, editors. Tracheostomies: the complete guide. New York: Springer; 2010.
- Mallick A, Bodenham AR. Tracheostomy in critically ill patients. Eur J Anaesthesiol. 2010;27(8):676–82.
- Groves DS, Durbin CG. Tracheostomy in the critically ill: indications, timing and techniques. Curr Opin Crit Care. 2007;13(1): 90–7.
- Blot F, Nitenberg G, Guiguet M, et al. Safety of tracheostomy in neutropenic patients: a retrospective study of 26 consecutive cases. Intensive Care Med. 1995;21:687–90.
- Kluge S, Meyer A, Kuhnelt P, et al. Percutaneous tracheostomy is safe in patients with sevbere thrombocytopenia. Chest. 2004;126: 547–51.
- American Society of Anesthesiologists Task Force on Management of the Difficult Airway. 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;98:1269–77.
- 9. Durbin CG. Tracheostomy: why, when, and how? Respir Care. 2010;55(8):1056–68.
- Durbin CG. Should tracheostomy be performed as early as 72 hours in patients requiring prolonged mechanical ventilation? Respir Care. 2010;55(1):76–87.
- Loh KS, Irish JC. Traumatic complications of intubation and other airway management procedures. Anesthesiol Clin N Am. 2002; 20(4):953–69.

- McWhorter AJ. Tracheotomy: timing and techniques. Curr Opin Otolaryngol Head Neck Surg. 2003;11(6):473–9.
- Engels PT, Bagshaw SM, Meier M, Brindley PG. Tracheostomy: from insertion to decannulation. Can J Surg. 2009;52(5):427–33.
- DeLeyn P, et al. Tracheostomy: clinical review and guidelines. Eur J Cardiothorac Surg. 2007;32(3):412–21.
- O'Connor HH, White AC. Tracheostomy decannulation. Respir Care. 2010;55(8):1076–81.
- Cotton RT, Manoukian JJ. Glottic and subglottic stenosis. In: Cummings CW, Fredrickson JM, Harker LA, Krause CJ, Schuller DE, editors. Otolaryngology head neck surg, vol. 3. 1st ed. St. Louis: Mosby; 1986. p. 2159–80.
- Allen TH, Steven IM. Prolonged nasotracheal intubation in infants and children. Br J Anaesth. 1972;44:835–40.
- Papisdero MJ, Pashley NRT. Acquired stenosis of the upper airway in neonates: an increasing problem. Ann Otol Rhinol Laryngol. 1980;89:512–4.
- Walner DL, Loewen MS, Kimura RE. Neonatal subglottic stenosis – incidence and trends. Laryngoscope. 2001;111(1):48–51.
- Choi SS, Zalzal GH. Changing trends in neonatal subglottic stenosis. Otolaryngol Head Neck Surg. 2000;122(1):61–3.
- McDonald IH, Stocks JG. Prolonged nasotracheal intubation. Br J Anaesth. 1965;37:161.
- Quiney RE, Gould SJ. Subglottic stenosis: a clinicopathologic study. Clin Otolaryngol. 1985;10:315.
- Ratner I, Whitfield J. Acquired subglottic stenosis in the very lowbirth-weight infant. Am J Dis Child. 1983;137:40.
- Little FB, Koofman JA, Kohut RI, Marshall RB. Effect of gastric acid on the pathogenesis of subglottic stenosis. Ann Otol Rhinol Laryngol. 1985;94:516.
- Wease GL, Frikker M, Villalba M, Glover J. Bedside tracheostomy in the intensive care unit. Arch Surg. 1996;131:552–5.
- Upadhyay A, Maurer J, Turner J, et al. Elective bedside tracheostomy in the intensive care unit. J Am Coll Surg. 1996;183: 51–5.
- Klotz DA, Hengerer AS. Safety of pediatric bedside tracheostomy in the intensive care unit. Arch Otolaryngol Head Neck Surg. 2001;127(8):950–5.
- Stauffer JN, Olson DE, Petty TL. Complications and consequences of endotracheal intubation and tracheostomy: prospective study of 150 critically ill adult patients. Am J Med. 1981;70: 65–76.
- Dayal VS, Masri WE. Tracheostomy in the intensive care setting. Laryngoscope. 1986;96:58–60.
- Altman KW, Waltonen JD, Kern RC. Urgent surgical airway intervention: a 3 year county hospital experience. Laryngoscope. 2005;115:2101–4.
- Berkow LC. Strategies for airway management. Best Pract Res Clin Anaesthesiol. 2004;18(4):531–48.
- Lavery GG, McCloskey BV. The difficult airway in adult critical care. Crit Care Med. 2008;36(7):2163–73.
- Miller RD, editor. Miller's anesthesia. New York: Churchill Livingstone; 2004.
- Harley EH, Collins MD. Neurologic sequelae secondary to atlantoaxial instability in down syndrome. Implications in otolaryngologic surgery. Arch Otolaryngol Head Neck Surg. 1994;120(2):159–65 [Review].
- 35. Kinley CE. A technique of tracheostomy. Can Med Assoc J. 1965;92:79–81.
- Myers EN, Stool SE, Johnson JT. Technique of tracheostomy. In: Myers EN, Stool SE, Johnson JT, editors. Tracheostomy. New York: Churchill Livingstone; 1985. p. 113–24.
- 37. Kost K. Percutaneous tracheostomy. Laryngoscope. 2005;115: 1–30.
- Barba CA, Angood PB, Kauder DR, et al. Bronchoscopic guidance makes percutaneous tracheostomy a safe cost-effective, and easyto-teach procedure. Surgery. 1995;118:879–83.

- Dierks EJ. Tracheostomy: elective and emergent. Oral Maxillofac Surg Clin North Am. 2008;20(3):513–20.
- Linstedt U, Zenz M, Krull K, Hager D, Prengel AW. Laryngeal mask airway or endotracheal tube for percutaneous dilatational tracheostomy: a comparison of visibility of intratracheal structures. Anesth Analg. 2010;110(4):1076–82.
- 41. Mandel JE. Laryngeal mask airways in ear, nose and throat procedures. Anesthesiol Clin. 2010;28(3):469–83.
- 42. David MD, De Marchi L. Dexmedetomidine sedation for awake tracheostomy: case report and literature review. J Clin Anesth. 2010;22(5):360–2.
- Bergese SD, Khabiri B, Roberts WD, Howie MB, McSweeney TD, Gerhardt MA. Dexmedetomidine for conscious sedation in difficult awake fiberoptic intubation cases. J Clin Anesth. 2007;19(2): 141–4.
- Abdelmalak B, Makary L, Hoban J, Doyle DJ. Dexmedetomidine as sole sedative for awake intubation in management of critical airway. J Clin Anesth. 2007;19(5):370–3.
- 45. Epstein SK. Late complications of tracheostomy. Respir Care. 2005;50(4):542–9.
- Davis GM. Tracheostomy in children. Paediatr Respir Rev. 2006;7 Suppl 1:S206–9.
- 47. Rogers ML, Nickalls RW, Brackenbury ET, Salama FD, Beattie MG, Perks AG. Airway fire during tracheostomy: prevention strategies for surgeons and anaesthetists. Ann R Coll SUrg Engl. 2001;83(6):376–80.

- 48. Lim HJ, Miller GM, Rainbird A. Airway fire during elective tracheostomy. Anesth Intensive Care. 1997;25(2):150–2.
- Rogers SA, Mills KG, Tufail Z. Airway fire due to diathermy during tracheostomy in an intensive care patient. Anaesthesia. 2001;56(5):441–3.
- Niskanen M, Purhonen S, Koljonen V, Ronkainen A, Hirvonen E. Fatal inhalation injury caused by airway fire during tracheostomy. Acta Anaesthesiol Scand. 2007;51(4):509–13.
- Sheinbein DS, Loeb RG. Laser surgery and fire hazards in ear, nose, and throat surgeries. Anesthesiol Clin. 2010;28(3): 485–96.
- Hess DR. Tracheostomy tubes and related appliances. Respir Care. 2005;50(4):497–510.
- 53. Bradley PJ. Bleeding around a tracheostomy wound: what to consider and what to do? J Laryngol Otol. 2009;123(9):952–6.
- Yaremchuk K. Regular tracheostomy tube changes to pre-vent formation of granulation tissue. Laryngoscope. 2003;113:1–10.
- Campos JH. Lung Isolation techniques for patients with difficult airway. Curr Opin Anaesthesiol. 2010;23(1):12–7.
- Fikkers BG, et al. Emphysema and pneumothorax after percutaneous tracheostomy: case reports and an anatomic study. Chest. 2004;125(5):1805–14.
- 57. Robinson 3rd AR, Gravenstein N, Alomar-Melero E, Peng YG. Lung isolation using a laryngeal mask airway and a bronchial blocker in a patient with a recent tracheostomy. J Cardiothorac Vasc Anesth. 2008;22(6):883–6.

Pediatric Otolaryngology

20

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Introduction

Otolaryngologic procedures are commonly performed on children. In fact, pressure equalizing tube placement (ear tubes) and adenotonsillectomy are among the most frequent surgical interventions in the pediatric population. Therefore, every anesthesiologist who manages children undergoing otolaryngologic procedures must be familiar with the special implications of sharing the pediatric airway with an otolaryngologist working in the head and neck region. In addition, it is imperative to be skilled in the challenges of compassionately yet safely managing anxious young patients and their parents from the time of preoperative assessment until discharge from the post anesthesia care unit.

While there are few anesthetic challenges as potentially serious as the emergency management of a compromised airway in a small infant, even general otolaryngologists and anesthesiologists need to develop the skills unique to pediatric practice. In addition, the presence of congenital anomalies that alter the architecture of the upper aerodigestive tract poses unique challenges in airway management. The principles outlined in this chapter will serve as a valuable tool in understanding the coordinated effort that is needed to manage the airway in this delicate population of patients.

General Considerations

There are general considerations for every pediatric otolaryngologic patient. These procedures involve the oral cavity, pharynx, larynx, tracheobronchial tree or structures in

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M.A. Rothschild (🖂) Department of Otolaryngology – Head and Neck Surgery, Icahn School of Medicine at Mount Sinai, Box 1189, One Gustave L. Levy Place, New York, NY 10029, USA e-mail: doctormike@kidsent.com close proximity to the airway such as the ear and neck. Therefore, close communication between the anesthesiologist and otolaryngologist throughout the procedure is of paramount importance.

Head and neck procedures often require turning of the head for the procedure and/or the operating table 90° or 180°. This renders the endotracheal tube (ETT) relatively inaccessible to the anesthesiologist. Extra care should be taken to ensure that the tracheal tube is securely fastened and that there are no kinks. The anesthesia circuit should be secured; however, it should not be anchored tightly to the patient or the operating room table since the otolaryngologist may move the head during the procedure which may result in inadvertent extubation or mainstem intubation. Also, decisions regarding the placement (e.g., nasal vs. oral, left-side vs. right-side), and the type (e.g., oral RAE, named after the inventors Ring, Adair & Elwyn, nasotracheal) of ETT are primarily dictated by patient considerations, the surgical procedure, and the otolaryngologist's preference. Therefore, a discussion between the anesthesiologist and the otolaryngologist should take place prior to the induction of anesthesia.

The Pediatric Patient: Unique Considerations

Pediatric Airway Anatomy

Anatomically, the pediatric airway is significantly different than the adult airway. This is most dramatic in the infant, becoming less so as the child ages and matures. By age 8–10, the larynx has developed into a configuration close to that in an adult. Marked differences are seen in the infant's head shape, tongue, hypopharynx, and larynx that impact on anesthetic management of these patients (Table 20.1).

In infants, the occiput is large obviating the need to place a pillow under the head to achieve the optimal intubating position ("sniffing") as in the adult. In fact, the large occiput

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Difference	Implication
Large occiput	Shoulder roll
Relative large tongue	Difficult mask ventilation and laryngoscopy
Epiglottis long, omega shaped, floppy, and angled away from the axis of the trachea	Straight laryngoscope blade
Larynx cephalad (C3-4)	Straight laryngoscope blade
Vocal cords anterior attachment more cephalad than posterior	ETT caught by anterior commissure of vocal cords
Cricoid ring narrowest area	Tight ETT may lead to

Table 20.1 Anatomic airway differences and their anesthetic implications

ETT endotracheal tube

may result in excessive flexion of the neck that necessitates placement of a shoulder role to provide adequate visualization of the larynx. The tongue is large in comparison with the oral cavity making mask ventilation and laryngoscopy more difficult. The epiglottis is relatively long, omega shaped, floppy, and angled away from the axis of the trachea. This makes visualization of the glottic opening difficult with a curved laryngoscope blade placed in the vallecula. Therefore, visualization of the glottic opening is more easily achieved with a straight rather than curved laryngoscope blade. The infant larynx is more cephalad than the adult larynx, lying at the C3-4 level in contrast to the C4-5 level of the adult larynx. In preterm infants, the larynx is even more cephalad, at the C3 level. The more cephalad position of the larvnx makes the straight laryngoscope blade more effective for visualizing the airway than a curved blade.

The anterior attachment of the vocal cords is more caudad than the posterior attachment, in contrast to the adult vocal cords that are perpendicular to the tracheal axis. This can lead to difficulty with tracheal intubation because there is a tendency for the ETT to be caught by the anterior commissure of the vocal cords or the anterior subglottis.

The narrowest portion of a child's airway is in the subglottis at the cricoid cartilage level, as opposed to the glottic opening in the adult. Clinically, this can result in the ETT passing easily through the vocal cords but fitting tightly in the subglottic area. Since the cricoid cartilage forms a complete ring that is not expandable, a snug ETT can compress the tracheal mucosa ^[1], possibly leading to laryngeal edema, post-extubation stridor or even residual stenosis. Based on this, the traditional teaching is that uncuffed ETTs should be used in the pediatric patient until the age of 8–10 years. The appropriate sized tube is determined by age ^[2] (Table 20.2).

It is important to confirm the presence of a leak around the ETT once intubation is accomplished and the position is Table 20.2 Uncuffed and cuffed ETT size based on age

Chedhed and curred ETT size based on age			
	Uncuffed ETT size	Cuffed ETT size	
Age	(ID mm)	(ID mm)	
0–3 months	3.0	3.0 uncuffed	
3–8 months	3.5	3.0	
8-15 months	4.0	3.5	
15-24 months	4.5	4.0	
2 years and older	(16+age in years)/4	16+age in years/3.5	
ID internal diameter			

ID internal diameter

confirmed by the presence of end tidal carbon dioxide (ETCO_2) and auscultation of bilateral breath sounds. Since the adult tracheal mucosal capillary pressure is considered to be 30 cm H₂O, the leak should be less than 30 cm H₂O but above the peak inflation pressure (PIP) generated during positive pressure ventilation. The leak is determined by listening over the patient's mouth while the PIP slowly rises by incremental closure of the adjustable pressure-limiting (APL) valve. The pressure at which a leak is first heard determines how snug the fit is. In some instances, the "perfect" sized uncuffed ETT cannot be found and either a "tight" fit must be tolerated or a large leak is compensated by the use of high fresh gas flows.

Over the past two decades, there has been increasing evidence that the use of cuffed ETTs in the pediatric population is not associated with an increased incidence of post-extubation stridor and may in fact be beneficial ^[3–5]. A cuffed ETT allows for adjustment of the leak around the ETT precluding the need for multiple laryngoscopies in an attempt to find the right size uncuffed ETT that allows for adequate ventilation but that is not too "tight." Multiple laryngoscopies in and of itself may lead to an increased incidence of post-extubation stridor.

Other advantages of cuffed ETTs are decreased operating room contamination with anesthetic gases, decreased fresh gas flows ^[4], more accurate respiratory parameter measurements (e.g., tidal volume, ETCO_2), and decreased risk of pulmonary aspiration ^[6]. Also, in patients whose pulmonary compliance may change during the surgical procedure (e.g., laparoscopy) or during the postoperative period (e.g., pulmonary edema), a cuffed ETT allows for adjustment of the leak as the need arises without compromising patient safety (Table 20.3).

Because the presence of a cuff increases the outer diameter of the ETT, the ETT placed should be one half size smaller than the calculated uncuffed ETT for that patient. For example, if a 5.0 uncuffed ETT is calculated, a 4.5 cuffed ETT should be placed (Table 20.2). The presence for a leak should be ascertained as described above and the cuff adjusted accordingly.

Uncuffed ETT		Cuffed ETT	
Advantages	Disadvantages	Advantages	Disadvantages
Largest internal diameter • ↓Airway resistance • ↓Work of breathing	Multiple laryngoscopiesTrauma to larynxPost-extubation stridor	Limited laryngoscopies ↓Trauma to larynx RSI 	 Smaller internal diameter ↑Airway resistance ↑Work of breathing
Easier to suction secretions	 Unable to adjust leak Need to reintubate when patient critically ill Unable to accommodate changes in pulmonary compliance intraoperatively 	Ability to adjust leak during changes in pulmonary complianceLaparoscopyARDS	Difficult to suction secretions because of smaller size
Less mucosal damage	Pulmonary aspiration risk	↓Pulmonary aspiration risk	Mucosal damage from cuff • Post-extubation stridor
	May need higher FGF	↓FGF	
	OR pollution	↓OR pollution	
	Inaccurate ETCO_2 and TV measurements	\uparrow Reliability of ETCO ₂ and TV measurements	

Table 20.3 Advantages and disadvantages of uncuffed versus cuffed ETT

ETT endotracheal tube, *RSI* rapid sequence induction, *FGF* fresh gas flow, *OR* operating room, *ETCO*₂ end tidal carbon dioxide, *TV* tidal volume, *ARDS* adult respiratory distress syndrome

Additional evidence to support the use of cuffed ETTs is that the cricoid ring is not circular but ellipsoid, with the transverse diameter being greater than the anterior to posterior diameter ^[7]. Thus, a round uncuffed ETT may place excessive pressure on the mucosa anteriorly and posteriorly when trying to occlude a leak that is caused by the larger transverse diameter. A cuffed ETT seals the airway at the tracheal level allowing for a more even pressure distribution when inflating the cuff.

Tracheal length varies with age, with infants being 5–9 cm ^[8, 9]. Appropriate positioning of the ETT is crucial to avoid endobronchial intubation or, if using a cuffed ETT, to avoid herniation of the cuff through the vocal cords. Due to the short length of the trachea in infants, there is less room for error. As measured at the alveolar ridge, appropriate insertion distance is as follows: 10 cm for a newborn, 11 cm for a 1 year old, and 12 cm for 2 years old ^[10]. The formula 12 + age/2 ^[11] or 13 + age/2 ^[12] is used for children older than 2 years. Of course, auscultation for equal bilateral breath sounds should be the final determinant of appropriate ETT position. Flexion of the head displaces the ETT distally whereas head extension displaces the ETT proximally possibly resulting in accidental extubation [9, 13]. Placement of a tongue depressor in the mouth causes distal displacement of the ETT as well^[13]. During otolaryngologic procedures, head extension commonly occurs, particularly during adenotonsillectomy, which may displace the ETT proximally. Placement of a mouth retractor may displace the ETT distally. Therefore, after positioning for adenotonsillectomy, position of the ETT should be reconfirmed.

The most common type of laryngoscopy blade used in the pediatric population is the straight blade for reasons described

above. A Miller 0 blade is usually used for preterm and full-term neonates. A Miller 1 blade is usually used for neonates up to 18 months of age. A Wis-Hipple 1.5 blade is used for patients 18 months to approximately 6 years of age. Thereafter, a Miller 2 blade is appropriate. A curved blade (Macintosh) may be used in children 2 years and older. It is always a good idea to prepare multiple size laryngoscope blades since not all pediatric patients are the same size despite being the same age.

Pediatric Physiology (Table 20.4)

Respiratory

Closing volume (CV) is increased in infants and is within resting tidal volume ^[14]. CV is defined as that lung volume at which small airways begin to close. This results in alveoli distal to the airway closure to collapse by absorption atelectasis causing shunt and hypoxia. For this reason, an infant's oxygen saturation decreases rapidly when they breath hold, cough, or "buck" with an ETT in place.

Chest wall compliance is increased in the infant because of predominantly cartilaginous ribs. During periods of increased negative pressure in the thorax, the chest wall collapses to a greater degree than in the older child resulting in an increased work of breathing and decreased ventilation. This is even more evident when airway obstruction exists. Furthermore, the intercostal and diaphragmatic muscles are immature. There are two types of muscle fibers, Type I and Type II. Type I muscle fibers consist of slow-twitch, high-oxidative fibers

Table 20.4 Physiologic changes and their anesthetic implications				
Physiologic change	Clinical implications	Anesthetic implication		
↑CV	Within tidal volume Absorption atelectasis and shunt	Oxygen desaturation with breath holding and "bucking" on ETT		
↑Chest wall compliance	Collapse of chest wall with negative intrathoracic pressure during airway obstruction	Intubation and mechanical ventilation under general anesthesia		
↓Type I muscle fibers	Respiratory fatigue with persistent airway obstruction	Intubation and mechanical ventilation under general anesthesia		
$\uparrow \dot{V}_{A}$	Increased respiratory rate	Quicker inhalation induction		
↑Ÿ _A /FRC	Low oxygen reserve	Rapid oxygen desaturation during airway obstruction or apnea		
↓Cardiac muscle compliance	Stroke volume fixed Cardiac output heart rate dependent	Bradycardia not well tolerated Anticholinergic administration		
Immature sympathetic system	Bradycardia with vagal stimulation and hypoxia	Anticholinergic administration		

CV closing volume, ETT endotracheal tube, \dot{V}_A alveolar ventilation, FRC functional residual capacity

	Infant (4 kg)	3 YO (15 kg)	5 YO (18 kg)	Adult (70 kg)
\dot{V}_{A}	600 150 mL/kg	1,755 117 mL/kg	1,800 100 mL/kg	4,200 60 mL/kg
FRC (mL)	120	490	680	2,800
॑ V _A /FRC	5/1	3.5/1	2.6/1	1.5/1
\dot{VO}_2 (mL/kg/min)	6–8	4–6	46	3–4

Table 20.5	V	/FRC at	various	ages
	Υ.	/I IC at	various	agua

 \dot{V}_{A} alveolar ventilation, FRC functional residual capacity; $\dot{V}O_{2}$ oxygen consumption

and are important for sustained activity. Type II muscle fibers consist of fast-twitch, low-oxidative fibers, and are important for short bursts of increased activity. The diaphragm in the infant is composed of 25% Type I muscle fibers in contrast to the adult that has 55% Type I muscle fibers. The infant's intercostal muscles are composed of 45% of Type I muscle fibers whereas in the adult there are 65% Type I muscle fibers. This places the infant at risk for respiratory fatigue when challenged with airway obstruction for a prolonged period of time. The adult composition of muscle fibers is achieved by 1 year of age ^[15]. For these reasons, decreased chest wall compliance and decreased presence of Type I muscle fibers, it is recommended that infants less than 12 months of age should be tracheally intubated and mechanically ventilated during general anesthesia except for possibly a short procedure lasting less than 1 h.

Alveolar ventilation (\dot{V}_A) in the infant is two to three times the adult value and is due to an increased respiratory rate since TV on a per kilogram basis (7 mL/kg) is the same in the infant and the adult. This accounts for the more rapid onset of general anesthesia during an inhalation induction in the infant when compared to an adult.

Young children and especially infants are particularly susceptible to rapid oxygen desaturation with even brief periods of airway obstruction. This is because of their increased oxygen consumption and increased ratio of alveolar ventilation (\dot{V}_A) to functional residual capacity (FRC) or apneic oxygen reserve ^[14]. The \dot{V}_A /FRC ratio can be as high as 5:1, as compared to the adult value of 1.5:1. (Table 20.5)

Cardiovascular

Cardiac output is increased in the infant to meet the increased oxygen demand. Cardiac output in the neonate is approximately 350 mL/kg/min and in the infant 150 mL/kg/min as compared to the adult value of 75 mL/kg/min. The heart of the infant is less compliant than the adult heart because of a decrease in the contractile muscle mass ^[16]. Because of this, the stroke volume is relatively fixed and the infant cannot compensate for a decreased cardiac output by increasing stroke volume. Therefore, the infant's cardiac output is heart rate dependent and bradycardia should be treated expeditiously.

The infant is considered "vagotonic" because of an immature sympathetic system. Therefore, vagal stimulation, such as during laryngoscopy, and hypoxia will result in significant bradycardia that in turn will result in a decreased cardiac

Table 20.6 Indications for anticholinergic medication

- 1. Age <6 months
- 2. Development of bradycardia
- 3. History of bradycardia during induction or intubation
- 4. Procedures inducing the vagal reflex (e.g., oculocardiac reflex)
- 5. Succinylcholine
- 6. High-dose opioids
- 7. Ketamine
- 8. Excessive secretions
- 9. Oral surgery
- 10. Position other than supine

output. Also, succinylcholine has been described to cause bradycardia with the first dose. In the past, most pediatric patients arrived to the operating room significantly sedated. When these sedated patients were induced with halothane, the induction agent of choice before sevoflurane, significant bradycardia and hypotension occurred. For this reason, many anesthesiologists routinely administered anticholinergic premedication, either atropine or glycopyrrolate, prior to or during induction. This practice has changed over the last two decades ^[17, 18] because of the use of newer anesthetics that do not cause excessive secretions or bradycardia. In addition, although premedication is still used, the depth of sedation is much less than it was in the past and bradycardia is rarely seen on induction of general anesthesia. Even the premise that succinylcholine causes significant bradycardia with the first dose has been challenged [19]. Indications for anticholinergic premedication are listed in Table 20.6.

The dose of atropine depends on the age of the child. A minimum dose of 0.1 mg has been advocated because of the theoretical concern of bradycardia occurring if a smaller dose is administered. However, this would be a massive dose on a per kg basis if given to a preterm infant and has resulted in tachycardia greater than 200 for a prolonged period of time. The current recommendation for atropine dosing is as follows:

<5 kg: 20 mcg/kg

 \geq 5 kg: 10–20 mcg/kg with a minimum dose of 0.1 mg

Laryngospasm

Laryngospasm is more common in the pediatric patient ^[20], especially in the patient with a URI or recent URI ^[21], use of a laryngeal mask airway (LMA) ^[22], and after surgical procedures involving manipulation of the airway, particularly adenotonsillectomy ^[23]. Stimulation of the larynx, either by laryngoscopy or secretions, during "light" anesthesia may result in either partial or complete laryngospasm ^[24]. Both partial and complete laryngospasm share the same clinical

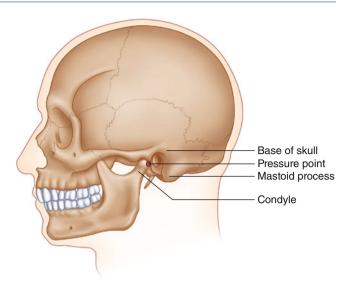


Fig. 20.1 Laryngospasm notch

signs of tracheal tug and paradoxical movement of the chest and abdomen. They are differentiated by the presence of a high-pitched inspiratory sound in partial laryngospasm versus the absence of respiratory sounds in complete laryngospasm ^[25]. If not treated expeditiously, rapid oxygen desaturation and bradycardia will ensue. Therefore, atropine and succinylcholine should always be available, in both intravenous (IV) and intramuscular (IM) doses, when caring for these patients. Treatment of partial laryngospasm consists of removing the noxious stimulus (surgical stimulus, secretions), applying positive pressure to the airway with 100% oxygen, and deepening the anesthetic with either a volatile agent or propofol (0.5 mg/kg) ^[26]. If these maneuvers fail, succinylcholine, 1-2 mg/kg IV or 4 mg/kg IM, should be administered. Atropine, 20 mcg/kg IV/IM, should be administered if hypoxia and bradycardia occurs ^[27]. Complete laryngospasm rarely resolves with positive pressure alone and a jaw thrust maneuver should be applied ^[27]. Alternatively, pressure on the laryngospasm notch (Fig. 20.1) has been shown to cause relaxation of the vocal cords. The laryngospasm notch is located behind the ear in the area posterior to the ascending ramus of the mandible, anterior to the mastoid process, and caudad to the base of the skull. Pain caused by pressure in this area results in afferent input to the vocal cords causing them to relax ^[28, 29].

Upper Respiratory Infection

Upper respiratory infections (URI) are very common in children, especially during the winter months. The decision whether to proceed with elective surgery as scheduled or to postpone the procedure still remains difficult and controversial. There is often considerable pressure to proceed in less than optimal circumstances for nonmedical reasons (such as parent's work obligations or a child's school calendar). While not every minor cold necessarily precludes a safe anesthetic, one must never compromise the patient's safety out of deference to convenience.

URI in the pediatric patient undergoing general anesthesia is associated with an increased risk of laryngospasm, bronchospasm, secretions clogging the ETT, post-extubation stridor, and desaturation in the post-anesthesia care unit (PACU) requiring supplemental oxygen ^[30, 31]. Although these complications are real, multiple studies have shown that they do not result in significant morbidity when identified and treated appropriately ^[30, 32, 33]. Risk factors associated with increased incidence of adverse respiratory events are listed in Table 20.7 ^[30]. The type of procedure, necessity of tracheal intubation, and the general health of the child preoperatively should all be considered in deciding whether to delay or proceed with elective surgery. A reasonable approach to the child with an URI is summarized in Fig. 20.2.

If surgery is postponed because of an active or recent URI, considerations for rescheduling should be made to minimize the risk of the aforementioned complications.

 Table 20.7
 Risk factors associated with increased incidence of adverse respiratory events in the child with a URI [30]

- 1. Copious secretions
- 2. Nasal congestion
- 3. Use of ETT in children <5 years of age
- 4. History of prematurity
- 5. History of reactive airway disease
- 6. Parental smoking
- 7. Surgery involving the airway
- URI upper respiratory infection, ETT endotracheal tube

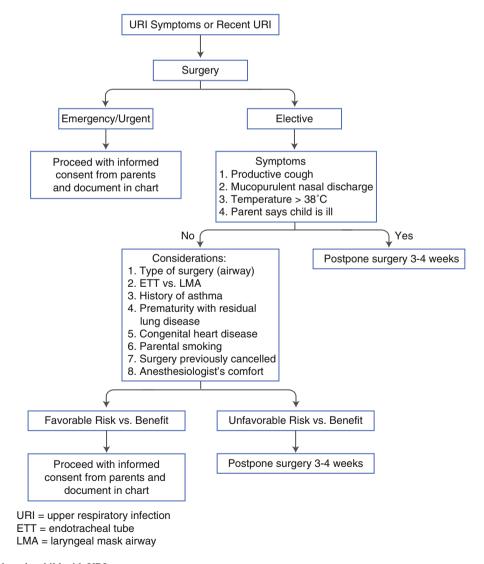


Fig. 20.2 Approach to the child with URI symptoms

The literature has shown that despite symptom resolution, the airways remain hyperreactive even 4–6 weeks after an URI ^[34, 35]. Due to the frequency with which pediatric patients have URIs, waiting such a long interval to proceed with surgery may not be feasible or practical. It is the authors' opinion that postponing surgery for 3–4 weeks is a reasonable option. If the anesthesiologist and otolaryngologist agree to proceed with surgery, the parents must be informed of the potential risks and benefits of proceeding with surgery. The parents must be active participants in the decision and the discussed plan documented in the medical record. Although a more in-depth discussion of this issue is beyond the scope of this chapter, a good review is available ^[36].

Malignant Hyperthermia

Malignant hyperthermia is a pharmacogenetic disorder of skeletal muscles that is triggered by potent inhalation agents and/or succinylcholine^[37]. This hypermetabolic syndrome is the result of a massive release of calcium in the muscle that causes sustained muscle contractures. The incidence of MH varies but what is clear is that there is a higher incidence of MH in the pediatric versus adult population ^[38]. However, the incidence of MH may be decreasing because of the decreased routine use of succinylcholine in the pediatric population secondary to reports of hyperkalemic arrest in patients with undiagnosed muscular disease [39]. Therefore, any anesthesiologist caring for the pediatric patient should be aware of the clinical manifestations of MH and know how to diagnose and treat it. Additionally, obtaining a prior history of MH, in the patient and family, should be part of every preoperative evaluation so that appropriate preparation can be made prior to the initiation of any anesthetic. The reader is referred to the Malignant Hyperthermia Association of the United States website (www.mhaus.org) and to other major pediatric texts for an extensive review of this disorder as well as how to manage these patients.

Preoperative Preparation

The induction of general anesthesia and the anticipation of surgery are both extremely anxiety provoking, both for children and their parents. One of the more difficult skills to master is the appropriate tone to take in the perioperative period. In addition, different families require different approaches, and it takes an experienced practitioner to determine whether to be serious, jocular, authoritarian, or deferential in order to put patients and their parents at ease. Unfortunately, there is little that we can add to this chapter that will supplant experience.

Our approach to minimizing perioperative stress for our patients and their families involves a multimodal, educational

program that starts before the day of surgery and includes written material, phone interviews, and on-site visitation. In addition to providing handouts that cover frequently asked questions about anesthesia (Appendix A), telephone access to members of the pediatric anesthesia team is made available since many questions are best answered before the stressful morning of surgery. Another extremely successful intervention offered by the child life service at our institution is the "Meet Me at Mt. Sinai" program that includes a pediatric nurse practitioner, pediatric social worker, and pediatric child life specialist. A developmentally appropriate program is designed based on the needs of the individual pediatric patient and includes a tour of the hospital, a video of a typical induction of general anesthesia, and introduction to some of the anesthetic equipment (e.g., face mask). Younger children are provided with a medical kit and a doll they can "doctor" to play out their upcoming surgical experience. While this program may not be appropriate for all children, many older children find the experience interesting and extremely effective in addressing anxiety ahead of time. This program has received very favorable feedback from patients and their families.

Premedication

Premedication can be administered to decrease preoperative anxiety. However, it should not be routinely used in all children but rather a judicious decision made based on the child's age, degree of preoperative anxiety, duration of surgery, and possible delay in discharge from the PACU. Other factors such as availability of appropriate monitoring in the preoperative area and the presence of comorbidities (e.g., airway compromise) must be taken into consideration as well. Parental request for premedication should be granted if possible. Children who are difficult to control preoperatively, particularly those with developmental delays and behavioral disorders, may benefit from premedication.

Midazolam is commonly used for premedication ^[40] through a variety of routes ^[41–43]. However, concern has been raised about its potential for neurotoxicity when administered intranasally since there is a direct communication from the nasal mucosa to the central nervous system via the cribiform plate ^[44, 45] Therefore, when administering midazolam via the intranasal route, the preservative-free formulation should be used. The same applies to intranasal ketamine.

Ketamine as a premedication is usually reserved for the difficult, uncooperative patient, particularly those with severe developmental and behavioral disorders. Although it may be administered via the oral or intranasal route, this is often difficult in the uncooperative and combative patient. In these cases, the intramuscular route becomes the only available option. Side effects of ketamine include excessive oral secretions and postoperative hallucinations and nightmares ^[46]. For these reasons, an antisialagogue, atropine or

Commonly used premedications				
			Onset	Duration
Medication	Dose (mg/kg)	Route	(min)	(min)
Midazolam	0.5–0.75 0.2	Oral Intranasal	15–30 10–15	60–90 60
Ketamine	3–6 3–5	Oral Intramuscular	10–20 5	30–90 30–60

Table 20.8 Commonly used premedications

glycopyrrolate^[47], and midazolam should be administered to counteract these possible side effects^[46].

An extensive review of pharmacologic agents available and their various routes for administration is not possible in this chapter and the reader is referred to major pediatric anesthesia texts for more detailed information. The most common pharmacologic agents and their routes are listed in Table 20.8.

Finally, the question often comes up regarding the presence of a parent in the operating room during induction. This is frequently requested, and we have developed guidelines balancing the potential positive and negative effects on anxiety for the patient and parent, as well as, our concern about patient safety. For children over 9 months of age, we allow one parent to be present during induction. While parents of younger children may also request this, the potential for a difficult induction as well as the lack of separation anxiety in very young infants leads us to deny this request in most circumstances. Furthermore, the presence of two parents often introduces new variables into an already stressful situation, and it is not uncommon for underlying unfavorable interpersonal dynamics to become evident. Parents with any condition that could put them at risk for syncope or the need for attention, such as extreme anxiety, unstable cardiac disease, or pregnancy, are also excluded from the operating room.

Anesthesia Induction

Preparation for the administration of an anesthetic is of paramount importance to the safety of your patient. Checking the anesthesia machine and preparing appropriate sized equipment should be performed prior to every anesthetic. Monitoring of the pediatric patient is in line with the standards set by the American Society of Anesthesiologists. The only caveat is that appropriate sized electrocardiogram pads, pulse oximeter probes, and blood pressure cuffs should be available. Although every effort should be made to apply these monitors prior to induction of anesthesia, this is not always possible. The combative, uncooperative child may not allow for placement of these monitors, or if placed remove them. Of all the monitors, the pulse oximeter probe provides the most valuable information, oxygen saturation and heart

Table 20.9 Comparison of halothane and sevoflurane MAC values

Halothane		Sevoflurane	
Age	MAC	Age	MAC
Neonate	0.87%	<6 months	3.3%
1-6 months	1.2%	1-10 years	2.5%
>1 year	1%	>10 years	2-2.5%
Adult	0.87%	Adult	2%

rate, and all efforts should be made to at least place this monitor prior to induction. In the rare circumstance when this is not possible, induction of anesthesia can proceed but the pulse oximeter probe should be placed as soon as possible. A precordial stethoscope is beneficial as well by providing the ability to assess breath and heart sounds continuously.

The most common method of induction in the pediatric patient is an inhalation induction by facemask with a nonpungent volatile agent. Sevoflurane has nearly replaced halothane because of its faster onset and its favorable cardiovascular profile ^[48]. The minimum alveolar concentration (MAC) of the volatile agents varies with age. Comparison of the MACs of halothane and sevoflurane is in Table 20.9 ^[48–50].

As the anesthetic depth deepens, there may be a period of excitation and agitation prior to entering the surgical plane of anesthesia. This period, Stage 2, is manifested by increased muscle tone, divergent eyes, large pupils, increased heart rate, increased respiratory rate, and at times difficulty in maintaining a patent airway. It is during this stage that laryngospasm is likely to occur, particularly if the patient is stimulated such as during IV placement. The surgical team should not be examining the patient at this time as well. Therefore, it is beneficial to quickly advance to Stage 3 (surgical plane). Maintaining a high concentration of sevoflurane until this stage of anesthesia is bypassed will decrease the time spent in Stage 2. These authors do not turn down the sevoflurane concentration until a decrease in the heart rate is seen heralding the onset of Stage 3. The patient should not be stimulated by anyone until the anesthesiologist has determined that the patient is at an appropriate depth of anesthesia.

The Difficult Airway

Before proceeding to specific recommendations for individual otolaryngologic procedures, a few words are appropriate about the pediatric "difficult airway." This is a term that is poorly defined, and often misunderstood in our experience. It is frequently used to describe a patient in whom previous attempts at elective endotracheal intubation have been problematic or unsuccessful, or who by physical examination is noted to have anatomic features that would predict difficult mask ventilation and/or intubation. Physical findings that would suggest difficulty are large tongue, small mouth opening, micrognathia, cervical spine abnormalities, and congenital anomalies (e.g., Goldenhar) involving the airway. The best way to think critically about a patient with a difficult airway, and to prepare for intubation, is to realize that there are two main categories of this patient class—children in whom it is difficult to pass the endotracheal tube *through* the glottis (group I), and children in whom it is difficult to maneuver the endotracheal tube tip *to* the glottis (group II).

Group I includes primarily glottic and subglottic obstructive lesions—laryngeal webs, foreign bodies, papilloma, subglottic stenosis, or intraluminal mass lesions (e.g., hemangiomas or cysts). Although these conditions are described elsewhere in this volume, a review of the techniques of airway surgery used to address them is beyond the scope of this chapter.

Group II includes patients with craniofacial malformations, especially those involving micrognathia: the Robin anomalad, Treacher–Collins syndrome, or Trisomy 18. Another set of conditions that may limit exposure of the larynx include macroglossia (e.g., Beckwith–Wiedermann syndrome, Down syndrome), and other situations in which soft tissue is obscuring the approach to the airway (e.g., hypopharyngeal cysts or tumors). Patients with trismus (e.g., temporomandibular joint ankylosis) also does not allow for standard direct laryngoscopy intubation techniques.

These patients are a challenge for the otolaryngology-anesthesiology team. Before induction of anesthesia, a full range of primary and backup plans for securing the airway must be planned and agreed upon, with all appropriate equipment immediately available. Consideration should even be given to a temporary tracheotomy done under local anesthesia if orotracheal intubation is not feasible. Even if this is not the primary plan, and other approaches to intubation are preferred, an open tracheotomy set must be in the room in case intubation is unsuccessful and ventilation by facemask becomes impossible during the administration of the anesthetic.

Many options exist for managing the difficult airway and include flexible fiberoptic intubation, Bullard laryngoscope ^[51], videolaryngoscope ^[52], lighted stylet ^[53], LMA ^[54], and retrograde wire-guided intubation ^[55]. It is not feasible to be an expert in all these techniques but rather the anesthesiologist should become very familiar and comfortable with one or two of them. The most commonly used options are the flexible fiberoptic bronchoscope, videolaryngoscope, and LMA.

The advent of small caliber flexible bronchoscopes, over which an endotracheal tube may be threaded, has been a great advance in the management of group II difficult airways. Flexible fiberoptic intubation is the gold standard; however, its use in difficult airways requires previous extensive practice in the normal airway and is not easily mastered. Careful advancement of the endoscope is key, as is the avoidance of airway trauma that can lead to hematoma formation, edema or mucosal hemorrhage. The use of adequate preoxygenation and topical anesthetics in the airway will facilitate this procedure. As always, there is no substitute for a thorough familiarity with the anatomy of the pharynx—it is easy to get disoriented when passing a small endoscope (limited field of view) through this space. The operator must identify known landmarks, maintain the correct orientation of the video feed, and keep the scope tip clear of obstructing secretions.

In the adult patient, flexible fiberoptic intubation is usually performed while the patient is awake. This option is often not possible in the uncooperative awake pediatric patient. In these patients, sedation or anesthesia is required but with great care to maintain spontaneous ventilation. This can be accomplished either with a potent inhalation agent and 100% oxygen or judicious use of intravenous agents. Remifentanil infusion can provide adequate intubating conditions ^[56] and is easily reversed in case of significant respiratory depression. Propofol should be used with extreme caution in that it may cause cessation of spontaneous ventilation or potentiate respiratory depression when used in combination with opioids. Dexmedetomidine may be useful in this situation as well [57, 58]. Muscle relaxants should never be administered unless manual control of the airway is assured. Caution should be exercised, however, since at times muscle relaxation can make an easily ventilated airway impossible.

Another good option for patients in which exposure of the glottis is compromised is the videolaryngoscope, a modified intubating laryngoscope with a fiberoptic video feed embedded in the tongue blade. Although the use of a videolaryngoscope should first be learned on normal airways, the learning curve is steep and makes this modality a popular one.

The LMA is advantageous in situations when ventilation and/or intubation are extremely difficult ^[54, 59] and as a conduit for intubation ^[60-62]. Once the LMA is situated, a flexible fiberoptic scope is placed through the LMA into the trachea over which an ETT is advanced into the airway. The difficulty with this technique arises during withdrawal of the LMA causing the ETT to withdraw as well. Different solutions for this problem have been proposed ^[63-66] with no one solution being superior. These include leaving the LMA in place, splitting the LMA, shortening the LMA, and using longer ETTs.

Although the rigid bronchoscope can be very useful in securing an airway in which there is glottic or subglottic obstruction, it is less applicable to group II patients who generally lack a straight path from mouth to glottis.

Finally, it must be kept in mind that in cases of elective surgery in group II patients, the option always remains of aborting the procedure and trying again in the future, especially if more experienced personnel will be available at another time. While multiple trips to the OR are typically discouraged by parents and physicians alike, discretion is the better part of valor in such challenging cases. If the option exists to optimize the patient's care, this will always be a better choice than to persist against all odds until the elective anesthetic becomes an airway emergency.

Otolaryngologic Surgical Procedures

Bilateral Myringotomy and Pressure Equalizing Tubes (BMT)

The sequelae of the immature pediatric eustachian tube, recurrent acute otitis media, and persistent middle ear effusion are among the most common conditions of childhood. Recurrent otitis can lead to the excessive use of antibiotics as well as frequent symptoms of otalgia (ear pain) and hearing loss. Chronic middle ear effusion results in a persistent hearing loss that can impair the development of communication skills. For children who reach a certain threshold, in terms of the number of infections or months with effusion, BMT placement is indicated to bypass the dysfunctional eustachian tube(s), ventilate the middle ears, reduce the need for antibiotics, and improve hearing. The average age for BMT is 1–3 years, and approximately 10–15% will require tube replacement following spontaneous extrusion of the tubes.

Although the surgery normally takes about 5–10 min, there are a few variables that may affect the duration of the procedure. Small ear canals, as seen in patients with Down syndrome, or significant bleeding from middle ear granulation tissue can make the operation more challenging and therefore lengthier. Furthermore, children who have had multiple sets of tubes may have little tympanic membrane area left appropriate for myringotomy that may also make the procedure more complex and longer.

Preoperative Assessment and Optimization

For the healthy child, a focused history and physical examination is all that is needed. Further evaluation will be dictated by the existence of comorbidities. Premedication with sedatives is rarely used for this procedure since most sedatives outlast the duration of the procedure. If premedication is chosen, midazolam 0.5 mg/kg may be given orally.

Intraoperative Management

General anesthesia is induced and maintained with a potent inhalation agent with or without nitrous oxide. Sevoflurane, because of its non-pungent odor and lack of irritation to the airways, has become the favored inhalation agent in pediatric anesthesia. Given the short duration of the procedure, IV access does not need to be obtained. However, IV equipment should be readily available should difficulty arise and access is needed emergently. Because there is no IV, it is especially important to ensure that an adequate depth of anesthesia is obtained prior to the start of the procedure to avoid laryngospasm from occurring (see above). Signs that the procedure may begin include convergent eyes with small pupils, normal muscle tone, and a relatively normal to low heart rate for age.

An oral airway may be needed to maintain airway patency while the head is turned from side to side during surgery. Continuous positive airway pressure (CPAP) of 5–8 cm H_2O can also be helpful in maintaining airway patency. Alternatively, an LMA may be used ^[67]; however, IV access should be considered prior to manipulation of the airway.

During the procedure, the anesthesiologist must not move the patient's head or manipulate the airway without first informing the otolaryngologist. Any patient movement, even minimal, will distort the otolaryngologist's microscopic view and could potentially result in injury to the ear canal or tympanic membrane by the surgical instrument present in the ear. Whenever an airway issue arises, it is important to remember that the "airway before ear" rule should apply. Once the anesthesiologist identifies an airway problem, the otolaryngologist is immediately informed so that the instrument present in the ear can be removed and the procedure stopped. When the problem is resolved, the otolaryngologist is informed that the procedure may resume.

Postoperative analgesia may be accomplished in a number of different ways with equal efficacy. Acetaminophen suppository may be given rectally at the start of the procedure. The analgesic dose of *rectal* acetaminophen is 40 mg/kg^[68] in contrast to the intravenous dose of 15 mg/kg. This higher dose should not be given orally. Subsequent dosing is 15 mg/ kg and should not be given until 6 h later to avoid exceeding the maximum daily dose of 100 mg/kg/day. The parents should be informed that acetaminophen was given rectally, and advised as to the next time they may give the child more medication if needed. It is rare that additional analgesics will be needed since the discomfort of tube placement usually subsides within 2 h.

Intranasal fentanyl (1–2 mcg/kg) given during the procedure has been shown to decrease pain scores and postoperative agitation without increasing vomiting, hypoxemia, or discharge times ^[69]. Peripheral blockade of the auricular branch of the vagus nerve (nerve of Arnold) has been shown to be as effective as intranasal fentanyl for postoperative pain relief ^[70]. This block is performed by everting the tragus, penetrating the cartilage with a small bore needle, and injecting 0.2 mL of 0.25% bupivacaine with 1:200,000 epinephrine bilaterally (Fig. 20.3).



Fig. 20.3 Nerve of Arnold Block

Postoperative Management

BMT is almost exclusively performed as an outpatient procedure. Patients may be discharged home as soon as they meet standard discharge criteria. The average stay in the PACU is less than 30 min; however, patients with a preexisting URI may require a longer stay. The addition of a second analgesic to acetaminophen (e.g., intranasal fentanyl) has not been shown to decrease the incidence of emergence delirium in these patients ^[71].

Tonsillectomy and/or Adenoidectomy

Adenotonsillectomy (AT) is an extremely old operation, described by the Roman aristocrat and physician Celsus in 50 AD. During the early part of the twentieth century, this operation was done frequently and in many cases on a routine basis without regard for specific indications. Backlash against this indiscriminate "kitchen table" surgery arose later in the century, and at one point tonsillectomy, like circumcision, was derided as "ritualistic surgery" ^[72].

Inflammatory disease of the pharynx was initially the predominate indication for AT. During the twentieth century, the pathophysiology of pediatric sleep disordered breathing was recognized. As adenotonsillar hypertrophy was identified as the cause of the vast majority of obstructive sleep apnea (OSA) in otherwise normal children, this became an increasingly common indication for AT. Even children with other contributing factors to their OSA, such as craniofacial abnormalities or obesity, may see symptomatic improvement after AT (Table 20.10).

Currently, the majority of AT (approximately 80%) is being done for OSA or other more limited forms of sleep disordered breathing. The indication for the remaining cases is

Table 20.10 Conditions associated with obstructive sleep apnea

- 1. Acromegaly
- 2. Cleft palate (following palatoplasty or pharyngeal flap)
- 3. Craniosynostoses (e.g., Crouzon, Apert or Pfeiffer syndrome)
- 4. Mucopolysaccharidoses (e.g., Hurler or Hunter syndrome)
- 5. Micrognathia/retrognathia (e.g., Treacher-Collins Syndrome,
- Goldenhar syndrome or Pierre Robin anomalad)
- 6. Trisomy 21
- 7. Neuromuscular disease involving hypotonia

primarily recurrent pharyngitis, specifically streptococcal infections. Adolescents and young adults may undergo the procedure for severe symptoms of chronic inflammation (e.g., halitosis, dysphagia, and tonsilloliths) even in the absence of documented bacterial infections. Other indications for tonsillectomy and/or adenoidectomy include adenoidal hypertrophy, peritonsillar abscess, asymmetric tonsillar hypertrophy, and post-transplant lymphoproliferative disorder (PTLD).

OSA and Adenotonsillar Hypertrophy

The pharyngeal airway (from the oropharynx to the glottis) may be thought of as a collapsible tube, the lumen of which is maintained patent by the tone of the pharyngeal musculature. In most neurologically normal children, this tone is adequate to keep the airway open during respiration. While the muscle tone diminishes cyclically during sleep, the pressure in the airway is generally above the critical closing pressure (CCP), which is needed to maintain airway patency. However, the airway can collapse when muscle tone is inadequate to maintain a patent lumen, as with sedative drugs, alcohol, or in patients with neuromuscular hypotonia. The airway can also collapse if the luminal air pressure, which is holding the tube open, drops sufficiently. This latter phenomenon is seen when the airway is compromised from adenotonsillar hypertrophy resulting in increased airspeed and concomitant diminished air pressure (i.e., Bernoulli effect).

The intermittent cessation of ventilation will result in progressive hypoxia and hypercapnea, until the point that the brainstem arousal reflex is triggered, increasing pharyngeal tone and restoring airflow. The problem with a cyclical interruption of the natural sleep architecture is that physiological rest is denied, resulting in the many symptoms of sleep apnea (e.g., daytime somnolence). In severe cases, this cycle can lead to cardiovascular complications such as arrhythmias and pulmonary hypertension. Longstanding pulmonary hypertension results in right atrial enlargement, right ventricular hypertrophy, and ultimately cor pulmonale (right-sided heart failure). Polycythemia may also be present. Furthermore, the brainstem carbon dioxide (CO₂) "set point" can be reset, resulting in a diminished respiratory drive after the adenotonsillar hypertrophy has been addressed surgically. This latter effect can be exacerbated by the administration of supplemental oxygen, which effectively shuts down the hypoxic component of the respiratory drive, as oxygen can diffuse into the alveoli even when volume ventilation is poor.

While the criteria for pediatric OSA are evolving, there are some standards that may be documented by polysomnography (PSG) during a formal overnight sleep study. In many cases, the decision to proceed with surgery will be a clinical one, based on parental observation or informal sleep videos or even home sleep studies. While the role of these guidelines remains controversial, it is certainly acceptable to recommend surgery in selected cases based on criteria short of PSG. However, PSG is the "gold standard" and is critical for the management of complex cases with hypotonia, obesity, craniofacial abnormalities, or any other condition in which AT alone may not suffice to address the OSA. Even if AT results in some improvement in the sleep disorder, PSG may be necessary to manage other adjuvant therapy such as nasal continuous positive airway pressure (CPAP) or biphasic positive airway pressure (BiPAP).

Post-transplant Lymphoproliferative Disorder

Post-transplant lymphoproliferative disorder (PTLD) is a complication of organ transplantation immunosuppressive therapy. Epstein–Barr virus (EBV) infection, which is generally self-limiting in the immune competent individual, can result in significant lymphoid hyperplasia in the immunosuppressed individual, resulting in adenotonsillar hypertrophy, airway obstruction, and death ^[73]. Heightened awareness of the implications of adenotonsillar hypertrophy secondary to PTLD and timely surgical and medical interventions are crucial to improve patient outcomes ^[74].

Preoperative Assessment and Optimization

During the preoperative assessment, particular attention should be directed toward the evaluation of the airway and those end-organs potentially affected by OSA, including the cardiac and pulmonary systems. A personal or family history of coagulopathy should also be sought. Airway evaluation is crucial in this patient population. The degree of tonsillar hypertrophy should be evaluated and the potential for difficulty with mask ventilation and intubation assessed. The presence of any craniofacial abnormality may make management of the airway even more difficult. In such cases, alternative airway management tools (e.g., LMA, fiberoptic intubation equipment, videolaryngoscope) should be readily available. In those patients with long-standing or severe OSA, further pulmonary and cardiovascular evaluation may be warranted. An arterial blood gas should be obtained to determine the presence of CO_2 retention. The degree of retention will aid in managing ventilation parameters in the operating room and postoperatively. In these patients, a chest X-ray (CXR) may show an enlarged cardiac silhouette.

A pediatric cardiac consultation should be sought if evidence of pulmonary hypertension exists including an electrocardiogram looking for right-sided heart changes, such as right atrial enlargement, right ventricular hypertrophy, and/ or right axis deviation. Echocardiography may be necessary if there is concern about cardiac function or cor pulmonale.

A preoperative hematocrit should be considered in the child with chronic hypoxia because the presence of polycythemia may be further evidence and indicative of longstanding OSA. An accurate baseline measurement is also crucial if significant blood loss is encountered.

Although bleeding in the postoperative period may occur after tonsillectomy, routine coagulation studies are not universally obtained ^[75]. Patients undergoing tonsillectomy are at no greater risk for the presence of an undiagnosed coagulopathy than the general population. Even in those patients with inherited coagulation disorders, preoperative coagulation studies may not be diagnostic, and the standard international normalized ratio (INR), prothrombin time (PT), or partial thromboplastin time (PTT) may be normal. Furthermore, routine preoperative screening of coagulation may not be cost-effective [76]. Of greater importance in detecting the possible presence of abnormal coagulation is the preoperative history. Parents should be asked about the presence of excessive bleeding after cuts, tooth extractions, previous surgeries, easy bruisability, recurrent nosebleeds, or any familial coagulation disorders. If the history is suggestive of the existence of a coagulation disorder either in the patient or family, coagulation studies should then be obtained prior to surgery. A hematology consult should be secured if the coagulation studies are abnormal and/or the history is strongly suggestive of a coagulopathy.

Premedication, if used, should be given cautiously to patients with a history of airway obstruction, particularly those with the diagnosis of OSA. These patients tend to be more sensitive to sedatives and opioids and may develop significant airway obstruction when sedated. If premedication is administered, it should be done in a monitored setting with pulse oximetry and the personnel and equipment available to manage airway problems should they develop.

Intraoperative Management

AT is performed under general anesthesia using a number of competing methodologies: cold dissection, electrocautery,

Coblation[®], or microdebrider. There is controversy about the appropriate indications for extracapsular tonsillectomy, intracapsular tonsillectomy, or partial tonsillectomy. A discussion of this is beyond the scope of this chapter, and variations in technique have little impact on anesthetic management. Intraoperative anesthetic considerations include management of the airway, sharing the airway with the otolaryngologist, and intraoperative and postoperative bleeding.

Both inhalation and IV inductions are appropriate for these patients. However, the usual induction technique in the pediatric patient is inhalational to avoid the need for a needle stick in an awake, anxious child. It also has the advantage of preserving spontaneous ventilation. Airway obstruction during induction of anesthesia is common but can usually be relieved with the placement of an oral airway once the patient is adequately anesthetized, as well as administration of CPAP to stent the airway, and positive pressure ventilation. In the patient with symptoms consistent with severe OSA, a preinduction IV may be indicated, even though an inhalational induction is planned, to allow for immediate access if emergency medications become necessary. When performing an IV induction, the anesthesiologist should be confidant that they can manage the patient's airway since spontaneous respirations will likely not be preserved once anesthetized.

If not already done, IV access should be obtained once the patient has reached an acceptable plane of anesthesia. An appropriate sized angiocatheter should be used and the function should be assured, as intraoperative bleeding may necessitate rapid volume resuscitation.

Airway Management

Airway management for these cases can be accomplished with either an ETT or LMA. If tracheal intubation is chosen, the use of a muscle relaxant is at the discretion of the anesthesiologist. An advantage of not using a muscle relaxant and maintaining spontaneous ventilation throughout the procedure is the ability to titrate opioids to the patient's respiratory rate. Since OSA patients are sensitive to opioids, this technique would ensure that the patient does not receive an excessive amount of these medications, resulting in apnea and inability to extubate at the end of the procedure.

An oral RAE tube, with its preconfigured bend, minimizes kinking and is optimal for these cases. Standard ETTs may obstruct the view of the otolaryngologist or may kink when bent out of the way. A mouth retractor (Crowe-Davis, McIvor, Boyle Davis) is placed to maintain oral patency and keep the tongue and ETT away from the adenotonsillar area. During placement of the mouth retractor, it is important to make sure that the ETT does not become obstructed or dislodged. The use of an RAE tube does not entirely prevent this from occurring. If mechanical ventilation is used, peak airway pressures should be noted prior to placement of the mouth gag. If peak airway pressure increases significantly during placement of the mouth retractor, the otolaryngologist should be asked to adjust the mouth retractor accordingly. ETT dislodgement may occur, either distally into the right mainstem bronchus or out into the pharynx or esophagus. Therefore, particular attention should be paid to the presence of breath sounds and ETCO₂. During the procedure, continued vigilance is necessary since ETT obstruction or displacement may occur at any time.

Although many pediatric anesthesiologists are using cuffed ETTs, there are still those who prefer uncuffed tubes. However, for AT surgery most anesthesiologists would place a cuffed ETT to provide additional protection against aspiration of blood from the airway during the procedure. In instances where electrocautery is used, a cuffed ETT provides the additional advantage of protecting against the possibility of airway fires when compared to uncuffed ETTs. The leak around an uncuffed ETT allows for an increased oxygen concentration in the oropharynx thereby increasing the potential for an airway fire to occur during electrocautery ^[77].

Reinforced LMAs may be used for these cases [78]; however, tonsillar hypertrophy may make placement difficult. Since the LMA stem is larger than that of an ETT, obstruction and dislodgement of the LMA occurs more frequently during mouth retractor placement [79]. During the procedure, dislodgement or obstruction of the LMA may require its removal and replacement with an ETT. This may be difficult in the face of a partially removed tonsil or bleeding in the airway. Airway fires with the use of LMAs and electrocautery have not been reported. Advantages of an LMA are a decreased incidence of postoperative stridor, hoarseness, and laryngospasm^[62]. LMAs have also been shown to protect against aspiration of blood ^[62, 80]. Examination of the larynx and the underside of the LMA at the conclusion of surgery showed no blood contamination in the LMA group [62, 64] whereas aspiration of blood was seen in 54% of children intubated with an uncuffed ETT [62]. However, these advantages must be weighed against the disadvantages mentioned above.

Analgesia

Intraoperatively analgesia should be judiciously administered. The goal is to provide for postoperative pain management while minimizing the possible respiratory complications that may occur in this patient population postoperatively. The optimal pain management regimen is yet to be determined. Hence, the regimen employed is usually based on the patient's medical condition and the individual anesthesiologist's preference. Confounding considerations include postoperative nausea and vomiting (PONV), sedation, respiratory depression, and airway obstruction. Options for pain control include opioids, nonsteroidal anti-inflammatory drugs (NSAIDs), acetaminophen, and local anesthetics. Postoperative pain management commonly consists of multiple modalities. Opioids, such as fentanyl or morphine, are most commonly used. As described above, titration to respiratory rate would ensure that these patients do not have excessive respiratory depression and can be safely extubated at the end of the procedure. Although the total dose of fentanyl administered during these cases will vary, in a patient with minimal OSA symptoms, approximately 3 mcg/kg would be a reasonable amount for adenotonsillectomy and 2 mcg/kg for adenoidectomy alone. PONV and sedation are a common side effect of opioid therapy.

NSAIDs, such as ketorolac, provide effective pain control as compared to morphine with a lower risk of nausea and vomiting ^[81]. However, the literature is unclear as to whether NSAIDs cause an increased risk of post-tonsillectomy bleeding ^[82–84]. Regardless, the majority of otolaryngologists prefer that NSAIDs not be administered in the perioperative period.

Acetaminophen 40 mg/kg may be given rectally as an adjunct to other analgesic modalities and should be given after induction but prior to the start of surgery to allow time for adequate plasma levels. Acetaminophen at this dose has been shown to decrease postoperative and at home requirement of analgesia ^[85]. In November 2010, the United States Food and Drug Administration approved the intravenous acetaminophen formulation. This can be administered in lieu of the rectal formulation in a dose of 15 mg/kg every 6 h not to exceed 75 mg/kg/day ^[86, 87]. The current labeling for intravenous acetaminophen is for patients 2 years and older.

Local anesthetics may be administered by the otolaryngologist directly at the surgical site. Tonsil packs soaked with local anesthetic are placed in the tonsillar beds for topical absorption. Alternatively, local anesthetic may be infiltrated. A recent study comparing the use of IV ketamine 0.5 mg/kg with peritonsillar bupivacaine 0.25% (3–5 mL per tonsil), versus only bupivacaine, versus only saline found that the ketamine/bupivicaine combination was safe and effective in reducing post-tonsillectomy pain ^[88]. Care should be taken to avoid the use of any solution containing vasoconstrictive agents in the surgical field, as they have been associated with cardiovascular complications and even mortality ^[89].

Postoperative Nausea and Vomiting

There is an increased incidence of PONV after AT ^[90]. Causes of PONV include opioids, the presence of blood in the stomach, and swelling and inflammation of the posterior pharynx and uvula. Dexamethasone, at a dose of 0.05–0.15 mg/kg, is commonly administered to decrease airway edema, and has the added benefit of antiemetic properties. The perioperative administration of dexamethasone is not associated with an increased incidence of bleeding postoperatively ^[91, 92] A second antiemetic (e.g., ondansetron), may also be administered or used as a rescue drug in the PACU. Adequate hydration may also play a key role in preventing PONV. At the conclusion of the procedure and prior to removal of the mouth retractor, the otolaryngologist should pass an orogastric tube under direct vision to suction any blood present in the stomach. The oropharynx should be suctioned as well. This will result in removal of secretions and blood that may contribute to PONV and laryngospasm.

Emergency and Extubation

Extubation of the trachea can be accomplished either while the patient is still deeply anesthetized or when fully recovered. The advantage of deep extubation is less chance of disrupting hemostasis from the patient coughing with the ETT in place. However, the disadvantage is increased risk of airway obstruction and laryngospasm. Awake extubation has the advantage of assuring intact airway reflexes thereby decreasing the risk of aspiration and laryngospasm.

An uncommon complication of AT in the severely obstructed patient is the development of negative pressure pulmonary edema (NPPE) once the airway obstruction is relieved ^[93, 94]. When chronic airway obstruction is relieved by adenotonsillectomy, the capillary wall pressure gradient is increased by a forceful inspiratory effort causing fluid leakage into the interstitial space ^[95]. This may become evident once the ETT is removed at the end of the procedure. Treatment is the same as for negative pressure pulmonary edema which consists of oxygen, diuretic administration, and, if need be, re-intubation to provide CPAP or positive end expiratory pressure (PEEP). This complication is selflimiting and usually resolves within 24–48 h ^[79].

Postoperative Management

Patients should be placed in the lateral decubitus position with the head down with supplemental oxygen for transport to the PACU. Analgesic and antiemetic medications should be ordered.

The most serious postoperative complication is postoperative hemorrhage that will be discussed in detail in the section on post-tonsillectomy bleeding.

Since pain on swallowing and nausea and vomiting occur frequently in these patients, discharge from the PACU should occur only when the child has demonstrated that they are able to swallow fluids and not vomit. Adequate analgesia and antiemetic therapy are, therefore, important to facilitate discharge.

Although tonsillectomy for OSA can be done as an ambulatory procedure, there is a subset of patients who should be observed in the hospital overnight in a monitored setting for apnea. In general, the most common criteria used for overnight monitoring are age less than 3 ^[96], the presence of craniofacial or neuromuscular abnormalities, the continued need for supplemental oxygen, or airway obstruction in the PACU. Postoperative airway obstruction may be due to post-surgical airway edema, residual anesthesia, and/or ongoing analgesic therapy, particularly with opioids.

AT is associated with a painful recovery that can last up to 2 weeks. The pain tends to peak between days 5 and 10, as the eschar separates from the tonsil beds. Good pain control is crucial, as the lack of appropriate analgesia will result in poor oral intake, and a vicious cycle in which the patient becomes even more uncomfortable and dehydrated. Acetaminophen with codeine elixir (120 mg/12.5 mg per 5 mL) works well in this population and may be alternated with plain acetaminophen when the pain is not so severe, to avoid the gastrointestinal side effects of codeine. Aggressive hydration also helps break the pain cycle, and should be encouraged.

Post-tonsillectomy Bleeding

Introduction

The incidence of post-tonsillectomy bleeding ranges from 2% to 4% ^[97, 98]. It is more common in teenagers and young adults than in small children. The vast majority of postoperative bleeding occurs between days 5 and 10, when the eschar separates from the tonsil beds. In rare cases, bleeding may occur in the immediate postoperative period. Initial management is tailored to the degree of bleeding. The clot in the tonsillar bed should be suctioned, as spontaneous hemostasis is rare with the clot in place. Minimal bleeding may resolve with ice water gargle, and older children may tolerate the application of a cautery stick with silver nitrate while awake. Significant bleeding in a young child, however, typically involves a return to the operating room for a thorough examination of the pharynx and electrocautery hemostasis.

Preoperative Assessment and Optimization

The preoperative assessment should be directed at airway evaluation and volume status (Table 20.11). Although it may be difficult to assess the airway in depth in the agitated child, observation of the external anatomy and information about airway management from the prior anesthetic should provide sufficient information. Even though airway management was uncomplicated previously, it may be more difficult at this time because of postoperative edema and blood obscuring visualization of the larynx.

Assessment of volume status is of paramount importance. It is easy to underestimate the degree of blood loss, since much of it may have been swallowed. Heart rate, blood pressure, and, if possible, orthostatic testing will provide

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Assessment	Action
Airway	Evaluate airway anatomy Obtain previous intubation records
Volume	Assess volume status Start large bore intravenous catheter Consider intraosseous access if intravenous access attempts not successful Resuscitation with isotonic non-glucose containing fluids Transfuse red blood cells if hemodynamically unstable and hematocrit is low
Hematology	Send hematocrit, type and cross, coagulation studies Proceed to OR even if results are unavailable Check results as soon as possible

 Table 20.11
 Preoperative assessment of the post-tonsillectomy bleeding patient

information regarding volume status and guide volume resuscitation. Assessing for the presence of tears, moist mucus membranes, skin turgor, and urine output will be helpful as well. Adequate IV access should be obtained, if not already present. Intraosseous access should be entertained in the hypovolemic, hypotensive patient if IV access cannot be obtained in a timely fashion. Volume resuscitation should be initiated with non-glucose containing isotonic fluids. If the patient is hypotensive, 20 mL/kg boluses of isotonic fluid should be administered until the blood pressure normalizes. Once the bleeding has been controlled and the patient is normovolemic, maintenance fluids should be continued. Maintenance fluids are calculated by the following formula:

- 0-10 kg: 4 mL/kg/h
- 11-20 kg: 2 mL/kg/h
- \geq 21 kg :1 mL/kg/h

Laboratory tests should be obtained, specifically hematocrit, platelet count, electrolytes, and coagulation studies. A specimen should also be sent for serotyping and crossmatch, as transfusion is a very real possibility. Surgery should not be delayed waiting for test results, since bleeding will continue without surgical intervention, and abnormal values would rarely by themselves preclude a general anesthetic.

Intraoperative Management

Management of the airway is of major concern, particularly the ability to visualize the larynx in the presence of ongoing bleeding. In preparation for induction, the following should be prepared: multiple laryngoscope blades, a styletted cuffed ETT, and two large bore suctions (a double suction setup) in case one clots or proves to be ineffective. The otolaryngologist should be present and tracheostomy equipment immediately available should a surgical airway become necessary.

The patient is considered to have a full stomach, even if they have not recently eaten, because of swallowed blood. This presents a dilemma for the anesthesiologist who may be concerned that visualization of the larynx and intubation may be difficult. An awake fiberoptic intubation may be an option in the older patient but it is not a feasible option in the young agitated child. Also, the presence of blood in the airway may make visualization with the flexible bronchoscope difficult, if not impossible. Thus, a rapid sequence induction is usually performed. However, in the situation where there is significant concern about the airway, a smooth mask inhalation induction with cricoid pressure can be performed with the patient in the right lateral decubitus position with the head down (tonsillar position) with suction immediately available. The tonsillar position minimizes aspiration risk by promoting the pooling of blood in the orapharynx.

The choice of IV induction agent will depend on the volume status. Etomidate or ketamine may be used if there is ongoing concern about volume status and hemodynamic stability. Alternatively, propofol may be used but in a decreased dose. Muscle relaxation can be achieved with either succinylcholine or rocuronium. However, the duration of action of rocuronium at the dose recommended for rapid sequence induction (1.2 mg/kg) will likely exceed the length of the procedure. Furthermore, in situations where the airway is of major concern, succinylcholine, with its rapid onset and short duration of action, may be preferable, despite the concern of its use in the pediatric population.

Volume status should be continually assessed in the face of ongoing bleeding and managed appropriately with isotonic fluids. The hematocrit, degree of hemodynamic stability, and the status of hemostasis will dictate the need for a blood transfusion. Replacement of coagulation factors is rarely necessary. However, if a previously undiagnosed coagulopathy is discovered, the appropriate therapy should be initiated and a hematology consult obtained.

All other intraoperative and postoperative concerns are the same as described above for tonsillectomy.

Airway and Esophageal Foreign Body

Introduction

Foreign body aspiration into the airway occurs most commonly in children less than 4 years of age ^[99], but is rare before age one, when a child develops a significant degree of mobility and the ability to manipulate small objects. A history of a witnessed episode of choking and/or coughing may be elicited, but the absence of such a history does not rule out the presence of an airway foreign body. Small foreign bodies may be small enough to pass silently through the larynx and impact in the distal bronchi, with signs and symptoms not developing for days or weeks. Diagnosis may be quite challenging, but a heightened index of suspicion must be raised when asymmetric breath sounds, persistent cough, new onset wheezing in the absence of reactive airway disease, or CXR evidence of air trapping and/or segmental collapse.

Occlusive pharyngeal foreign bodies that obstruct the larynx and result in complete airway obstruction are true lifethreatening emergencies that rarely make it to the operating room. Accidental asphyxia from items such as grapes, hot dogs, or fruit skins can result in death within minutes and are best managed by widespread familiarity with emergency procedures such as the Heimlich maneuver. Aspirated airway foreign bodies, on the other hand, are rarely emergencies, and may present in subtle ways, as mentioned above. Therefore, operative intervention should not be undertaken until the circumstances are optimized and the appropriate otolaryngologist and anesthesiologist are available, even if it means delaying the procedure ^[100]. Furthermore, all necessary equipment should be available and tested prior to the induction of anesthesia.

Children, who present with agitation, wheezing, and/or cyanosis, are true surgical emergencies and should be taken to the OR as soon as possible. The aspiration of watch and hearing aid batteries are especially notorious since they can induce a corrosive reaction that can result in complete transmural injury in as little as an hour ^[101]. Sharp objects, such as open safety pins, are also typically considered emergencies, although in some cases these can present months after the initial aspiration (Fig. 20.4).

At the other extreme, patients with a chronic airway foreign body, especially if suspected to be organic in nature, may have significant granulation tissue surrounding the object but a stable airway. Such patients will benefit from 24 to 48 h of systemic steroid therapy prior to endoscopy, which will minimize bleeding and make extraction easier. Nuts and seeds are the most common objects aspirated by children. Most nuts are oily and may cause a localized inflammatory reaction. They can also be difficult to extract during bronchoscopy, as they may break apart during manipulation. Foreign bodies more commonly lodge in the right main bronchus and less frequently in the larynx and trachea. Coins are the most common esophageal foreign body. Less common are plastic or metal parts of toys.

Preoperative Assessment and Optimization

The degree of airway obstruction and/or respiratory distress should be assessed and will guide the timeline of surgical management. Children with foreign bodies lodged in the supraglottic region will present with inspiratory stridor, dyspnea, cough, and possibly cyanosis. Children with foreign bodies lodged in the glottis or subglottis will present with

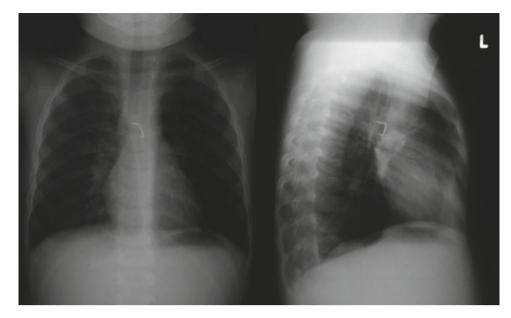


Fig. 20.4 PA and lateral chest X-ray of a child who had aspirated a carpet staple several months prior to presentation. Symptoms included a lingering cough and failure to thrive, but no significant stridor or

respiratory distress. At bronchoscopy, the foreign body was found to be lodged at the carina, surrounded by a large mass of granulation tissue

biphasic stridor, cough, and hoarseness. Foreign bodies in the intrathoracic airways will demonstrate expiratory stridor that is more pronounced on collapse of the airway during exhalation. A foreign body that passes the subglottis will almost always pass the carina and lodge in the mainstem bronchus or possibly more distally depending on the size of the object. This condition will result in asymmetric wheezing, more pronounced on the involved side. However, a chronic foreign body resulting in atelectasis or consolidation may alternatively produce diminished breath sounds on the involved side. Room air oxygen saturation will be helpful in determining the severity of the respiratory compromise. Premedication in most cases should not be administered to these children, especially if respiratory compromise is apparent. However, anxiety due to respiratory compromise should be expected and managed with a calm demeanor and parental presence throughout the process.

Although routine preoperative studies are not indicated, a CXR should be obtained in stable patients with suspected foreign body aspiration. Although many foreign bodies are not radiopaque, the CXR may be helpful in determining the location of the foreign body by evidence of secondary changes. Unilateral air trapping or hyperinflation would suggest the presence of a foreign body on that side. Additionally, atelectasis on the effected side distal to the foreign body, an infiltrate on the effected side, or mediastinal shift may be seen.

If the patient has recently eaten, surgery should be postponed only if the patient is stable and delaying surgery would not place the patient at increased risk for worsening of respiratory function or even complete obstruction. Removal of foreign bodies lodged in the hypopharynx should be considered an urgent procedure. A foreign body in this position poses a hazard of dislodging, particularly if the patient is gagging and coughing, and entering the larynx completely occluding the airway. While the majority of tracheobronchial foreign bodies are not emergencies and standard nil per os (NPO) guidelines may be observed, certain foreign bodies warrant immediate surgical intervention, even in the face of a full stomach, as outlined above (Fig. 20.5).

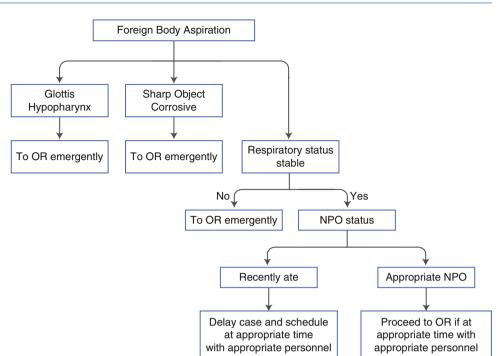
Intraoperative Management

Communication between the anesthesiologist and otolaryngologist is critical to the safe and successful care of these patients. The anesthetic plan, particularly management of the airway, should be discussed and agreed upon prior to the start of the case.

Either an inhalation or IV induction is appropriate. However, IV access should be obtained pre-induction in the compromised child, even if an inhalation induction is planned. The administration of an antisialagogue, glycopyrrolate (0.01 mg/kg), or atropine (0.01–0.02 mg/kg) should be considered to decrease secretions that may impair the otolaryngologist view. Dexamethasone should be administered to decrease airway edema.

If proceeding in a patient determined to have a full stomach, the anesthesiologist must weigh the risk of pulmonary aspiration of gastric contents against the risk of losing a patent airway if a rapid sequence induction is performed.

Fig. 20.5 Approach to the patient with foreign body aspiration



It should be remembered that even in the patient with a full stomach, removal of a foreign body from the respiratory system precludes placement of an ETT and the airway will be unprotected until the otolaryngologist introduces the rigid bronchoscope. Suctioning of the stomach after induction but prior to inserting the bronchoscope may be helpful in decreasing the risk of gastric aspiration. Tracheal intubation is possible when esophageal foreign bodies are present. However, cricoid pressure during rapid sequence induction may be contraindicated if the foreign body is at the level of the cricopharyngus. Of note, the ETT may become compressed by the esophagoscope and can be detected by close monitoring of the peak airway pressure and the presence and quality of the ETCO₂ waveform.

The anesthetic management of patients undergoing bronchoscopy for foreign body removal is controversial as to whether to maintain spontaneous ventilation or to control ventilation^[102]. There is a theoretical concern that controlled ventilation and positive pressure may push the foreign object deeper into the small airways making retrieval more difficult or creating a greater obstruction through a "ball-valve" effect. Conversely, spontaneous ventilation may prove inadequate to maintain adequate oxygenation or ventilation and increases the risk of unexpected movement or coughing causing airway trauma or rupture. Although both methods of ventilation are acceptable, studies have shown that the need for conversion from spontaneous to controlled ventilation occurs frequently [86, 103]. The reverse, conversion from controlled to spontaneous, has not been reported. The ultimate decision will be based on the preference of the anesthesiologist and

otolaryngologist, the medical condition of the patient, and the nature of the foreign body aspiration.

If spontaneous ventilation is maintained, topical anesthesia of the vocal cords may help to decrease stimulation by insertion of the bronchoscope. Lidocaine is commonly used and the dose should not exceed 2–3 mL/kg to avoid toxicity since absorption via mucosal surfaces may approach that of IV administration. This can be administered either by a prefilled laryngo-tracheal-anesthesia (LTA) device or by using a syringe with an attached angiocatheter. In the latter case, it is important to ensure that the catheter is tightly fixed to the syringe by Luer-lock or tape, to decrease the risk of catheter dislodgement into the airway. During controlled ventilation, muscle relaxation should be administered to ensure immobility of the patient.

There is no preferred method of general anesthesia maintenance for these cases. In all cases, nitrous oxide should be avoided because of the presence of air trapping in the lungs and the likely presence of decreased oxygenation. The anesthesia circuit can be attached to the rigid bronchoscope (Fig. 20.6) with a standard 15 mm ventilatory side port that allows for the administration of a potent inhalation agent. However, since ventilation may vary during the procedure, particularly if spontaneous, the depth of anesthesia may be inconsistent, requiring an IV agent as an adjunct to ensure an adequate depth of anesthesia. If an unsheathed optical forceps is used in place of a ventilating bronchoscope (which is occasionally a useful technique in very small airways), total intravenous anesthesia (TIVA) will be necessary. This can be accomplished in a number of ways and no particular method

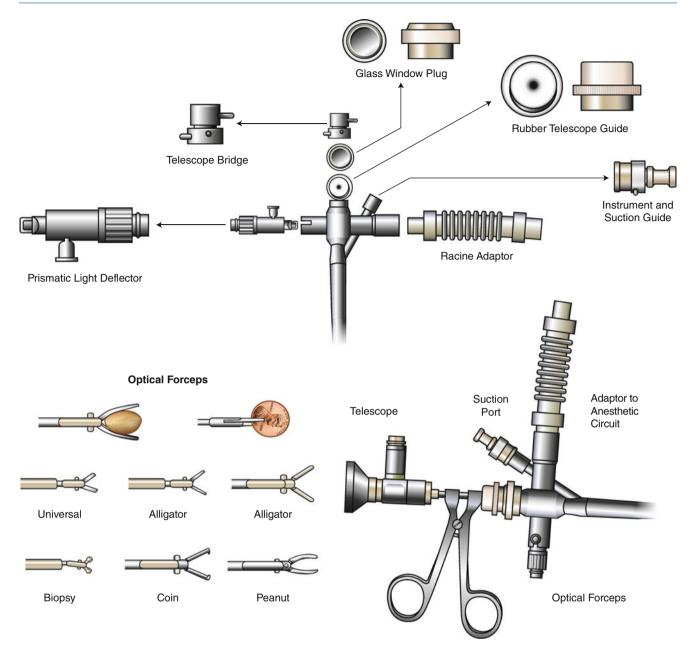


Fig. 20.6 Ventilating bronchoscope with accessories for the use of optical forceps. Modified with permission from KARL STORZ Endoscopy America

has been shown to be superior compared to another. Commonly used agents are propofol and remifentanil. The advantage of these agents is that they are short-acting and, therefore, will not contribute to respiratory compromise postoperatively.

Problems encountered during ventilation via the bronchoscope include: (1) dislodgement of the anesthesia circuit, (2) increased work of breathing through the narrower lumen of the bronchoscope in the spontaneously breathing patient, (3) leak around the bronchoscope especially when removing foreign bodies from the proximal airway, and (4) one-lung ventilation of the compromised lung when the bronchoscope is advanced into the effected lung. The latter will result in oxygen desaturation and elevated CO_2 . Periods of oxygen desaturation and ineffective ventilation may need to be tolerated briefly to allow the otolaryngologist time to retrieve and remove the foreign body. During this period, it is crucial that effective communication between the otolaryngologist and anesthesiologist is ongoing. The video feed commonly available with modern bronchoscopy equipment greatly facilitates this communication, allowing all members of the team to see exactly where the bronchoscope is in the airway at all times. It is strongly recommended that all anesthesiologists become familiar with bronchoscopic images so that during bronchoscopy the otolaryngologist's progress can be closely followed.

During the procedure, the otolaryngologist may need to withdraw the bronchoscope into the trachea in order to allow for adequate oxygenation and ventilation. Once these parameters improve, the otolaryngologist can reintroduce the bronchoscope. Depending on the underlying pulmonary compromise, this may need to be repeated multiple times. Occluding the patient's nose and mouth can minimize tidal volume and anesthetic leakage around the bronchoscope.

When working high in the airway, as is the case during the rare tracheal foreign body (Fig. 20.7), significant leakage from the circuit may occur through side holes located in the end of a typical ventilating bronchoscope, which may be above the level of the glottis. To prevent this problem, a tracheoscope may be used, which is identical to a ventilating bronchoscope but without the side holes near the tip (Fig. 20.8). If such a device is not available, the holes can be occluded by wrapping the distal portion of the bronchoscope with an occlusive plastic dressing (e.g., TegadermTM), but care must be taken to prevent such items from being left in the airway. During removal of a fragmented foreign body, the bronchoscope may be removed and reinserted with each fragment removed. This may necessitate mask ventilation with 100% oxygen in between each reinsertion.

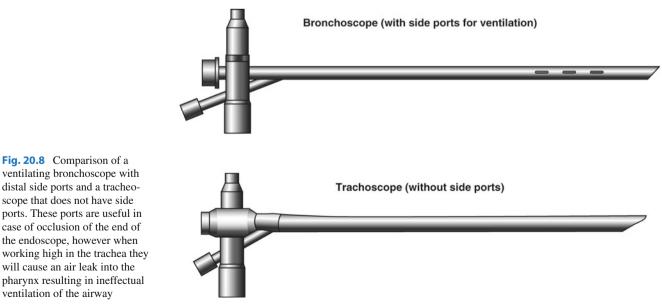
Large foreign bodies in the bronchus may be dropped during removal in the trachea or larynx causing complete airway obstruction. If prompt removal of the foreign body is not possible, the otolaryngologist should push it back into one of the mainstem bronchi in which it was originally impacted, so that ventilation of at least one lung can resume. If the foreign body advances into the "good lung", airway edema in the

other lung can further compromise the patient's pulmonary function.

Intraoperative hypoxia, hypercarbia, and laryngospasm may be avoided by maintaining an adequate depth of anesthesia and ensuring adequate oxygenation and ventilation. An inadequate depth of anesthesia and stimulation of the airway may lead to arrhythmias. Inhalation agents, by



Fig. 20.7 A high tracheal foreign body (a plastic bead) in the subglottic area. Ventilation using a standard ventilating bronchoscope with side ports may be impossible due to the proximal gas leak. A tracheoscope may be helpful in this situation, or temporary occlusion of the side ports with an adhesive wrapper if a tracheoscope is not available



ventilating bronchoscope with distal side ports and a tracheoscope that does not have side ports. These ports are useful in case of occlusion of the end of the endoscope, however when working high in the trachea they will cause an air leak into the pharynx resulting in ineffectual ventilation of the airway

sensitizing the myocardium to catecholamines, contribute to the development of arrhythmias as well. This was seen more often with halothane than sevoflurane.

Pneumothorax, although rare, is a life-threatening complication and should be considered anytime there is an acute change in pulmonary and/or cardiovascular parameters. If suspected, placing a needle in the suspected side may be life saving and should not be delayed while waiting for CXR confirmation. Needle aspiration is performed in the 2nd intercostal space at the midclavicular line entering above the 3rd rib to avoid damaging the neurovascular bundle located below the 2nd rib. After sterilely preparing the area, a 14–18 gauge angiocatheter attached to a 5 or 10 mL syringe is advanced in a downward angle until a loss of resistance or a "pop" is felt. Advance the catheter over the needle into the pleural space and replace the syringe with a three-way stopcock and 50 mL syringe to aspirate air. This should be replaced with a chest tube if necessary.

After completion of the procedure, extubation of the trachea may be considered if significant respiratory compromise was not present preoperatively and is not expected postoperatively. Reversal of muscle relaxation should be assured. Once adequate ventilation, oxygenation, and muscle relaxation are demonstrated, extubation of the trachea may take place.

If intubation was not performed for the procedure, the anesthesiologist may opt to awaken the patient utilizing mask ventilation if pulmonary function is adequate and significant airway edema and inflammation is not expected. If there is any concern, the trachea should be intubated until that time when the patient has completely emerged from general anesthesia and extubation criteria have been met. At times, significant respiratory compromise may be present at the end of the procedure secondary to edema and inflammation of the airways either present preoperatively or exacerbated during difficult removal of the foreign body. In these instances, postoperative intubation and ventilation should be considered and the patient transferred to the intensive care unit. This allows time for lung expansion and resolution of any airway edema and inflammation.

Postoperative Management

The respiratory status, both preoperative and postoperative, and the difficulty in removal of the foreign body will be determining factors in the postoperative disposition of the patient. For uncomplicated cases, the patient may be discharged home the same day. Depending on the type of foreign body and the duration of impaction, an inflammatory response may persist well beyond the time of removal. For those with respiratory compromise, admission to the hospital, and possibly the intensive care unit, will be necessary.

Table 20.12 Indications for tracheostomy in the infant

- Prolonged mechanical ventilation

 (a) Bronchopulmonary dysplasia
 (b) Central hypoventilation

 Airway obstruction

 (a) Craniofacial abnormalities
 (b) Congenital or acquired subglottic stenosis
 (c) Severe tracheomalacia

 - (d) Bilateral vocal cord paralysis
- 3. Pulmonary toilet in children with severe neurological or pulmonary disease

Neonatal Tracheostomy

Introduction

Indications for tracheostomy in the infant are outlined in Table 20.12. The most common diagnosis requiring tracheostomy in the neonatal population is inability to wean from mechanical ventilation usually secondary to bronchopulmonary dysplasia (BPD) in the preterm infant. Upper airway obstruction comprises the other major group of patients requiring tracheostomy. Presenting symptoms in the patient who is not already intubated are persistent hypoxia, hypercarbia, or airway obstruction.

In most instances, this procedure is usually performed electively with the patient already intubated. However, the patient who presents with airway compromise and is not intubated poses a significant challenge for the anesthesiologist.

Preoperative Assessment and Optimization

Preoperative assessment will be focused on evaluation of the airway and comorbidities. In patients who are already intubated, the indication for tracheostomy and the ease of prior intubations should be ascertained. In preterm infants, the course in the neonatal intensive care unit should be reviewed with special attention focused on the pulmonary, cardiac, and neurologic status of the patient. Patients with craniofacial abnormalities often have associated cardiac congenital anomalies that require preoperative evaluation.

Patients with craniofacial abnormalities who are not already intubated pose an additional challenge to the anesthesiologist. Besides the preoperative assessment outlined above, evaluation of the airway is of paramount importance in order to formulate an appropriate anesthetic plan that would not place the patient in jeopardy for complete airway obstruction leading to hypoxia prior to the establishment of an artificial airway. The airway evaluation should include a determination of whether airway patency can be maintained by facemask once general anesthesia is induced and the ease of visualization of the larynx by standard laryngoscopy. Additional airway adjuncts should be available and prepared including oral and nasal airways of various sizes, LMAs of various sizes, a flexible pediatric bronchoscope, and a pediatric videolaryngoscope. Occasionally, a rigid pediatric bronchoscope may be necessary as well.

Premedication with a sedative is contraindicated due to possible development of airway obstruction and hypoxia. Certainly, respiratory depressants should be avoided in the already respiratory compromised patient. Administration of an antisialagogue, glycopyrrolate or atropine, may be beneficial to decrease airway secretions. The additional anticholinergic property of these medications may offer protection against bradycardia secondary to hypoxia if airway obstruction should develop.

Intraoperative Management

Based on the preoperative evaluation of the airway, all necessary airway equipment should be prepared accordingly even if the patient arrives to the operating room with their trachea already intubated. If a difficult airway is anticipated, another experienced anesthesiologist should be present until the airway is safely secured. The otolaryngologist should be present from the start of the case, as well, prepared to either pass a bronchoscope or perform an emergency surgical airway if necessary.

If the patient arrives to the operating room tracheally intubated, placement of the ETT should be confirmed by the presence of bilateral breath sounds and $ETCO_2$ because the ETT may have become dislodged or advanced during patient transport. If an IV is present, it should be checked for functionality. The technique for the induction of general anesthesia will be dictated by the presence of comorbidities.

For the patient who is not tracheally intubated, an inhalation induction is preferred. The goal is to maintain spontaneous ventilation until the anesthesiologist confirms that a patent airway can be maintained, manual ventilation is possible, and/or intubation of the trachea successful. In the compromised airway or identified difficult airway, IV access should be obtained prior to induction so that emergency medications can be administered as needed. An anticholinergic/antisialagogue medication should be administered prior to induction for reasons stated above. The addition of positive end expiratory pressure (PEEP) or continuous positive airway pressure (CPAP) may help in stenting the airway open and facilitate assisted ventilation. Patients with micrognathia (e.g., Pierre Robin anomalad) may be helped by placement of a nasal airway. If mask ventilation proves to be difficult, placement of an LMA may relieve the obstruction and allow for adequate ventilation and oxygenation ^[104, 105].

Intubation in the presumed difficult airway should be performed without muscle relaxation so that if the attempts are unsuccessful, the patient can resume spontaneous ventilation. If muscle relaxation is deemed necessary for optimal intubation conditions, a short-acting medication, such as succinylcholine, should be administered if not contraindicated.

If intubation using a standard laryngoscope is unsuccessful, then other intubation devices should be tried as long as adequate oxygenation and ventilation are maintained. Whether a fiberoptic scope or videolaryngoscope is used is unimportant. The anesthesiologist should use the device with which they are most familiar. In the event that intubation is not possible with these other devices, tracheotomy may be performed with an LMA in place or by facemask as long as adequate oxygenation and ventilation can be achieved ^[106].

When selecting the size of the ETT, the presence of abnormal airway narrowing should be taken into account. The use of a stylette should be entertained to optimize intubation conditions.

Once the airway is secured, maintenance of general anesthesia will be dictated by the presence of comorbidities; 100% oxygen should be administered in the event that the airway is lost at any time during the procedure. The use of muscle relaxation is at the preference of the anesthesiologist. Those who do not use muscle relaxants believe that if the ETT should become dislodged or ventilation becomes compromised (i.e., when the trachea is entered by the otolaryngologist) oxygenation will be maintained by the spontaneously breathing patient. Those who use muscle relaxants believe that the ETT is less likely to dislodge if the patient does not move. It is also important for the patient to be immobile during critical points in the surgery such as when the otolaryngologist enters the trachea.

Positioning consists of a shoulder roll that extends the neck to provide adequate surgical access. The chin will be pulled up by strategic placement of a tape sling to maintain extension and to stabilize the soft tissue over the trachea. Head extension withdraws the ETT from the airway and care must be taken to prevent a premature extubation.

The use of opioids is at the discretion of the anesthesiologist. Local infiltration by the otolaryngologist with a local anesthetic containing epinephrine is performed prior to the start of the procedure and will provide adequate analgesia postoperatively. The use of epinephrine rarely causes cardiovascular compromise when injected locally, however, if there is any concern epinephrine may be omitted.

Prior to entering the trachea, the otolaryngologist must alert the anesthesiologist. If a cuffed ETT is used, the cuff should be deflated to avoid being damaged during this part of the procedure. When the otolaryngologist enters the trachea, a large leak will develop causing a loss of tidal volume possibly compromising oxygenation and ventilation. It is during this time that communication between the otolaryngologist and anesthesiologist is of paramount importance. If oxygenation and ventilation become significantly compromised, it may be necessary for the otolaryngologist to stop and occlude the opening made in the trachea. This maneuver may be necessary multiple times until the tracheotomy tube is ready to be inserted. Non-absorbable Prolene or nylon stay sutures (4-0 in infants, 3-0 in older children) are placed on either side of the tracheal opening to stabilize the trachea for cannulation. Postoperatively and prior to establishment of the tracheal tract, these stay sutures may be life-saving in the event that the tracheotomy tube becomes dislodged and re-insertion of an airway device becomes necessary. The stay sutures should be clearly marked right or left so that pulling on the sutures in the proper direction results in opening of the tracheotomy site. Absorbable sutures are used to adapt the skin to the tracheotomy site, maturing the tract, making the surgical airway more stable and minimizing the possibility of passing a tube into a false passage.

Just prior to insertion of the tracheotomy tube, the ETT, if present, is withdrawn to just above the tracheal window but still within the trachea. This allows for re-advancement of the ETT beyond the tracheal window in the event of difficult tracheal cannulation. The ETT is not completely removed until correct positioning of the tracheotomy tube is confirmed by attaching the anesthesia circuit and auscultating for bilateral breath sounds and confirming the presence of $ETCO_2$. It should be noted that ventilation through the tracheotomy tube might be compromised by the presence of the ETT, as it allows for easy shunting of gas out of the airway. Therefore, when checking ventilation through the tracheotomy tube, the ETT should be occluded but not withdrawn. Once positioning of the tracheotomy tube and integrity of the cuff, if present, is confirmed and the otolaryngologist agrees, the ETT is completely withdrawn.

Intraoperative complications include airway fire (discribed in detail later in chapter), the development of subcutaneous emphysema, pneumothorax, pneumomediastinum, bleeding, recurrent laryngeal nerve injury, and passage of the tracheotomy tube into a "false passage" in the soft tissues outside of the airway. Subcutaneous emphysema is diagnosed by the presence of crepitus in the neck and shoulders and should prompt investigation into the correct positioning of the tracheotomy tube. Tube position can be quickly ascertained by passage of a flexible bronchoscope (2.5 mm outer diametet) through the lumen that will immediately determine tracheostomy tube location and position. Acute deterioration in the pulmonary or cardiac status of the patient, if not related to malpositioning of the tracheotomy tube, may be secondary to pneumothorax or pneumomediastinum. Bleeding from soft tissue, thyroid vessels, or vascular anomalies may obscure the otolaryngologist's view.

Postoperative Management

At the end of the procedure, the patient is transferred to the intensive care unit. Either mechanical or spontaneous ventilation is continued and humidified air or oxygen provided. Suctioning of the tracheotomy tube by experienced personnel is important to prevent clogging by blood clots or secretions. Due to the small tracheal tube lumen size, any obstruction can significantly impede oxygenation and ventilation. Extreme care should be taken not to dislodge a newly placed tracheotomy tube. The stay sutures should be readily accessible and clearly marked in case of tracheotomy tube dislodgement. A backup tracheotomy tube and appropriate sized ETTs should be available at the bedside should any problems arise whether it is secondary to lumen obstruction or dislodgement.

Recurrent Respiratory Papilloma

Introduction

Recurrent respiratory papillomas (RRP) in infants are usually caused by exposure to human papilloma virus present in the birth canal. However, cases of neonatal RRP have been reported even after cesarean delivery. They can be found at any site in the upper aerodigestive tract, but they are most common, and most potentially dangerous, in the laryngotracheal complex. Presenting symptoms consist of stridor and hoarseness. Rapid growth of the papillomas results in progressive airway obstruction and some of these patients present emergently to the operating room. Because the papillomas cannot be eradicated surgically, these children frequently return to surgery for debulking at intervals, with the timing being dictated by the rapidity of papilloma regrowth. The goal of surgical resection is to maintain a safe airway without causing permanent damage to the larynx. Although there are some adjuvant medical treatments that might prevent spread of the virus or diminish the rate of recurrence, the primary management of RRP at this point remains surgical [107].

In the past, laser surgery was the most common method used for RRP debulking. The benefit of laser surgery is that the beam can be focused on a very small area for precise tissue excision and hemostasis. However, the use of a laser in the airway is complicated and potentially dangerous, as plastic ETTs are flammable and not safe in this application. Furthermore, even in skilled hands there is a potential for permanent scarring and residual vocal problems ^[108]. While still used in some cases of papilloma (more commonly in adults treated in an office setting), the microdebrider has for practical purposes replaced the laser in the treatment of pediatric RRP in our practice. This device allows for precise, rapid excision of papilloma tissue around a small ETT with minimal injury to the surrounding structures of the larynx. Despite this improved technique, however, laryngeal injury is still possible [109]. Therefore, it should be clear that the goal of any such resection is not complete elimination of gross disease, but rather maintenance of a safe airway while awaiting the remission of viral expression with time.



Fig. 20.9 Glottic opening obscured by overgrowth of laryngeal papilloma

Preoperative Assessment and Optimization

Preoperative assessment is directed at assessing the degree of airway obstruction and respiratory compromise. Difficult mask ventilation and tracheal intubation should be anticipated and prepared for if significant disease is present. However, if the child is moving air well while awake, even in the setting of marked papilloma obstruction at the glottis, a small ETT can often still be used. While no airway should ever be taken for granted, as long as equipment and personnel are standing by for an emergency surgical airway, the appearance of the glottis alone should not preclude a gentle attempt at orotracheal intubation (Fig. 20.9).

Keeping the child calm is important since agitated breathing may exacerbate the symptoms of airway obstruction. Premedication, if used, should be done cautiously and judiciously. The risk of causing further or total airway obstruction in the sedated child should be weighed against the benefit of decreasing anxiety in a patient who may be psychologically affected by the frequent repeated visits to the operating room. It would be prudent to withhold premedication in the patient presenting emergently for progressive airway obstruction and provide sedation for the child presenting for maintenance therapy. If premedication is undertaken, it should be done in a monitored setting with emergency airway equipment immediately available.

Intraoperative Management

While lasers are rarely used in our practice at the time of this writing in the management of RRP, we will outline our approach to their use in case the reader encounters such a situation. When laser surgery is planned, precautions need to be taken to avoid harming the patient and operating room personnel by inadvertent contact with the laser beam. Signs stating that laser is being used should be placed on all doors to the operating room. Since the laser beam can damage the cornea or retina, all operating room personnel should wear appropriate eye goggles and the patient's eyes should be taped shut and covered with saline soaked eye pads or metal shields. The head and neck of the patient should be covered by saline soaked towels to prevent laser injury or fire. Appropriate highdensity masks should be worn by all operating room personnel in close contact to the smoke plume emanating from the airway. The smoke plumes contain fine particulate (0.1-0.8 mcm) matter that may contain viral particles ^[110]. Regular operating room masks cannot protect against such small particles.

The risk of fire is a particular concern with the use of lasers, with airway fires being the most serious complication. Surgical drapes are flammable, and are unnecessary for this procedure, so should not be used. The patient's head and neck should be shielded with saline soaked towels, as mentioned above. The laser should always be switched off or in stand-by mode when not in use by the surgeon. Airway fires are caused by the laser beam contacting flammable material in the airway such as polyvinyl chloride ETTs or cotton pledgets. Therefore, during laser surgery of the airway, a specialized ETT should be used that will not catch on fire if contacted by the laser beam. Red rubber tubes wrapped with reflective aluminum have been used in the past, but such a coating may easily fail. Furthermore, the aluminum wrap can be applied only up to the cuff leaving the area below the vocal cords vulnerable to fire. Commercially available laser-safe tubes that have metal exteriors are more commonly used. However, their use in small infants or patients with narrowed airways is limited because of the larger outer diameter of laser-safe tubes when compared with the same sized PVC tube. The larger sized tubes come with two cuffs so that if the laser beam damages the proximal cuff, the second cuff will remain inflated and protect the airway. The cuffs should be filled with a normal saline solution not air. In this way, if the laser beam ruptures the proximal cuff, the spray of saline solution may extinguish early combustion, thereby, preventing an airway fire [111]. In some cases, the ETT can be dispensed with altogether during RRP excision by relying on apneic technique with intermittent intubation between brief laser sessions. This is challenging, however, for both the otolaryngologist and the anesthesiologist, and should not be the method of choice since better alternatives exist.

Because oxygen increases the combustibility of flammable materials, the inspired concentration of oxygen should be kept at less than 30%, as long as adequate oxygenation of the patient is maintained. Nitrous oxide should be avoided as well since it can support combustion at 450 °C ^[95].

Although airway fires are rare, the operating room team should be ready to manage it expeditiously. The mnemonic of the 4Es should be executed as quickly as possible in order to limit the damage ^[112]. All combustible material should be EXTRACTED from the airway, the source of oxygen should be ELIMINATED, all fires should be EXTINGUISHED with normal saline, and any damage caused by the fire should be EVALUATED. If the ETT is on fire, it should be immediately removed and the airway secured as soon as it is possible with a new ETT.

During induction of general anesthesia, the otolaryngologist should be present and ready to advance a bronchoscope or perform an emergency surgical airway if necessary. Induction of general anesthesia is usually performed by facemask with sevoflurane and oxygen with or without nitrous oxide. If nitrous oxide is used, it should be discontinued as soon as an adequate plane of anesthesia is attained. In the severely obstructed patient, consideration should be given to obtaining IV access prior to induction. Otherwise, IV access is obtained after an adequate depth of anesthesia is achieved. Spontaneous ventilation is maintained until the anesthesiologist can prove that they can assist or manually ventilate without difficulty. Dexamethasone and a muscle relaxant should be administered at this time. Muscle relaxation is important to provide a still surgical field so that the otolaryngologist will be able to precisely excise the papillomas without interference from laryngeal motion. An antisialagogue may be administered at this time as well.

On occasion, the airway obstruction from overgrowth of the papillomas is so great that induction of anesthesia becomes hazardous and tracheal intubation almost impossible because the glottic opening is totally obscured. Because these patients frequently return for repeated debulking, the decision may be made to perform a tracheotomy to allow for safer management of these patients during future anesthetics. Although the mechanism of distal papilloma spread into the trachea is not clear, tracheotomy has classically been associated with exacerbation of this risk. This may be due to postoperative mucosal changes, or simply the fact that tracheotomy is done in patients with the most aggressive disease. In any case, heroic measures to avoid a tracheotomy, such as very frequent debulking sessions, are themselves risky in patients with marginal airways. It does not seem prudent to withhold this operation solely based on unproven fears of distal spread [113].

Management of the airway can be achieved in multiple ways, the method chosen based on the preference of the anesthesiologist and otolaryngologist and the need for surgical access to papillomas that may be obscured by the ETT. The three methods available are placement of an ETT for the

 Table 20.13
 Comparison of techniques for airway management

 during papilloma surgery of the airway

ETT and controlled ventilation	Intermittent apnea	Jet ventilation
Specialized ETT to avoid airway fire if using laser	No specialized ETT needed	No specialized ETT needed
Inhalation, TIVA, or combination	TIVA	TIVA
<30% oxygen, avoid nitrous oxide if using laser	Intermittent intubation with oxygenation and hyperventilation	Provide controlled ventilation throughout, <30% oxygen if using laser
Obscures anterior and posterior commissures	Unobstructed surgical field	Unobstructed surgical field
Small ETT sizes may not be appropriate for small infants and narrowed airways	Increased ETCO ₂	Complications: Pneumothorax Pneumomediastinum Air trapping Stomach distension; risk of regurgitation Possible distal spread of viral particles

ETT endotracheal tube, $TIV\!A$ total intravenous anesthesia, $ETCO_2$ end-tidal carbon dioxide

entire procedure, intermittent apnea with removal of the ETT during use of the laser, and jet ventilation (Table 20.13).

Placement of an ETT ensures a secure airway and allows for ventilation throughout the procedure. It also protects the airway from particulate debris from entering the airway. However, the ETT may obscure visualization by the otolaryngologist of the anterior and posterior commissures. Maintenance of general anesthesia can be accomplished either by inhalation, TIVA, or a combination of both. No one technique has been proven to be superior.

Intermittent apnea with removal of the ETT [114] allows for greater access to the papillomas present in the posterior commissure. Another advantage to this method is that the risk of an airway fire is greatly reduced since there is no flammable object (ETT) in the airway and oxygen is not insufflated during the laser treatment. A polyvinyl chloride ETT may be used for intubation. Initially, the trachea is intubated while the otolaryngologist is positioning the patient and equipment. During this time, the patient should be on 100% oxygen and hyperventilated in preparation for apnea. Once the otolaryngologist is ready to laser, the oxygen should be turned off and the ETT removed. The otolaryngologist under direct vision performs re-intubation during laryngeal suspension when there is a decrease in oxygen saturation. ETCO, will rise during the apneic period but is usually well tolerated. TIVA will be necessary to maintain an adequate level of anesthesia since there will be significant periods of time when the inhalation agent will be discontinued.

Jet ventilation is another alternative to airway management. In our practice, we do not utilize this technique for RRP management in children. However, this discussion is included for the sake of completeness, as it is occasionally used in some circumstances. In children, a metal injector attached to a Venturi apparatus is placed 1–2 cm above the glottic opening. Oxygen concentrations should be kept at <30%. Care should be taken to keep the driving pressure less than 15 psi and to allow enough time for expiration (2–4 s) to occur. This method has the advantage of providing ventilation and oxygenation without the presence of a flammable object thus reducing the risk of an airway fire. The risks of this approach include air trapping and barotrauma in an obstructed airway, as well as the potential for distal dissemination of papilloma virus. Stomach distension may occur increasing the risk of regurgitation and pulmonary aspiration.

When the procedure is completed, emergence can occur with or without tracheal intubation. If the ETT was in place the entire procedure, the patient should be awakened with the ETT in place. Reversal of muscle relaxation should be assured and the ETT removed when the patient is awake and meets extubation criteria. If intermittent apnea or jet ventilation was used, it is acceptable to have the patient emerge from general anesthesia with a facemask. However, if significant respiratory compromise was present preoperatively, it would be wise to awaken the patient with an ETT in place.

Postoperative Management

Many of these cases are done on an ambulatory basis, particularly the regularly scheduled maintenance procedures. If there is no respiratory compromise noted in the PACU, these patients can be discharged home. However, the patient who presented emergently with airway compromise or who continues to have signs of airway obstruction in the PACU may require admission to a monitored setting for further management. In a small subset of patients, a return to the operating room for further debulking may be necessary. In these patients, adequate debulking of the papillomas may have been compromised by the presence of the ETT during the laser treatment. If further surgery is necessary, one of the alternative methods described above may be necessary to provide optimal visualization for the otolaryngologist.

Postoperative pain is not a major issue in these patients and can easily be managed with acetaminophen.

Acute Upper Airway Infections

Introduction

Some acute infections of the upper airway may require operative intervention in addition to medical therapy. These are often problems in the pediatric population, given a smaller airway's increased susceptibility to compromise with edema or inflammatory debris. Some of these (e.g., epiglottitis) have all but vanished due to vaccinations and herd immunity ^[115, 116]. The most common of these, croup (laryngotracheobronchitis), rarely requires an artificial airway or other surgical management.

Epiglottitis usually occurs in children 2–7 years of age and is most commonly caused by Haemophilus influenzae. The child with epiglottitis will present with symptoms of upper airway obstruction consisting of inspiratory stridor, tachypnea, and retractions. The presence of stridor is concerning in that complete airway obstruction may be pending. The child will prefer to be sitting with their chin up and mouth open and supporting themselves on their hands (tripod position). The inability to handle secretions and difficulty swallowing will result in drooling.

Croup usually occurs in children 6 months to 3 years of age and is viral in origin. Presenting symptoms are a barking cough, inspiratory stridor, and hoarseness. Medical management consists of cool mist, oxygen as needed, and steroids ^[117]. Although cool mist is used often, the literature does not support its usefulness ^[118]. Biphasic stridor represents severe airway compromise as do chest retractions and cyanosis in room air. Nebulized epinephrine may be useful in relieving the obstruction and repeat dosing may be necessary.

Bacterial tracheitis involves the accumulation of inspissated mucus in the upper airway. This may require the techniques of foreign body extraction to keep the tracheobronchial tree clear while antibiotic therapy is used to manage the underlying disease.

In each of these cases, however, it is important to remember that airway compromise may develop rapidly in a patient who is also suffering from a systemic infection, making efficient coordinated efforts between the otolaryngology and anesthesia teams crucial for success.

Preoperative Assessment and Optimization

Just as with other cases involving a tenuous airway, children with croup, epiglottitis, or other such infectious airway problems should be kept as calm as possible during the time prior to securing the airway as anxiety or panic can precipitate deterioration in the airway status. Preoperative evaluation is directed at the degree of airway compromise. Radiographic examination of these patients should be entertained only in the stable patient. In epiglottitis, a lateral soft tissue neck film shows a large round epiglottis ("thumb sign"). In croup, an anteroposterior soft tissue neck film shows narrowing of the subglottic area ("steeple sign"). If there is any doubt regarding the stability of the airway, radiographic examination should not be performed nor is it necessary.

Supplemental oxygen should be administered as needed and IV access attempted in the stable patient for fluid and antibiotic administration. IV access will also be helpful in the event that the airway becomes unstable and intervention is needed emergently. Otherwise, these children should be brought emergently to the OR suite for definitive intervention with the appropriate personnel and equipment available.

While in the past clinicians have been cautioned about airway inspection in cases of epiglottitis (for fear of precipitating further airway compromise), the information gained by awake flexible laryngoscopy can be invaluable when considering the need for a tracheotomy under local anesthesia. A *skilled* otolaryngologist or anesthesiologist should be able to see the hypopharynx and larynx with a minimum of direct airway trauma using a small flexible endoscope, and in such cases the benefits outweigh the risks. However, this should not be done by someone inexperienced in this technique in children with compromised and tenuous airways.

Once the decision has been made to secure the airway in the operating room, it is the responsibility of the otolaryngologist and anesthesiologist to ensure that all necessary equipment is immediately available before the induction of anesthesia. This includes the full range of ventilating bronchoscopes, as well as a tracheotomy set that has been opened and prepared for emergent use.

Intraoperative Management

In cases of epiglottitis, it may not be possible to visualize the glottic airway due to edema and cellulitis of the epiglottis and other supraglottic structures. Patients with impending asphyxia should undergo tracheotomy under local anesthesia, or even a cricothyrotomy if the situation is truly dire. However, if the patient is breathing spontaneously and moving air, there may yet be a transoral pathway to the trachea. This would be preferable to a surgical airway, as attempts at an awake tracheotomy in a struggling child could convert a partial obstruction into a complete one. Even if tracheotomy is ultimately necessary, it is far safer to secure the airway first by orotracheal intubation if possible.

An inhalation induction is preferable with the goal of maintaining spontaneous ventilation until the airway has been safely secured. Topical anesthesia delivered directly to the larynx prior to instrumentation once the patient is adequately anesthetized may be beneficial. Visualization of the glottis may be difficult in epiglottitis secondary to the enlarged epiglottis and care should be taken not to traumatize the epiglottis further by manipulation of the laryngoscope blade. A styletted ETT a half size smaller than predicted should optimize the chances of successful intubation.

The key in epiglottitis is not to blindly attempt intubation that may result in worsening of airway compromise from hematoma or exacerbation of existing edema. The ventilating bronchoscope would be more appropriate in this situation, allowing the optical element at the tip to be manipulated precisely past the supraglottic pathology to the (presumably normal) glottis and tracheobronchial tree. Once the airway has been secured in this manner and ventilation confirmed. tracheotomy may be done over the bronchoscope. Attempts at replacing the bronchoscope with an endotracheal tube using the Seldinger technique would inappropriately risk loss of the airway and exacerbating the existing airway compromise. However, it is important that good positioning of the scope be maintained throughout the tracheotomy, in the center of the lumen and above the carina. This would be the responsibility of a second endoscopist. Unlike an endotracheal tube, which may be safely secured by tape once proper placement is ensured, a rigid steel bronchoscope can potentially cause life-threatening trauma to the tracheobronchial tree if it is displaced distally or driven against the tracheal wall while being used to ventilate the patient.

If the airway is secured with an ETT, the patient is transferred to the intensive care unit for further management. Either orotracheal or nasotracheal intubation is appropriate and will be dictated by the preference of the critical care team. In some instances, tracheotomy is performed electively after ETT intubation.

Patients with croup may require endotracheal intubation in severe cases. However, due to the acute inflammation in the subglottis, such patients are particularly prone to injury from the ETT ^[119]. It is best to avoid the need for intubation in the first place if possible (through the use of steroids, racemic epinephrine or heliox). If intubation is required, the smallest tube which will allow for adequate ventilation should be used, and extubation should be done as soon as clinically feasible. Since in cases of croup, visualization of the glottis is not typically a problem, consideration should be given to nasotracheal intubation. By fixing the endotracheal tube at the nares instead of the oral commissure, motion of the ETT (with associated axial shearing trauma of the subglottic mucosa) may be minimized, reducing the risk of airway injury. Tracheotomy through an inflamed tracheal airway (i.e., croup) is also associated with an increased risk for long-term complications such as stenosis, and should be used sparingly if at all. This was recognized as early as the nineteenth century ^[120].

In cases of bacterial tracheitis, the anesthetic and endoscopic techniques of foreign body extraction would apply, with the appropriate modifications for working high in the airway (as described above).

Postoperative Management

When such cases are brought to the operating room, it is usually to secure the airway. Therefore, postoperative management will be primarily by the critical care team, who will determine the timing of extubation following standard clinical guidelines.

Conclusion

The pediatric patient has different physiologic characteristics and anatomical aspects of the upper aerodigestive tracts that require special equipment, training and experience on the part of both the otolaryngologist and the anesthesiologist. These small airways are prone to rapid luminal compromise from either the underlying disease processes or from inexpert attempts at manipulation and intubation. In addition, infants and young children are much more susceptible to rapid oxygen desaturation than are older patients, making the margin for error even smaller. We have described our methods for sharing the pediatric airway, and our preferred approach to the perioperative management of children and their families. As with all areas of otolaryngology and anesthesiology, careful planning and effective communication between team members before the patient even arrives in the operating room will optimize outcomes and minimize complications.

Appendix A

General Anesthesia and Your Child

For most parents, the thought of their child undergoing general anesthesia is by far the most frightening part of any planned surgery. This is understandable, since anesthesia is unfamiliar to most families. Furthermore, the media occasionally reports on a terrifying story of a life-threatening problem associated with a surgical anesthetic. In reality, though, modern anesthesia is extremely safe. It is only because it is so safe—with millions of uncomplicated anesthetics administered every year—that such problems are considered news at all. Here are answers to some commonly asked questions.

Why Can't You Do the procedure Under Local Anesthesia?

For most young children, it is simply not possible to safely perform a surgical procedure without complete (general) anesthesia. Although this may be possible for dental procedures in older patients, it would be far from appropriate for the common operations in my practice.

The administration of local anesthesia itself is often painful and terrifying to a child, as would be the need for restraint. For example, during the placement of ear tubes, even the smallest degree of motion could result in permanent damage to the ear. It simply isn't worth the risk.

Can You Just Use the Smallest Amount of Anesthesia Possible, or Just Some Sedation?

This can actually be more dangerous than general anesthesia. Again, for some clinical situations (such as painless but frightening procedures like a CAT scan), it can be useful. But in a young child with a small airway, the chance of breathing problems is greater if the airway isn't under the anesthesiologist's continual control.

In fact, the period requiring the greatest amount of attention is when the patient is "light," or only slightly anesthetized, during the start or finish of the procedure.

The best analogy is that of flying in an airplane. Most accidents occur during takeoff and landing, when the plane is close to the ground. Similarly, the start and end of anesthesia (induction and emergence) are the most difficult parts of the anesthetic, when the level of anesthesia is lightest. Asking an anesthesiologist to use a small amount of anesthesia (a very common request) would be like asking the pilot to keep the altitude to a minimum by flying just above the treetops!

Who Will Give My Child Anesthesia? Can I Meet That Doctor Ahead of Time?

Your child's anesthetic will be given by a fully trained and experienced attending anesthesiologist, who may have one or more assistants. In almost every case, this doctor will be a specialist in pediatric anesthesiology. In rare situations (usually related to scheduling issues) a general anesthesiologist will be working with me, but in no case will this change the safety of the anesthetic. I would never work with anyone that I did not trust completely.

You will meet this doctor in the hospital just before the surgery, but if you would like to speak to one of the pediatric anesthesiologists ahead of time, you can call the anesthesia office (my staff will give you their number).

I Heard About a Case Where Someone Died Under Anesthesia. Is That Possible?

While this is possible, and has happened, it is extremely rare, especially for healthy children. The overwhelming majority of deaths during surgery involve elderly and/or extremely sick patients undergoing major operations. Millions of people have general anesthesia every year without any difficulty. The actual risk of a fatal event under anesthesia (for an otherwise healthy child) is about 1 in 300,000. To put that number into perspective, the risk of death from an unexpected reaction to penicillin is about 1 in 80,000. The risk of a fatal

automobile accident while riding in a car (in the USA, over a 1 year period) is about 1 in 6,500! Remember, these are extremely rare events, so that when something like that does happen, it makes the news.

What If My Child Is Allergic to Anesthesia? Can You Test for That?

There really is no anesthesia allergy, but there is a very rare condition in which people have a bad reaction to certain anesthetic agents. This is a congenital muscle disease (malignant hyperthermia), which causes a patient to be unstable under anesthesia. Every anesthesiologist knows about this and how to react if this scenario occurs. However, there is no reason to test for this ahead of time (by muscle biopsy) in the absence of anything else that might suggest that the disease is present.

Can I Be There When My Child Goes to Sleep?

My main concern is, of course, the safety of your child. However, I also understand that the stress of surgery (both on the patient and the parent!) can be reduced by your presence in the operating room. In general, one parent is allowed into the operating room while the child goes to sleep.

However, there are some limitations to this general policy. The anesthesiologist is the one who makes the ultimate determination about who is allowed in the operating room. Parents are not allowed in the operating room for patients who are under 9 months of age, or who have certain medical conditions—you should speak to the anesthesiologist about your own child's individual case.

If you yourself feel unsure about how you will react, it is better if you are not there. Seeing a parent having a strong emotional reaction is not reassuring to the child, and may actually be worse than having to go through the procedure alone. And it goes without saying that having a parent faint is not only frightening to the child, but also would result in the need to direct medical attention away from the patient!

Can I Stay During the Procedure?

The only reason for a parent to be in the operating room is to help their child feel better as they go off to sleep. This is not for the parent's benefit. Parents are not allowed in the operating room during the surgery itself, even if they are physicians. This is potentially disruptive. Once again, your child's safety is my primary concern.

Can I Be There When My Child Wakes Up?

This is another very common request. While I do all that I can to make sure that you are separated from your child for the shortest amount of time possible, allowances have to be made for safety. Emergence from anesthesia often requires a good deal of work on the part of the anesthesiologist, and your child need to regain a certain level of consciousness before it is safe to leave the monitors and equipment in the operating room.

While most children are to some degree awake by the time you are reunited with them, they are slowly emerging from a very deep sleep, and usually don't remember much until later on in the recovery period. I know that it is hard to be separated from them when they are going through a stressful experience. I always do my best to keep that time as short as possible.

Why Is My Child Crying in the Recovery Room?

Unlike adults, most children do cry in the recovery room, especially if they are very young or have had a painful procedure (such as a tonsillectomy). This is not because children feel more pain than adults, or get less pain medication. It is because there are many things in this environment that cause stress, and children tend to cry in stressful situations.

In addition to the pain of surgery (which will be treated with a variety of medications), children are often disoriented, frightened, nauseated, hungry, and dehydrated after surgery. All of these things can add to stress. However, children usually feel better within 30 min or so, once they have woken up more fully and have had something to eat or drink.

References

- Dobrin P, Canfield T. Cuffed endotracheal tubes: mucosal pressures and tracheal wall blood flow. Am J Surg. 1977;133: 562–8.
- Slater HM, Sheridan CA, Ferguson RH. Endotracheal tube sizes for infants and children. Anesthesiology. 1955;16:950–2.
- Deakers TW, Reynolds G, Stretton M, et al. Cuffed endotracheal tubes in pediatric intensive care. J Pediatr. 1994;125:57–62.
- Khine HH, Corddry DH, Kettrick RG, et al. Comparison of cuffed and uncuffed endotracheal tubes in young children during general anesthesia. Anesthesiology. 1997;86:27–31.
- Newth CJL, Rachman B, Patel N, et al. The use of cuffed versus uncuffed endotracheal tubes in pediatric intensive care. J Pediatr. 2004;144:333–7.

- Browning DH, Graves SA. Incidence of aspiration with endotracheal tubes in children. J Pediatr. 1983;102:582–4.
- Litman RS, Weissend EE, Shibata D, et al. Developmental changes of laryngeal dimensions in unparalyzed, sedated children. Anesthesiology. 2003;98:41–5.
- Fearon B, Whalen JS. Tracheal dimensions in the living infant (preliminary report). Ann Otol Rhinol Laryngol. 1967;76: 965–74.
- Weiss M, Knirsch W, Kretschmar O, et al. Tracheal tube-tip displacement in children during head-neck movement-a radiological assessment. Br J Anaesthesia. 2006;96:486–9.
- Wheeler M, Cote CJ, Todres ID. The Pediatric Airway. In: Cote CJ, Lerman J, Todres ID, editors. A practice of anesthesia for infants and children. 4th ed. Philadelphia: Saunders; 2009. p. 237–78.
- Cole F. Pediatric formulas for the anesthesiologist. Am J Dis Child. 1957;94:72–673.
- Lau N, Playfor SD, Rashid A, Dhanarass M. New formulae for predicting tracheal tube length. Pediatr Anaesth. 2006;16: 1238–43.
- Sugiyama K, Yokoyama K. Displacement of the endotracheal tube caused by change of head position in pediatric anesthesia: Evaluation by fiberoptic bronchoscopy. Anesth Analg. 1996;82:251–3.
- Berry FA. Physiology and surgery of the infant. In: Berry FA, editor. Anesthetic management of difficult and routine pediatric patients. 2nd ed. New York: Churchill Livingstone; 1990. p. 121–65.
- Keens TG, Bryan AC, Levison H, et al. Developmental pattern of muscle fiber types in human ventilatory muscles. J Appl Physiol. 1978;44:909–13.
- Friedman WF. The intrinsic physiologic properties of the developing heart. Prog Cardiovasc Dis. 1972;15:87–111.
- Kentala E, Kalia T, Arola M, et al. Anticholinergic prememdication in Finland 1988. Eur J Anaesthesiol. 1991;8:135–40.
- Rautakorpi P, Manner T, Kanto J. A survey of current usage of anticholinergic durgs in paediatric anaesthesia in Finland. Acta Anaesthesiol Scand. 1999;43:1057–9.
- McAuliffe G, Bissonnette B, Boutin C. Should the routine use of atropine before succinylcholin in children be reconsidered ? Can J Anaesth. 1995;42:724–9.
- Olsson GL, Hallen B. Laryngospasm during anaestheisa. A computer-aided incidence study in 136,929 patients. Acta Anaesthesiol Scand. 1984;28:567–75.
- Schreiner MS, O 'Hara I, Markakis DA, et al. Do children who experience laryngospasm have an increased risk of upper respiratory tract infection? Anesthesiology. 1996;85:475–80.
- Flick RP, Wilder RT, Peiper SF, et al. Risk factors for laryngospasm in children during general anesthesia. Pediatr Anesth. 2008;18:289–96.
- 23. Mamie C, Habre W, Delhumeau C, et al. Incidence and risk factors of perioperative respiratory adverse events in children undergoing elective surgery. Paediatr Anaesth. 2004;14:218–24.
- 24. Ead H. Post-anesthesia tracheal extubation. Dynamics. 2004;15:20–5.
- Roy WL, Lerman J. Laryngospasm in paediatric anaesthesia. Can J Anaesth. 1988;35:93–8.
- Batra YK, Ivanova M, Ali SS, et al. Efficacy of a subhypnotic does of propofol in preventing laryngospasm following tonsillectomy and adenoidectomy in children. Pediatr Anesth. 2005;15:1094–7.
- Fink BR. The etiology and treatment of laryngeal spasm. Anesthesiology. 1956;17:59–577.
- Larson Jr CP. Laryngospasm-the best treatment. Anesthesiology. 1998;89:1293–4.
- Johnstone RE. Laryngospasm treatement an explanation. Anesthesiology. 1999;91:581–2.
- Tait AR, Malviya S, Voepel-Lewis T, et al. Risk factors for perioperative adverse respiratory event in children with upper respiratory tract infections. Anesthesiology. 2001;95:299–306.

- De Soto H, Patel RI, Soliman IE, Hannallah RS. Changes in oxygen saturation following general anesthesia in children with upper respiratory infection signs and symptoms undergoing otolaryngological procedures. Anesthesiology. 1988;68:27–279.
- Parnis SJ, Barker DS, Van Der Walt JH. Clinical predictors of anaesthetic complications in children with respiratory tract infections. Paediatr Anaesth. 2001;11:29–40.
- 33. Morray J, Geiduschek J, Ramamoorthy C, et al. Anesthesia related cardiac arrest in children: initial findings of the Pediatric Perioperatie Cardiac Arrest (POCA) Registry. Anesthesiology. 2000;93:6–14.
- 34. Empey DW, Laitinen LA, Jacobs L, et al. Mechanisms of bronchial hyperreactivity in normal subjects after upper respiratory tract infection. Am Rev Respir Dis. 1976;113:131–9.
- 35. Aquilina AT, Hall WJ, Douglas Jr RG, Utell MJ. Airway reactivity in subjects with viral upper respiratory tract infections: the effects of exercise and cold air. Am Rev Respir Dis. 1980;122:3–10.
- 36. Tait AR, Malviya S. Anesthesia for the child with an upper respiratory tract infection: still a dilemma? Anesth Analg. 2005;100:59–65.
- Nelson TE. Malignant hyperthermia: a pharmacogenetic disease of Ca⁺⁺ regulating proteins. Curr Mol Med. 2002;2:347–69.
- 38. Warner LO, Beach TP, Garvin JP, et al. Halothane and children: the first quarter century. Anesth Analg. 1984;63:838–40.
- Larach MG, Rosenberg H, Gronert GA, et al. Hyperkalemic cardiac arrest during anesthesia in infants and children with occult myopathies. Clin Pediatr (Phila). 1997;36:9–16.
- 40. Kain ZN, Mayes LC, Bell C, et al. Premedication in the United States: a status report. Anesth Analg. 1997;84:427–32.
- Feld LH, Negus JB, White PF. Oral midazolam preanesthetic medication in pediatric outpatients. Anesthesiology. 1990;73:831–4.
- Rita L, Seleny FL, Mazurek A, et al. Intramuscular midazolam for pediatric preanesthetic sedation: a double-blind controlled study with morphine. Anesthesiology. 1985;63:528–31.
- Wilton NC, Leigh J, Rosen DR, et al. Preanesthetic sedation of preschool children using intranasal midazolam. Anesthesiology. 1988;69:972–5.
- Malinovsky JM, Cozian A, Lepage JY, et al. Ketamine and midazolam neurotoxicity in the rabbit. Anesthesiology. 1991;75:91–7.
- 45. American Academy of Pediatrics. Alternative routes of drug administration-advantages and disadvantages (subject review). Committee on Drugs. Pediatrics. 1997;100:143–52.
- White PF, Way WL, Trevor AJ. Ketamine-its pharmacology and therapeutic uses. Anesthesiology. 1982;56:119–36.
- Gingrich BK. Difficulties encountered in a comparative study of orally administered midazolam and ketamine. Anesthesiology. 1994;80:1414–5.
- Lerman J, Sikich N, Kleinman S, et al. The pharmacology of sevoflurane in infants and children. Anesthesiology. 1994;80:814–24.
- Gregory GA, Eger 2nd EI, Munson ES. The relationship between age and halothane requirements in man. Anesthesiology. 1969;30:488–91.
- Lerman J, Robinson S, Willis MM, et al. Anesthetic requirements for halothane in young children 0-1 month and 1-6 month of age. Anesthesiology. 1983;59:421–4.
- Harea J. Bullard laryngoscope proven useful in difficult intubations in children with Treacher Collins. Anesth Analg. 2004;98:1815–6.
- 52. Holm-Knudsen R. The difficult pediatric airway-a review of new devices for indirect laryngoscopy in children younger than two years of age. Paediatr Anaesth. 2011;21:98–103.
- Rehman MA, Schreiner MS. Oral and nasotracheal light wand guided intubation after failed fiberoptic bronchoscopy. Paediatr Anaesth. 1997;7:349–51.
- Benumof JL. Laryngeal mask airway and the ASA difficult airway algorithm. Anesthesiology. 1996;84:686–99.

- Borland LM, Swan DM, Leff S. Difficult pediatric endotracheal intubation: a new approach to the retrograde technique. Anesthesiology. 1961;55:577–8.
- Vennila R, Hall A, Ali M, et al. Remifentanil as a single agent to facilitate awake fiberoptic intubation in the absence of premedication. Anaesthesia. 2011;66:368–72.
- Bergese SD, Khabiri B, Roberts WD, et al. Dexmedetomidine for conscious sedation in difficult awake fiberoptic intubation cases. J Clin Anesth. 2007;19:141–4.
- Jooste EH, Ohkawa S, Sun LS. Fiberoprtic intubation with dexmedetomidine in two children with spinal cord impingements. Anesth Analg. 2005;101:1248.
- 59. 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;98:1269–77.
- Benumof JL. Use of the laryngeal mask airway to facilitate fiberscope-aided tracheal intubation. Anesth Analg. 1992;74: 313–5.
- Goldie AS, Hudson I. Fiberoprtic tracheal intubation through a modified laryngeal mask. Paediatr Anaesth. 1992;2:343–4.
- 62. Heard CM, Caldicott LD, Fletcher JE, et al. Fiberoptic-guided endotracheal intubation via the laryngeal mask airway in pediatric patients: a report of a series of cases. Anesth Analg. 1996;82: 1287–9.
- Chen L, Sher Sa, Aukburg SJ. Continuous ventilation during translaryngeal mask airway fiberoptic bronchoscope-aided tracheal intubation. Anesth Analg. 1996;82:891–2.
- 64. Brimacombe J, Berry FA. The split laryngeal mask and the difficult airway. Acta Anaesthesiol Scand. 1994;38:744.
- Osborn IP, Soper R. It's a disposable LMA, just cut it shorter-for fiberoptic intubation. Anesth Analg. 2003;97:299–300.
- 66. Yamashita M. Longer tube length eases endotracheal intubation via the laryngeal mask airway in infants and children. J Clin Anesth. 1997;9:432–3.
- Watch MF, Garner FT, White PF, Lusk R. Laryngeal mask airway vs. face mask and Guedel airway during pediatric myringotomy. Arch Otolaryngol Heak Neck Surg. 1994;120:877–80.
- Birmingham PK, Tobin MJ, Henthorn TK, et al. Twenty-four-hour pharmacokinetics of rectal acetaminophen in children. Anesthesiology. 1997;87:244–52.
- 69. Galinkin JL, Fai LM, Cuy RM, et al. Use of intranasal fentanyl in children undergoing myringotomy and tube placement during halothane and sevoflurane anesthesia. Anesthesiology. 2000;93:1378–83.
- 70. Voronov P, Tobin MJ, Billings K, et al. Postoperative pain relief in infants undergoing myringotomy and tube placement: comparison of a novel regional anesthetic block to intranasal fentanyl - a pilot analysis. Pediatr Anesth. 2008;18:119–1201.
- Rampersad S, Jiminez N, Bradford H, et al. Two-agent analgesia versus acetaminophen in children having bilateral myringotomies and tubes surgery. Pediatr Anesth. 2010;20:1028–35.
- Bolande RP. Ritualistic surgery-circumcision and tonsillectomy. N Engl J Med. 1969;280:591–6.
- 73. Hague K, Catalano P, Rothschild M, et al. Posttransplant lymphoproliferative disease presenting as sudden respiratory arrest in a three-year-old child. Ann Otol Rhinol Laryngol. 1997;106: 244–7.
- Sturm-O'Brien AK, Hicks MJ, Giannoni CM, et al. Tonsillectomy in post-transplant lymphoproliferative disease in children. Laryngoscope. 2010;120:608–11.
- 75. Asaf T, Reuveni H, Yermiahu T, et al. The need for routine pre-operative coagulation screening tests (prothrombin time PT/ partial thromboplastin time PTT) for healthy children undergoing elective tonsillectomy and/or adenoidectomy. Int J Pediatr Otorhinolaryngol. 2001;61:217–22.

- Cooper JD, Smith KJ, Ritchey AK. A cost-effective analysis of coagulation testing prior to tonsillectomy and adenoidectomy in children. Pediatr Blood Cancer. 2010;55:1153–9.
- Kaddoum RN, Chidiac EJ, Zestos MM, et al. Electrocauteryinduced fire during adenotonsillectomy: report of two cases. J Clin Anesth. 2006;18:129–31.
- Webster AC, Morley-Foster PK, Dain S, et al. Anaesthesia for adenotonsillectomy: a comparison between tracheal intubation and the armoured laryngeal mask airway. Can J Anaesth. 1993;40:1171–7.
- Wilson MN, Long LS, Ved S, Harley E. Younger paediatric adenotonsillar surgical patients exhibit more complications at mouth gag insertion with LMA use. Int J Pediatr Otorhinolaryngol. 2009;73:1173.
- Williams PJ, Bailey PM. Comparison of the reinforced laryngeal mask airway and tracheal intubation for adenotonsillectomy. Can J Anaesth. 1993;40:1171–7.
- Watcha MF, Jones MB, Laguerueta RG, et al. Comparison of ketorolac and morphine as adjuvants during pediatric surgery. Anesthesiology. 1992;76:368–72.
- Agrawal A, Gerson CR, Seligman I, Dsida RM. Postoperative hemorrhage after tonsillectomy: use of ketorolac tromethamine. Otolaryngol Head Neck Surg. 1999;120:335–9.
- Cardwell M, Siviter G, Smith A. Non-steroidal anti-inflammatory drugs and perioperative bleeding in paediatric tonsillectomy. Cochrane Database Syst Rev. 2005;18:CD003591.
- Marret E, Flahault A, Samama C-M, Bonnet F, et al. Effects of postoperative nonsteroidal, antiinflammatory drugs on bleeding risk after tonsillectomy. Anesthesiology. 2003;98:1497–502.
- Korpela R, Korvenoja P, Meretoja OA. Morphine-sparing effect of acetaminophen in pediatric day-case surgery. Anesthesiology. 1999;91:442–7.
- Murat I, Baujard C, Foussat C, et al. Tolerance and analgesic efficacy of a new i.v. paracetamol solution in children after inguinal hernia repair. Paediatr Anaesth. 2005;15:663–70.
- Alhashemi JA, Daghistani MF. Effects of intraoperative i.v. acetaminophen vs. i.m. meperidine on post-tonsillectomy pain in children. Br J Anaesth. 2006;96:790–5.
- Inanoglu K, Ozbakis Akkurt BC, Turhanoglu S, et al. Intravenous ketamine and local bupivacaine infiltration are effective as part of a multimodal regimen for reducing post-tonsillectomy pain. Med Sci Monit. 2009;15:CR539–43.
- Groudine SB, Hollinger I, Jones J, DeBouno BA. New York state guidelines on the topical use of phenylephrine in the operating room. The phenylephrine advisory committee. Anesthesiology. 2000;92:859–64.
- Pandit UA, Malviya S, Lewis IH. Vomiting after outpatient tonsillectomy and adenoidectomy in children: the role of nitrous oxide. Anesth Analg. 1995;80:230–3.
- Shakeel M, Trinidade A, Al-Adhami A, et al. Intraoperative dexamethasone and the risk of secondary posttonsillectomy hemorrhage. J Otolaryngol Head Neck Surg. 2010;39:732–6.
- Brigger MT, Cunningham MJ, Hartnick CJ. Dexamethasone administration and postoperative bleeding risk in children undergoing tonsillectomy. Arch Otolaryngol Head Neck Surg. 2011;136:766–72.
- Feinberg AN, Shabino CL. Acute pulmonary edema complicating tonsillectomy and adenoidectomy. Pediatrics. 1985;75:112–4.
- 94. Thomas CL, Palmer TJ, Shipley P. Negative pressure pulmonary edema after a tonsillectomy and adenoidectomy in a pediatric patient: care report and review. AANA J. 1999;67:425–30.
- Thiagarajan RR, Laussen PC. Negative pressure pulmonary edema in children-pathogenesis and clinical management. Pediatr Anesth. 2007;17:307–10.
- Berkowitz RG, Zalzal GH. Tonsillectomy in children under 3 years of age. Arch Otolaryngol Head Neck Surg. 1990;116:685–6.

- 97. Chowdhurry K, Tewfik TL, Scloss MD. Post-tonsillectomy and adenoidectomy hemorrhage. J Otolaryngol. 1988;17:4–49.
- Collison PJ, Mettler B. Factors associated with post-tonsillectomy hemorrhage. Ear Nose Throat J. 2000;79:40–42, 644, 646.
- 99. Fidkowski CW, Zheng H, Firth PG. The anesthetic considerations of tracheobronchial foreign bodies in children: a literature review of 12,979 cases. Anesth Analg. 2010;111:1016–25.
- 100. Mani N, Soma M, Massey S, et al. Removal of inhaled foreign bodies-middle of the night or the next morning? Int J Pediatr Otorhinolaryngol. 2009;73:1085–9.
- Stubberud ES, Jacobs JP, Harmel Jr RP, et al. Successful reconstruction of traumatic carinal tissue loss using the esophagus in an infant. Ann Thorac Surg. 2007;84:1031–3.
- 102. Litman RS, Ponnuri J, Trogan I. Anesthesia for tracheal or bronchial foreign body removal in children: an analysis of ninety-four cases. Anesth Analg. 2000;91:1389–91.
- 103. Soodan A, Pawar D, Subramanium R. Anesthesia for removal of inhaled foreign bodies in children. Paediatr Anaesth. 2004;14: 947–52.
- 104. Mishra P, Chengode S, Narayanan A, et al. Utility of LMA for emergency tracheostomy in an infant with Pierre Robin syndrome. Pediatr Anaesth. 2009;19:409–10.
- 105. Stricker PA, Budac S, Fiadjoe JE, et al. Awake laryngeal mask insertion followed by induction of anesthesia in infants with the Pierre Robin sequence. Acta Anaesthesiol Scand. 2008;52:1307–8.
- 106. Wrightson F, Soma M, Smith JH. Anesthetic experience of 100 pediatric tracheostomies. Pediatr Anesth. 2009;19:659–66.
- 107. Gallagher TQ, Derkay CS. Pharmacotherapy of recurrent respiratory papillomatosis: an expert opinion. Expert Opin Pharmacother. 2009 Mar;10(4):645–55.
- Crockett DM, McCabe BF, Shive CG. Complications of laser surgery for recurrent respiratory papillomatosis. Ann Otol Rhinol Laryngol. 1987;96:639–44.

- 109. Mortensen M, Woo P. An underreported complication of laryngeal microdebrider: vocal fold web and granuloma: a case report. Laryngoscope. 2009;119:1848–50.
- 110. Garden JM, O'Banion MK, Bakus AD, et al. Viral disease transmitted by laser generated plume (aerosol). Arch Dermatol. 2002;138:1303–7.
- 111. Keon TP. Anesthetic considerations for laser surgery. Int Anesthesiol Clin. 1988;26:50–3.
- 112. Landsman IS, Werkhaven JA, Motoyama EK. Anesthesia for pediatric otorhinolaryngologic surgery. In: Motoyama EK, Davis PJ, editors. Smith's anesthesia for infants and children. 7th ed. Philadelphia: Mosby Elsevier; 2006. p. 789–822.
- 113. Shapiro AM, Rimell FL, Shoemaker D, et al. Tracheotomy in children with juvenile-onset recurrent respiratory papillomatosis: the Children's Hospital of Pittsburgh experience. Ann Otol Rhinol Laryngol. 1996;105(1):1–5.
- 114. Cohen SR, Herbert WI, Thompson JW. Anesthesia management of micorlaryngeal laser surgery in children: apneic technique anesthesia. Laryngoscope. 1988;98:347–8.
- Frantz TD, Rasgon BM. Acute epiglottitis: changing epidemiology patterns. Otolaryngol Head Neck Surg. 1993;109:457–60.
- 116. Adams WG, Deaver KA, Cochi Sl, et al. Decline of childhood Haemophilus influenza type b (Hib) disease in the Hib vaccine era. JAMA. 1993;269:221–6.
- Rittichier KK. The role of corticosteroids in the treatment of croup. Treat Respir Med. 2004;3:139–45.
- Moore M, Little P. Humidified air inhalation for treating croup. Cochrane Database Syst Rev. 2006;3:CD002870.
- Zulliger JJ, Schuller DE, Beach TP, Garvin JP, Birck HG, Frank JE. Assessment of intubation in croup and epiglottitis. Ann Otol Rhinol Laryngol. 1982;91:403–6.
- 120. Colles CJ. On stenosis of the trachea after tracheotomy for croup and diphtheria. Ann Surg. 1886;3:499–507.

Office-Based Otolaryngology

Laurence M. Hausman

21

Introduction

When one considers surgery of the head and neck, they generally think of an operating room in a large hospital. This traditional idea of surgery and anesthesia is understandable, but with the development of less invasive surgeries and "faster-acting" anesthetics with fewer hemodynamic side effects ^[1, 2], many surgeries have moved into a yet smaller environment, namely the private surgical office. This migration has not been sudden but rather stems from changes in health-care delivery that have been occurring over the past several decades.

Surgical procedures have long been shifting to ambulatory settings. In fact by 1998, 60–70% of all surgical procedures were being done on an outpatient basis ^[3]. Of these ambulatory procedures, many are now being done neither in hospitals nor freestanding ambulatory surgery units, but in private doctors' and dentists' offices. In fact, it is estimated that 12 million procedures (approximately 16% of all ambulatory procedures) are performed in this venue ^[4]. This is not to say that office-based anesthesia and surgery is a completely new innovation. On the contrary, there have been reports of office-based surgery and anesthesia for centuries. In 1856, John Snow reported his experience in providing anesthesia with chloroform for 867 dental extraction procedures ^[5].

In this chapter the unique principles and considerations of office-based surgery and anesthesia will be reviewed followed by a discussion of the specifics of office-based otolaryngology. A detailed discussion on establishing a safe and efficient office-based anesthetic practice is included for those otolaryngologists anesthesiologists interested in pursuing such an endeavor.

Hospital-Based Versus Office-Based Surgery and Anesthesia

How does an office-based anesthesia and surgical practice differ from a hospital-based or freestanding ambulatory one? surgery center. The most obvious difference is that an office-based surgical practice is entirely housed within a doctor's office; one in which other administrative and clinical functions occur. These activities will often include initial patient consultation, physical exams, follow-ups, as well as administrative duties. While office-based practices will generally be confined to one specialty, such as gastroenterology, urology, plastic surgery, otolaryngology or dentistry ^[6, 7], a free-standing ambulatory surgery center will usually accommodate a breadth of surgical subspecialties.

In order to operate, freestanding ambulatory surgery center are required to apply for and receive a "certificate of need" from the state. These centers must also usually have state licensure and/or accreditation by a major accrediting body. An office-based operating room, on the other hand, does not require a "certificate of need" and thus will likely not be licensed and may not even be accredited, depending on the state in which the office is located. This lack of oversight implies that the assurance of patient safety rests almost entirely upon the physicians who practice within the office.

Despite potential patient safety issues, office-based procedures are increasing in number mainly because they have many advantages when compared to hospital-based ones, including: ease of scheduling, improved patient privacy, fewer nosocomial infections, patient preference, and cost containment ^[8–15]. Private surgeons often not only find it easier to schedule cases and have the convenience of operating and performing pre- and postoperative care in the same location, but in some cases they may even collect an enhanced professional fee.

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Office-Based Otolaryngology and Anesthesia

Specific to otolaryngology practices, office-based procedures are often viewed by patients as being less invasive than ones involving general anesthesia within a hospital ^[16]. Thus, patients themselves are helping to drive the popularity and acceptance of surgery within this venue. Approximately 100 years ago, Chavalier Jackson introduced the first rigid, distal lighted esophagoscope. Examination of the awake patient was routinely performed using his techniques for many years to follow. Then, during the 1960s, with the advent of the operating room microscope and new modes of ventilation (jet ventilation, smaller endotracheal tubes, etc.), general anesthesia was performed for all types of larvngeal procedures including polypectomies and foreign body removal ^[17]. Later, in the 1970s with the introduction of flexible fiberoptic bronchoscopes, examinations could be performed safely in the office with minimal to no sedation and only topical anesthesia (Chap. 10)^[17].

Over the past decade, many types of office-based otolaryngologic procedures have been described. These include: treatment of glottal papillomatosis and dysplasia ^[18], laryngeal steroid injections ^[19, 20], ultrasound-guided fine needle aspiration and biopsies ^[21], endoscopic incisional biopsies of nasal masses ^[22], laryngoplasty ^[23], vocal cord polypectomies ^[24], vocal cord injections ^[25], nasal endoscopies with biopsy, polypectomy, or debridement, turbinate reductions, sinus endoscopy ^[26–28], CO₂ laser laryngeal surgery ^[29], laryngeal foreign body removal ^[30,31], and placement of pressure equalization tubes ^[14,32].

Interestingly, most of these procedures were described as being performed with only local anesthesia without the aid of sedation or intravenous agents. Employing local anesthesia without sedation for laryngeal procedures has many challenges. Patient selection becomes rather strict and limited as complete patient cooperation is crucial. The patient must not have an anxiety disorder or be very anxious about the procedure, they must be able to sit upright for 10–15 min without moving their head (eliminating all patients with a tremor), and they must have a patent nasal airway of at least 3-4 mm ^[33]. Some of the benefits of avoiding any type of sedation may include elimination of the need for preoperative fasting and perioperative monitoring. Cost reduction is appreciated with the removal of the storing and administering of sedation drugs or the need for post sedation recovery. However, local anesthesia alone is not without its share of risks. Patients may experience pain with associated hemodynamic responses even during a relatively noninvasive procedure [34]. Even seemingly benign procedures, such as a flexible endoscopy, may provoke a generalized sympathetic response. Yung et al. recently described significant tachycardia and hypertension during flexible endoscopy [35]. One patient in this report had the procedure aborted secondary to dangerously high systolic

and diastolic blood pressures. Further, local anesthetic toxicity is always a danger secondary to excess systemic absorption or from intravascular or intrathecal injection. Hence, in these cases the lack of an intravenous becomes a liability.

The Anesthesiologist's Role in Office-Based Otolaryngologic Procedures

Just as has been the case with virtually every other surgical subspecialty, the need for anesthesiologists to provide a safe anesthetic in the private otolaryngologist's office exists and will continue to rise. Every anesthesia provider working in an office setting must be capable of assuring that a surgical office is a safe anesthetizing location. This determination is made from a number of factors including: the physical office itself focusing on its layout and design, the administrative staff, the governing body, the policies and procedures, the practitioners' qualifications, patient selection, procedure selection, the operating room, the post-anesthesia care unit and patient follow-up.

Deciding Upon an Office-Based Procedure

It is intuitive that an office-based surgical center cannot offer a level of service identical to that offered at a tertiary care hospital or even a small private hospital. The office will by its very nature have a more limited staff, laboratory capabilities, and ancillary services. However, any practitioner who embarks on a procedure in the office must be able to provide the patient with the same level of care for that limited procedure. Accordingly, the office should be staffed and equipped to handle any intra or post-procedure complication. Therefore, not all patients or procedures are suitable for the office setting. For instance, patients with significant comorbidities (i.e., morbid obesity, obstructive sleep apnea, chronic obstructive pulmonary disease, significant coronary artery disease), potentially difficult airways, or those at risk for aspiration should likely be deferred to the hospital for most procedures. Likewise, procedures associated with severe postoperative pain, the need for intensive postoperative monitoring, potential for significant blood loss or fluid shifts, or requirements for invasive monitoring are not ideal candidates for this surgical venue. Ultimately though, each office should decide for itself what procedures they will allow to be performed and on whom. The American Society of Anesthesiologists (ASA) has published recommendations regarding which patients and procedures are good candidates for an office-based procedure (Table 21.1) [36].

 Table 21.1
 Patients and procedures in which office-based anesthesia

 and surgery may not be appropriate

Patients with morbid obesity		
Patients with a history of a difficult airway		
Patients with a history of substance abuse		
Patients with a latex allergy		
Patients with brittle diabetes, chronic obstructive pulmonary disease, or cardiovascular disease		
Patients with a history of malignant hyperthermia		
Patients or procedures requiring invasive monitoring		
Procedures associated with significant fluid shifts or blood loss		
Procedures associated with significant postoperative pain and/or nausea and vomiting		
Procedures greater than 6 h in duration		

It is imperative that the office-based practitioner practice under the edict that any procedure performed in the office must be as safe as if it was performed in a hospital or ambulatory surgery center. Therefore, the anesthesiologist must be aware that all perioperative guidelines mandated by the ASA must be adhered to and apply equally to the office-based setting ^[37].

Office-Based Safety

Is it really possible to provide a patient with same level of safety in an office setting as within a hospital? There have been many anecdotal reports both in the medical and lay press suggesting that an office-based procedure is riskier than the standard hospital-based procedure ^[38–40]. In 2003, Vila reported a 10% increase in morbidity and mortality for office-based surgery when compared to that within a more traditional setting [41]. However, just as there have been reports of the pitfalls of office-based surgery, there have been many reports claiming that it is a safe and viable alternative ^[42–46]. These widely differing reports may be due to several factors. Although mandated by several states, by and large, most reporting of morbidity and mortality is voluntary and relies on self-reporting. Further, the health-care professionals delivering the anesthetics in offices often have very different levels of training. Some offices employ anesthesiologists, others use certified registered nurse anesthetists supervised by an anesthesiologist or even by the surgeon performing the procedures, and still others utilize dental anesthetists. Further compounding the difficulty in determining the true morbidity and mortality rate is the fact that modern day anesthesia is quite safe, with morbidity and mortality estimated at approximately 1/200,000-400,000. Thus, in order to accurately calculate true morbidity and mortality

rates, very large sample sizes would be required. As of this writing, the true level of safety in the office-based setting is still a matter of debate.

Developing an Office-Based Practice

While the level of care provided in an office-based setting is still controversial, physicians must attempt to ensure the best possible care for their patients. Office selection including physical design and layout, staffing and administration, physician qualifications, procedure selection and patient selection are considerations of the utmost importance (Table 21.2) ^[36].

Office Selection

A surgical office must be of ample size to perform the intended procedure and recover the patient. There must be adequate room within the operating suite to perform the procedure and allow for patient monitoring equipment. There must also be space for the anesthesia provider at the head of the patient to monitor and/or secure the airway, and at both sides of the patient to provide advanced cardiac life support should the need arise (i.e., 360° access is necessary should an emergency occur). The office should of course be clean, well lit, and ventilated and have adequate emergency egress for an anesthetized patient on a stretcher. Hallways and doorframes should be wide enough to accommodate an anesthetized patient in a stretcher being manually ventilated. Arm rails in the hallways and stairwells should terminate on the floor or in such a way as not to "catch" on to clothing as people rush by.

The operating room must be equipped with a reliable source of oxygen as well as backup and vacuum/suction. There must be a means of delivering oxygen both passively (e.g., nasal cannula or face mask) and by positive pressure ventilation. Positive pressure ventilation can be administered via a manual self-inflating resuscitation device, a freestanding ventilator or an anesthesia machine. Although a modern computerized anesthesia workstation is not mandated, the anesthesia machine must meet modern ASA standards ^[47]. If volatile anesthetics are being used, a functioning scavenging system must be in place. All emergency equipment and medications should be immediately available. If succinylcholine is stocked, or if volatile agents are used, a malignant hyperthermia cart with dantrolene must also be present.

Many practitioners spend a great deal of time assuring that the operating room is a safe anesthetizing location, and often overlook the adequacy of the post-anesthesia care unit

Table 21.2 Evaluating a new office-based setting

- 1. Surgical suite of adequate size, well lit, and ventilated
- 2. Presence of adequate number of properly maintained physiologic monitors with battery backup for operating room and post-anesthesia care unit
- 3. Source of oxygen with backup
- Presence of adequate vacuum and/or scavenging if anesthesia machine present
- 5. Presence of device(s) for passive and manual ventilation
- Post-anesthesia care unit of adequate size, well lit and ventilated
- 7. Properly maintained "crash cart" with defibrillator
- 8. Malignant Hyperthermia cart (when applicable)
- 9. Double lock narcotic storage cabinet
- 10. Accreditation status
- 11. Physician credentialing and privileging
 - (a) License and registration
 - (b) DEA certificate
 - (c) Malpractice
 - (d) CV
 - (e) ACLS certification
- 12. Policy and procedures manual
 - (a) Governance
 - (b) Patient care
 - (c) Quality improvement
 - (d) Peer review
 - (e) Emergency preparedness
 - (f) Risk assessment
 - (g) Operating room procedures
 - (h) Job responsibilities

(PACU) equipment and staffing. This can lead to a postoperative disaster. Domino reported that based on the anesthesia closed claims database, 100% of all respiratory events in the PACU of an office-based practice that led to patient morbidity could have been prevented by the use of an oxygen saturation monitor ^[48]. Equally critical to the use of an oxygen saturation monitor is assurance that there are adequately trained PACU personnel responsible for monitoring and treating the patient appropriately.

The post-anesthesia care unit, if one exists must also be clean, well lit and of ample size. It should be staffed according to the number of recovering patients present at any one time. Physiologic monitoring, with battery backup power must be utilized and documented at regular intervals on all recovering patients in accordance with ASA standards [49-51]. The ability to monitor a continuous electrocardiogram (ECG), a noninvasive blood pressure monitor and an oxygen saturation monitor is mandatory. In addition to these monitors, the operating suite should have an end tidal carbon dioxide (ETCO₂) monitor and a means to check patient temperature. The PACU should also be equipped with a supplemental oxygen source and suction. A crash cart with a defibrillator must be present. The defibrillator must be checked daily as documented with the maintenance of a written log. All monitors and equipment must undergo preventative maintenance according to manufacturer's specifications.

Physician Qualifications

All practicing physicians should be qualified to perform the procedures they plan on performing in an office. They should have an active state medical license and registration, a current drug enforcement authority (DEA) certificate, adequate malpractice insurance, and post-graduate training commensurate with their responsibilities. There should be a current curriculum vitae (CV) on file for each practitioner that reflects his/ her education and training. It is recommended that the physician be either board certified or board eligible by the American Board of Medical Specialties. It is vital that an independent second party such as the American Medical Association or the National Practitioners Database verify all information on the physician's CV. It may be helpful to require that each practitioner has privileges to perform each procedure they seek to perform in the office, at a local hospital.

It would be prudent to ensure that all physicians be certified in basic life support (BLS). At a minimum, all anesthesia providers should carry current certification in advanced life support (ACLS) and/or pediatric advanced life support (PALS) if the office performs surgery on children. It is suggested by the author that the nurses also be certified in both BLS and ACLS. Regardless, there should be a licensed health-care provider with BLS and ACLS/PALS certification immediately available in the office until the last patient is physically discharged.

All physicians should maintain continuous medical education (CME) activities. Additionally, all should be involved in a quality improvement and peer review program. In the case of a solo practitioner, a qualified peer from the area should be asked to periodically and routinely perform peer/ chart review. In addition to random selection of charts for review, predefined sentinel events should trigger an occurrence chart review. Examples of sentinel events include patient reintubation, return to the operating room, prolonged postoperative nausea and vomiting, nerve injury, medication error, infection, and unplanned admission to the hospital.

The office-based quality improvement program should consist of members through the various levels of the office organization and should meet regularly and documented with written minutes. Since there is some variation throughout the country with regard to protected information and its discoverability, it is the authors' recommendation that every officebased practice consult with counsel as to best protect the information shared at these meetings.

Policies and Procedures

There should be a written policy and procedures manual that clearly defines the organization as a legal entity. Even in the case of a solo practice, there should be a medical director and a governing board. There should be policies in place for credentialing and privileging all physicians and health-care workers. This again is even necessary in the case of a solo practice. Ancillary staff job descriptions and job training should be clearly delineated. Ongoing safety in the workplace education should be mandated for all employees. Patient rights and responsibilities should also be addressed within the policies and procedures manual.

All Occupational Safety and Health Administration (OSHA) rules must be followed. State and local laws regarding the transport, storage, and discarding of medical gases and controlled substances must be defined within the policies. There should be routine emergency drills including, but not limited to, fire, cardiopulmonary arrest, and malignant hyperthermia (if triggering agents, volatile agents, succinylcholine are stocked). Patient charts must be securely maintained and in accordance with state, local, and federal law. There should exist a written transfer agreement to a local hospital for any unplanned admissions.

Office Accreditation

One way of attempting to standardize and improve the quality of care between surgical offices is through accreditation. The Joint Commission (TJC) has long been responsible for accrediting inpatient hospitals, and this process has likely improved the quality of care within these institutions. Although hospital accreditation is considered voluntary, governmental and third party reimbursement is hinged to the hospitals' participation. Therefore, virtually every hospital must be accredited for financial viability.

In addition to TJC, the Accreditation Association for Ambulatory Health Care (AAAHC) and the American Association for Accreditation of Ambulatory Surgical Facilities (AAAASF) are recognized entities empowered to accredit office-based practices. Although accreditation is voluntary in some states, most require accreditation for surgical offices in which any depth of anesthesia greater than minimal sedation is offered. As of 1996, the American Society of Plastic Surgeons (ASPS) mandated that its membership only operate in accredited offices. Whether or not accreditation is linked to improved quality of care has not yet been determined. Although somewhat controversial, some authors have found that there is actually no connection between office accreditation and safety ^[52].

Each accrediting organization is unique, but they all look at, and have similar requirements for accreditation of an office-practice. As discussed previously in this chapter, each organization will look at attributes such as governance, patient care, facilities, management of medical records, anesthesia care, infection control, peer review, risk management, quality improvement, and surgical and postoperative care, amongst other aspects. A complete look at each organization's requirements can be found on their respective websites: www.jointcommission.org, www.aaaasf.org, and www.aaahc.org.

Patient and Procedure Selection

Regardless of the accreditation status and the technologic and personnel resources of the office, not all patients and/or procedures are suitable for an office-based anesthetic. Immediate consultation with colleagues, stat laboratories, invasive monitoring, blood products, and intensive postoperative monitoring are generally not available. Thus, any patient or procedure in which there is a chance that one or more of these elements may be necessary should be referred to a facility with the necessary resources. The ASPS has published recommendations for its members identifying the types of surgeries and patient populations that are and are not ideal for an office-based procedure ^[53, 54]. Likewise, the ASA has made similar recommendations ^[36].

Generally ASA physical status 1 and 2 patients undergoing procedures that are not associated with significant postoperative pain, fluid shifts, hypothermia, or postoperative nausea and vomiting are ideal patients. Patients with significant comorbidities or those with potentially difficult airways are generally not considered good candidates for surgery in these remote sites.

Patients with OSA are complex for a number of reasons. They often possess potentially difficult airways. Additionally, they commonly have other significant comorbidities such as systemic and pulmonary hypertension, coronary artery disease, or heart failure. OSA patients have complex postoperative courses. Most of these patients will require overnight oxygen saturation monitoring and at a minimum must remain in the PACU for upwards of 3–7 h depending on whether they have had an apneic event during their recovery ^[55, 56]. It would therefore seem prudent to eliminate this patient population from an office-based setting, especially for procedures requiring general anesthesia and the need for postoperative narcotics.

Conclusion

Office-based anesthesia in the field of otolaryngology can be a safe and efficient use of medical resources. This unique venue is often favored by physicians and patients alike. Safety during an office-based procedure is the obligation of each of the health-care providers working in that setting. Many medical societies including the ASPS and the ASA are champions of this surgical site and have provided practitioners with a number of recommendations and guidelines. Keys to success rely on the proper office, patient and procedure selection.

References

- Tang J, White PF, Wender RH, et al. Fast-track office-based anesthesia: a comparison of propofol versus desflurane with antiemetic prophylaxis in spontaneously breathing patents. Anesth Analg. 2001;92(1):95–9.
- White PF, Song D. New criteria for fast-tracking after outpatient anesthesia: a comparison with the modified Aldrete's scoring system. Anesth Analg. 1999;88(5):1069–72.
- Owings MF, Kozac LJ. Ambulatory and inpatient procedures in the United States, 1996. Vital Health Stat. 1998;13:1–119.
- American Hospital Association. Chart 2.5: percent of outpatient surgeries by facility type, 1981–2005. Available at http://www.aha.org/ aha/trendwatch/chartbook/2-7/07chapter2.ppt#265.8. Accessed 1 Mar 2012.
- Snow J. On chloroform and other anesthetics. London: John Churchill; 1858. p. 314–5.
- Twersky R. Office-based anesthesia: challenges and success. Available at http://www.csaol.cn/img/2007asa/rcl_src/204_twersky.pdf. Accessed 1 Mar 2012.
- Maurer W. Office-based anesthesia: a critical look. Available at http://cms.clevelandclinic.org/body.cfm?wyzpdqabs=o&id=227& action=detail&ref=640. Accessed 1 Mar 2012.
- 8. Byrd HS, Barton FE, Orenstein HH, et al. Safety and efficacy in an accredited outpatient plastic surgery facility: a review of 5316 consecutive cases. Plast Reconstr Surg. 2003;112(2):636–41.
- Rees CJ, Postma GN, Koufman JA. Cost savings of unsedated office-based laser surgery for laryngeal papillomas. Ann Otol Rhinol Laryngol. 2007;116:45–8.
- Bove MJ, Jabbour N, Krishna P, et al. Operating room versus office-based injection laryngoplasy: a comparative analysis of reimbursement. Laryngoscope. 2007;117:226–30.
- Morris KT, Pommier RF, Vetto JT. Office-based wire-guided open breast biopsy under local anesthesianis accurate and cost effective. Am J Surg. 2000;179:422–5.
- Rees CJ, Halum SL, Wijewickrama RC, et al. Patient tolerance of in-office pulsed dye laser treatments to the upper aerodigestive tract. Otolaryngol Head Neck Surg. 2006;134:1023–7.
- Catalano PJ, Choi E, Cohen N. Office versus operating room insertion of bone-anchored hearing aid: a comparative analysis. Otol Neurol. 2005;26:1182–5.
- Siegel GJ, Chandra RK. Laser office ventilation of ears with insertion of tubes. Otolaryngol Head Neck Surg. 2002;127:60–6.
- Akbar NA, Bodenner DL, Kim LT, et al. Considerations in incorporating office-based ultrasound of the head and neck. Otolaryngol Head Neck Surg. 2006;135:884–8.
- Woo P. Office-based laryngeal procedures. Otolaryngol Clin North Am. 2006;39:111–33.
- Koufman JA. Introduction to office-based surgery in laryngology. Curr Opin Otolaryngol Head Neck Surg. 2007;15:383–6.
- Zeitels SM, Akst LM, Burns JA, et al. Office-based 532-nm pulsed ktp laser treatment of glottal papillomatosis and dysplasia. Ann Otol Rhinol Laryngol. 2006;115:679–85.

- Mortensen M, Woo P. Office steroid injections of the larynx. Laryngoscope. 2006;116:1735–9.
- Bastian RW, Delsupehe KG. Indirect larynx and pharynx surgery: a replacement for direct laryngoscopy. Laryngoscope. 1996;106: 1280–6.
- Smith RB. Ultra-sound guided procedures for the office. Otolaryngol Clin North Am. 2010;43:1241–54.
- 22. Han MW, Lee BJ, Jang YJ, et al. Clinical value of office-based endoscopic incisional biopsy in diagnosis of nasal cavity masses. Otolaryngol Head Neck Surg. 2010;143:341–7.
- Tirado Y, Lewin JS, Hutcheson KA. Office-based injection layngoplasty in the irradiated larynx. Laryngoscope. 2010;120:703–6.
- Lan MC, Hsu YB, Chang SY, et al. Office-based treatment of vocal cord polyp with flexible laryngovideostroboscopic surgery. J Otolaryngol Head Neck Surg. 2010;39:90–5.
- Zeitler DM, Amin MR. The thyrohyoid approach to in-office injection augmentation of the vocal fold. Curr Opin Otolaryngol Head Neck Surg. 2007;15:412–6.
- Armstrong M. Office-based procedures in rhinosinusitis. Otolaryngol Clin N Am. 2005;38:1327–38.
- Belafsky PC. It's time to wake up and smell the lidocaine. Curr Opin Otolaryngol Head Neck Surg. 2007;15:381–2.
- Siegel GJ, Seiberling KA, Aguado AS. Office CO₂ laser turbinoplasty. Ear Nose Throat J. 2008;87:386–91.
- 29. Halum SL, Moberly AC. Patient tolerance of the flexible CO₂ laser for office-based laryngeal surgery. J Voice. 2010;24:750–4.
- Sato K, Nakashima T. Office-based foreign body management using videoendoscope. Am J Otolaryngol. 2004;25:167–72.
- Sato K, Nakashima T. office-based videoendoscopy for the hypopharynx and cervical esophagus. Am J Otolaryngol. 2002;23: 341–4.
- Brodsky L, Brookhauser P, Chait D, et al. Office-based insertion of pressure equalization tubes: the role of laser-assisted tympanic membrane fenestration. Laryngoscope. 1999;109:2009–14.
- Rosen CA, Amin MR, Sulica L, et al. Advances in office-based diagnosis and treatment in laryngology. Laryngoscope. 2009;119:S185–212.
- Postma GN, Cohen JT, Belafsky PC, et al. Transnasal esophagoscopy; revisited (over 700 consecutive cases). Laryngoscope. 2005;115:321–3.
- Yung KC, Courey MS. The effect of office-based flexible endoscopic surgery on hemodynamic stability. Laryngoscope. 2010; 120:2231–6.
- American Society of Anesthesiologists. Office-based anesthesia: considerations for anesthesiologists in setting up and maintaining a safe office anesthesia environment. 2nd ed. Parkridge: ASA; 2008.
- American Society of Anesthesiologists. Guidelines for office-based anesthesia. Available at http://www.asahq.org/publicationsAnd-Services/standards/12.pdf.
- Maier T. Risky operations? Newsday. September 15, 2000. Available at http://www.newsday.com/news/health/esurg17.htm. Accessed 1 Mar 2012.
- Quattrone MS. Is the physician office the wild, west of health care? J Ambul Care Manage. 2000;23:64. Accessed 1 Mar 2012.
- 40. Zuger A. Surgeons leaving the O.R. for the office. The New York Times. May 18, 1999.
- Vila H, Soto R, Cantor AB, Mackey D. Comparative outcomes analysis of procedures performed in physician offices and ambulatory surgery centers. Arch Surg. 2003;138:991–5.
- 42. Hoefflin SM, Bornstein JB, Gordon M. General anesthesia in an office-based plastic surgical facility: a report on more than 23,000 consecutive office-based procedures under general anesthesia with no significant anesthetic complications. Plast Reconstr Surg. 2001;107:243–57.

- Keyes GR, Singer R, Iverson RE, et al. Analysis of outpatient surgery center safety using an internet- based quality improvement and review program. Plast Reconstr Surg. 2004;113(6):1760–70.
- Morello DC, Colon GA, Fredericks S, Iverson RE, Singer R. Patient safety in accredited office surgical facilities. Plast Reconstr Surg. 1997;99:1496–9.
- Keyes GR, Singer R, Iverson RE, et al. Mortality in outpatient surgery. Plast Reconstr Surg. 2008;122:245–50.
- 46. Bitar G, Mullis W, Jacobs W, et al. Safety and efficacy of officebased surgery with monitored anesthesia care/sedation in 4778 consecutive plastic surgery procedures. Plast Reconstr Surg. 2003;111(1):150–6.
- 47. American Society of Anesthesiologists. Guidelines for determining anesthesia machine obsolescence. Available at http://www.asahq. org/publicationsAndServices/machineobsolescense.pdf.
- Domino KB. Office-based anesthesia: lessons learned from the closed-claims project. ASA Newsl. 2001;65:9.
- American Society of Anesthesiologists. Standards for basic anesthetic monitoring, last amended 2005. Available at http://www. asahq.org/publicationsAndServices/standards/02.pdf. Accessed 1 Mar 2012.
- American Society of Anesthesiologists. Practice guidelines for postanesthetic care. Available at http://www2.asahq.org/

publications/pc-181-4-practice-guidelines-for-postanesthetic-care. aspx.

- American Society of Anesthesiologists. Standards for postanesthesia care, last amended 2009. Available at http://www.asahq.org/ publicationsAndServices/standards/36.pdf. Accessed 1 Mar 2012.
- Coldiron B, Shreve E, Balkrishnan R. Patient injuries from surgical procedures performed in medical offices: three years of Florida data. Dermatol Surg. 2004;30:1435–43. Accessed 1 Mar 2012.
- Iverson R. ASPS task force on patient safety in office-based surgery facilities. Patient safety in office-based surgery facilities: I. Procedures in the office-based surgery setting. Plast Reconstr Surg. 2002;110:1337. Accessed 1 Mar 2012.
- Iverson RE, Lynch DJ. ASPS task force on patient safety in officebased surgery facilities. Patient safety in office-based surgery facilities: II. Patient selection. Plast Reconstr Surg. 2002;110:1785.
- 55. A report by the American Society of Anesthesiologists Task Force on perioperative management of patients with obstructive sleep apnea. Practice guidelines for the perioperative management of patients with obstructive sleep apnea. Anesthesiology. 2006;104; 1081–93.
- Kaw R, Michota F, Jaffer A, et al. Unrecognized sleep apnea in the surgical patient: implications for the perioperative setting. Chest. 2006;129:198–205.

Acute and Chronic Pain Management

22

Yury Khelemsky

Introduction

The high prevalence of acute and chronic pain, its psychological, physiological, and financial implications, as well as emerging trends of quality of care assessment and pay-for-performance necessitate a robust and multidisciplinary approach to pain management ^[1–3]. Pain must be considered and addressed during the preoperative, perioperative, and postoperative periods. Collaboration between surgeons, anesthesiologists, and pain medicine specialists is critical for the optimization of outcomes.

This chapter is divided into two sections. The first section will focus on acute perioperative pain management, detailing the concepts of preemptive and multimodal analgesia, intraoperative management, and postoperative care. Chronic pain management, with a focus on head and neck pain syndromes including craniofacial pain, cancer pain, headache, and neck pain will be covered in the remaining section. This chapter serves as a fairly comprehensive, but basic overview of acute and chronic pain management for both the otolaryngologists and anesthesiologists.

Background

Definition of Pain

Pain is defined as "an unpleasant sensory and emotional experience associated with actual or potential tissue damage." For practitioners it is therefore critical to appreciate that pain is subjective and an identifiable etiology or pathophysiology is an unnecessary prerequisite for pain to occur. Acute pain is defined as an anticipated response to a noxious mechanical, thermal, or chemical stimulus, while pain is considered chronic when it lasts longer than the anticipated normal healing time ^[4]. Pain can further be classified as nociceptive or non-nociceptive. Nociceptive pain has an identifiable etiology caused by tissue damage, while non-nociceptive pain is generally neuropathic pain caused by either central nervous system or peripheral nerve dysfunction or lesions ^[5].

Assessment of Pain

Since the perception of pain is subjective, several tools were developed to quantify and standardize the intensity of pain reported for patient care, research, and quality assurance processes. The visual analog pain scale (VAS) and verbal numerical rating scale (VNRS) (Fig. 22.1) are frequently used as pain assessment tools. The VAS scale is a linear scale that is 10-cm in length bordered by the descriptors "no pain" and "worst imaginable pain," while the VNRS simply requires the patient to assign a number between 0 and 10 (0 corresponds to no pain and 10 the worst pain imaginable) that corresponds to their level of pain. The VAS is a sensitive way of assessing pain change in a particular individual and has been found to correlate with the VNRS ^[6, 7].

Scales such as the Faces Pain Scale Revised (FPS-R) (Fig. 22.2), Verbal Descriptor Scale (VDS), Numeric Rating Scale (NRS), and Iowa Pain Thermometer (IPT) can be used for cognitively impaired adults or infants and children; however, simple observation of pain behavior may be the most effective tool in detecting pain in these populations ^[7, 8]. Cognitively impaired and preverbal children can be also effectively assessed with the FLACC (Face, Legs, Activity, Cry, Consolability) tool ^[9].

Physiology and Pharmacology of Pain

The physiology of pain is complex and is modulated by a multitude of neurologic pathways, receptors, and chemical modulators. Understanding this complexity is critical to

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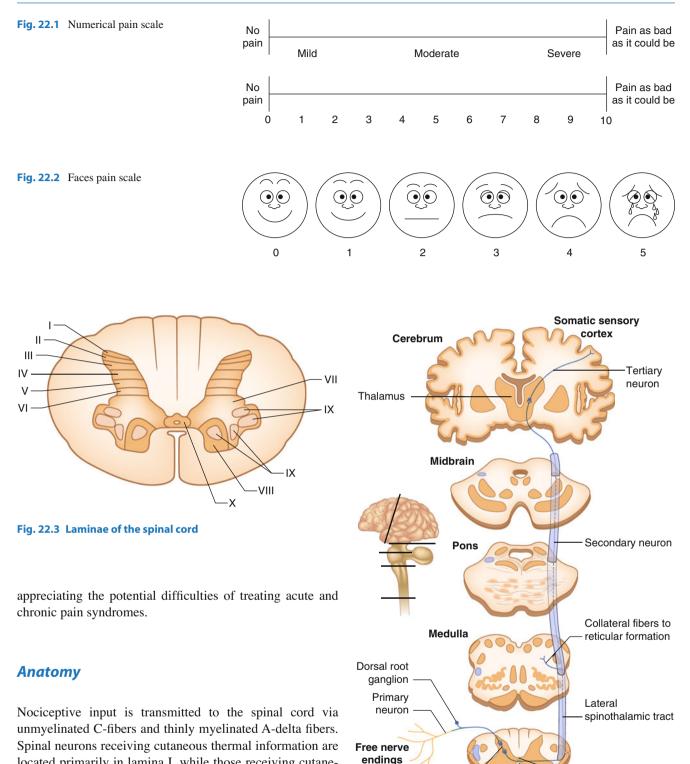
Gray commissure

White commissure

Spinal cord

The ventral posterolateral nucleus of the thalamus provides site-specific information to the somatosensory

cortex. Other thalamic nuclei relay nociceptive input to



Association neuron

Fig. 22.4 Major pain pathways

located primarily in lamina I, while those receiving cutane-

ous mechanical information are located in laminae IV and V.

Visceral input is received by neurons located in laminae III,

synaptic dorsal column pathway conducts primarily visceral

Two ascending pathways carry nociceptive information to the thalamus. The spinothalamic tract (Fig. 22.4) conducts primarily cutaneous nociceptive information and the post-

VII, and X (Fig. 22.3).

nociceptive input.

cingulate cortices, which generate affective responses to pain. Descending modulation of input adds another level of complexity to nociceptive signal transduction.

Physiology

Tissue injury prompts a release of multiple mediators from macrophages, mast cells, and blood vessels. These mediators, which include histamine, serotonin, bradykinin, arachidonic acid metabolites (i.e., PGE2, TXA2, etc.), cytokines (i.e., TNF- α), interleukins, thrombin, trypsin, nerve growth factor (NGF), hydrogen ions, potassium, calcitonin, and substance P, evoke spontaneous activity in C-fibers and decrease their stimulation threshold.

Primary afferent transmitters released at the level of the dorsal horn and corresponding receptors, include substance P (neurokinin 1, NK-1), CGRP (CGRP1), brain-derived nerve growth factor (TRK B), ATP (P2X, P2Y), aspartate and glutamate (AMPA, NMDA). In addition to excitatory input, inhibition of both first- and second-order neurons is exerted by release of glycine and gamma-aminobutyric acid (GABA) from specific interneurons. Endogenous opiates (enkephalins, dynorphins) modulate pain transmission at the level of the spinal cord, as well as the brain by binding to opiate receptors (i.e., mu, kappa, delta, etc.). Norepinephrine and serotonin are implicated in pain signaling in the brain.

Hyperalgesia (abnormally increased sensitivity to painful stimuli in the area surrounding injury) and allodynia (pain sensation in response to non-noxious stimuli) can occur secondary to release of local inflammatory mediators leading to sensitization of peripheral pain receptors or increased excitability of CNS (secondary hyperalgesia)^[10].

Pharmacology

Analgesic drugs interrupt or modulate transmission of signals along multiple points of the nociceptive pathways. For example, peripheral input is modulated by nonsteroidal antiinflammatory drugs (NSAIDS), steroids, capsaicin, and local anesthetics, among others. Central (spinal cord and brain) processing is modulated by a variety of pharmacologic agents including, opioids, ketamine, and other NMDA-receptor antagonists, alpha-2 agonists, tricyclic antidepressants, serotonin–norepinephrine reuptake inhibitors (SNRIs), acetaminophen, and NSAIDS.^[11].

Acute postoperative pain continues to be undermanaged. Even though there is a growing awareness of the importance of pain control, and pain is the most common concern of surgical patients, the vast majority of patients still experience at least a moderate level of postoperative pain^[12]. In the otolaryngologic population, patients who undergo oropharyngeal operations and those with preoperative pain are at increased risk for significant postoperative pain^[3]. Effective management of postoperative pain leads to increased patient satisfaction, reduced hospital costs, earlier mobilization, reduced postoperative morbidity, and shorter hospitalizations ^[13–16]. Inadequate control of acute postoperative pain increases morbidity and mortality, decreases quality of life, and increases the likelihood of developing persistent postoperative pain ^[12, 17]. Interestingly, patient satisfaction does not directly correlate with pain intensity. Satisfaction is, however, linked positively to staff responsiveness to pain complaints, use of patient controlled analgesia (PCA) over nurse-administered analgesia, and treatment involvement by a dedicated acute pain service [18, 19].

Treatment Plans: Multimodal and Preemptive Analgesia

Multimodal Analgesia

Traditionally, pain has been treated with single agents (opioids); however, it is likely that the use of several agents, each acting at a different site in the pain pathway, can result in synergism, better than an additive effect, between medications leading to decreased doses and a reduction of side effects ^[20]. The afferent pathways via which sensation of pain is transmitted may be modulated with multiple pharmacologic agents, such as local anesthetics, anti-inflammatory drugs (e.g., steroids, NSAIDS), alpha 2 agonists (i.e., clonidine, dexmeditomidine, etc.), *N*-methyl-D-aspartic acid (NMDA) receptor antagonists (i.e., ketamine, etc.), and anticonvulsants (i.e., neurontin, pregabalin, etc.) among others. Combining these medications (either locally or systemically) to treat pain constitutes multimodal analgesia.

Preemptive Analgesia

Administering anti-pain therapies prior to tissue injury is defined as preemptive analgesia. It is speculated that

preemptive analgesia alters peripheral and central nervous system processing of noxious stimuli that would lead to a reduction of both hyperalgesia and allodynia ^[10] and perhaps to a modulation of acute pain management needs . Of note, however, is that pre-incisional administration of these agents (excluding local anesthetics and anticonvulsants) does not seem to provide increased analgesic benefit when compared with post-incisional administration ^[21]. Therefore, the concept of preemptive analgesia is being replaced by the idea of preventive analgesia ^[22].

Local Analgesia

A meta-analysis of randomized trials found significantly decreased analgesic consumption and increased time to first rescue analgesic request, but no significant differences in postoperative pain scores in patients who had preemptive local anesthetic wound infiltration. A notable exception to this analysis is a randomized double blind trial that revealed that pre-incisional infiltration of local anesthetic and general anesthesia for pediatric tonsillectomy reduces posttonsillectomy pain and provides a more rapid return to activity when compared with general anesthesia alone ^[23]. Other randomized trials have shown that local anesthetic injection around small incision sites reduces postoperative somatic pain (arising from integumentary and musculoskeletal systems) relief, but is inadequate for visceral pain (arising from organs) ^[24-26].

The use of local anesthetic does seem to provide a reduction in post-tonsillectomy pain. Topical local anesthetic on swabs applied in the tonsillar bed post excision appears to provide a similar level of analgesia to that of infiltration without the potential adverse effects ^[27].

Local Multimodal Analgesia

The use or addition of adjunct medications to local anesthetics may be used to achieve improved local analgesia. The addition of clonidine (alpha-2 agonist) to a pre-incisional injection of ropivacaine (a local anesthetic with equal efficacy of bupivacaine in post-tonsillectomy pain), prior to tonsillectomy has a preemptive analgesic effect that outlasts the local anesthetic and decreases pain, opioid use, and the time to return to normal activity ^[28, 29]. Peritonsillar infiltration with tramadol, an opioid agonist, can provide good intraoperative analgesia, less postoperative pain on awakening, and lower analgesic requirement within the first hour after surgery ^[30]. A preliminary study suggested that a single dose of inhaled morphine administered preemptively prior to septoplasty or septorhinoplasty provides effective postoperative analgesia ^[31].

Systemic Preemptive Analgesia

A single, intraoperative dose of intravenous dexamethasone may reduce post-tonsillectomy pain on postoperative day 1, by a factor of 1 on a 10-point pain scale. Although it is suggested that the risk of postoperative bleeding is increased, it is believed that the side effects and cost of dexamethasone are quite negligible, that its routine use seems reasonable ^[32]. Dexamethasone along with the use of a transdermal patch of diclofenac, a nonsteroidal anti-inflammatory drug (NSAID), reduces opioid requirement and postoperative morbidity following tonsillectomy ^[33]. Ketamine, however, did not reduce post-tonsillectomy morphine consumption in children ^[34].

NSAIDS are not routinely administered to otolaryngologic surgery patients due to a concern about inhibition of platelet function and increased perioperative and postoperative bleeding. A study of pediatric patients undergoing tonsillectomy and adenoidectomy revealed no differences in postoperative bleeding rate in patients provided postoperative NSAID (ibuprofen) versus control, concluding that ibuprofen is not contraindicated in this setting and should be used in the control of postoperative pain, if otherwise indicated ^[35]. Evidence exists that NSAIDs can be used after induction of anesthesia in pediatric adenotonsillectomy and may have a preemptive analgesic effect while significantly reducing the incidents of nausea, vomiting, and narcotic use ^[36–40].

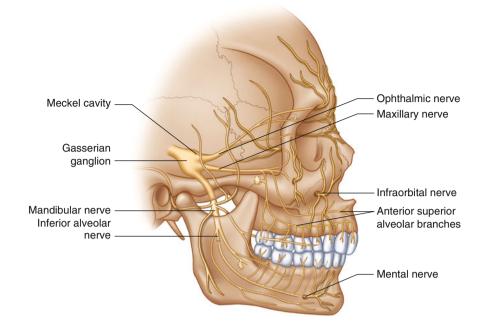
Although preoperative administration of GABA analogues such as gabapentin (Neurontin) and pregabalin (Lyrica) has been found to effectively reduce postoperative pain, opioid consumption, and opioid-related adverse effects after surgery in several patient populations, conclusions and recommendations about the use, optimal dose, and duration of the treatment, for otolaryngologic patients cannot be made at this time because of the heterogeneity of the trials ^[41, 42].

Acetaminophen, a centrally acting analgesic with a poorly understood mechanism of action, may have a preemptive analgesic effect and decrease postoperative opioid consumption ^[39, 43]. Combination of acetaminophen with NSAIDS may be more effective than use of acetaminophen alone ^[43].

Alpha-2 agonists (i.e., clonidine, dexmedetomidine, etc.) can exert a potent analgesic response. Oral clonidine at doses of 150–200 mcg preoperatively has been shown to provide perioperative hemodynamic stability and reduce the amount of postoperative analgesics ^[44, 45].

Preoperative anxiety may be associated with postoperative pain. In a prospective study, children who were assessed to be anxious before surgery experienced more pain and slower decline in pain during the first three postoperative days than children who were assessed to be not anxious before surgery. Therefore, behavioral anxiolysis with preparation of the child and caregiver for the surgical experience may provide a level of preemptive analgesia ^[46].

Fig. 22.5 Distribution of the trigeminal nerve



Perioperative Regional Anesthesia

Otolaryngologic practice may benefit from regional anesthesia as it can provide superior pain control, enhanced recovery, and earlier discharge and return to work ^[47–49]. Regional anesthetics have been used successfully in sinus surgery, thyroidectomy, parathyroidectomy, sphenopalatine artery ligation, eye surgery, facial plastic/reconstructive surgery, myringotomy/tympanostomy, and transsphenoidal hypophysectomy ^[50–55]. Specific block technique is beyond the scope of this chapter and is discussed elsewhere in this text (Chaps. 2, 12–14) and others in detail ^[50].

Peripheral Nerve Anatomy and Blocks

The trigeminal nerve (Cranial Nerve V) (Fig. 22.5) provides sensory innervation to the face and motor function to the muscles of mastication. After originating at the gasserian ganglion, it branches into the ophthalmic (V1), maxillary (V2), and mandibular (V3) nerves—each providing targets for regional anesthetics.

The ophthalmic division of the trigeminal nerve (V1) enters the orbit via the superior orbital fissure and divides into the supraorbital, supratrochlear, and nasociliary nerves. The nasociliary branch innervates the sinuses, as well as the mucous membrane of the nasal cavity. Regional anesthesia of the ophthalmic division of the trigeminal nerve can be utilized in ophthalmologic and sinus surgery; however, serious adverse events relating mostly to permanent ocular damage and local anesthetic toxicity have been reported ^[56, 57].

The maxillary nerve (V2) becomes the infraorbital nerve at the infraorbital fossa and exits the infraorbital foramen to provide sensory innervation to the palate, teeth, gums, nasal mucosa, lower eyelid, and upper lip. In combination with the nasociliary branch of the ophthalmic (V1) division, the infraorbital nerve also provides sensation to the sinuses and nasal mucosa. Infraorbital blocks have been successfully employed to decrease postoperative pain, opioid requirements, and decrease intraoperative anesthetic dosing for otolaryngologic surgery ^[52–54].

A branch of the mandibular nerve (V3), the mental nerve, emerges from the mental foramen and with input from the facial nerve provides sensory input from the skin of the chin and the mucous membrane of the lower lip. Blockade of the mandibular nerves prior to bilateral mandibular osteotomy has been shown to effectively decrease total perioperative opioid consumption ^[58]. A computed tomography (CT)guided approach to the foramen ovale to block the mandibular nerve in a patient with mandibular deformity caused by segmental mandibulectomy has been successfully performed ^[59].

Block of the inferior alveolar nerve, a branch of the mandibular nerve (V3), is routinely used for dental and oral surgical procedures. Nerve stimulation technique has been attempted to decrease the failure rate of these blocks and has been ineffective ^[60]. When used in conjunction with cervical plexus blocks, the inferior alveolar nerve block has been employed during carotid endarterectomy in which high carotid bifurcations necessitate cranial retraction ^[61].

The auricular branch of the vagus nerve (Fig. 22.6) (Cranial Nerve X) innervates the external auditory meatus and the inferior portion of the tympanic membrane. Blockade

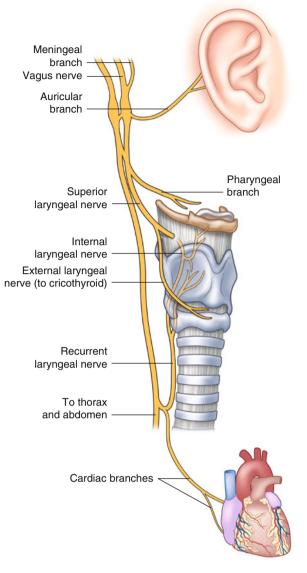


Fig. 22.6 Vagal nerve branches

of this nerve can be achieved by infiltration of anesthetic posterior to the tragus and can be useful for myringotomy and tympanostomy procedures ^[50, 51, 55].

The cervical plexus is comprised of the ventral rami of cervical nerves 1 through 4. Superficial cervical plexus can be blocked (Fig. 22.7) to provide anesthesia for otoplasty, cochlear implant, thyroidectomy, tympanomastoid surgery, and carotid endarterectomy—in which case it is as effective as a combined (superficial and deep cervical plexus blocks) cervical plexus block ^[62, 63] Superficial cervical plexus blocks may also be employed for postoperative analgesia after thyroid and parathyroid surgery ^[64].

Neuraxial Anesthesia

Cervical epidural anesthesia has been used as a primary anesthetic for thyroidectomy ^[65]. Preemptive epidural regimens did not contribute to overall improvement when compared to post-incisional continuous epidural regimens in thoracic and abdominal surgeries; however, epidural analgesia did significantly reduce postoperative pain ^[21].

Perioperative Intravenous Medications

Opiates and Opioids

Opiates are derived from alkaloids found in the opium poppy (e.g., morphine and codeine), while opioids (opiate "like") are fully synthetic substances (i.e., fentanyl, remifentanil, methadone, etc.) and semisynthetic substances (i.e., hydromorphone, hydrocodone, oxycodone, oxymorphone, etc.). Both substances act by binding to the same opioid receptors (i.e., mu, kappa, delta, etc.) predominantly in the CNS.

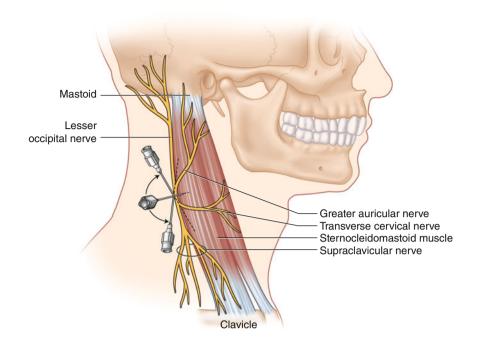
Common life-threatening and non-life-threatening side effects of opioid therapy include sedation, respiratory depression, nausea or vomiting, constipation, urinary retention, laryngospasm, chest wall rigidity, mental confusion or dizziness, mood change, nightmares or hallucinations, and sleep disorders. Non-immunologic histamine release (anaphylactoid response) via mast cell degranulation may follow morphine and meperidine administration and may produce flushing, tachycardia, hypotension, pruritus, and bronchospasm. This would be considered a relative contraindication for patients with reactive airways disease (asthma and COPD) and cardiovascular disease (coronary artery and stenotic valvular disease).

Development of opioid addiction is extremely unlikely during treatment of acute pain ^[66] and pain should be managed aggressively, since the intensity of acute postoperative pain correlates with the risk of developing a persistent chronic pain state ^[17].

Opioids and Patient Controlled Analgesia

Although intravenous opioids can be delivered by intermittent nursing bolus, use of IV PCA may lead to improved pain control, patient satisfaction, nursing satisfaction, and efficiency ^[12, 18]. PCA use without basal infusions (a continuous low-dose infusion) have been shown to cause little respiratory depression with an incidence lower than seen with

Fig. 22.7 Superficial cervical plexus blockade



intermittent IM injections. Since use of basal infusions contributes little to postoperative pain relief (excluding chronic pain population) and can increase the incidence of respiratory depression, routine use is not recommended. Consultation with a pain service should be considered in surgical patients with preexisting chronic pain syndromes treated with opioids, as a basal infusion may be warranted to replace their chronic opioid dose. Even with appropriate management these patients will consistently report higher pain scores in the perioperative period than patients without chronic pain or prior opioid exposure.

Morphine is the "gold standard" medication for use in IV PCA. Its use may be limited in patients with hemodynamic instability or pulmonary disease (histamine release), renal dysfunction (delayed respiratory depression with accumulation of morphine-6-glucuronide, an active metabolite), or intolerance of other side effects. Hydromorphone and fentanyl are good alternative for morphine-intolerant patients, those with renal insufficiency, and opioid-tolerant patients ^[67]. Meperidine is not recommended for use in pain management due to risk of CNS toxicity from its metabolite normeperidine ^[68]. Suggested IV PCA settings are outlined in Table 22.1 while opioid conversion is summarized in Table 22.2. Practice guidelines for the sustained use of analgesics in critically ill adults have been established by the Society of Critical Care Medicine ^[69].

 Table 22.1
 Commonly used initial IV patient controlled analgesia regimens for opioid naïve patients

Opioid	Demand dose	Lockout (min)
Morphine	1–2 mg	6–10
Hydromorphone	0.2–0.4 mg	6–10
Fentanyl	20-50 mcg	5-10

Ketorolac and Other IV Nonsteroidal Anti-inflammatory Drug

Ketorolac was the first nonsteroidal anti-inflammatory drug (NSAID) available for intravenous administration in the USA. Although intravenous acetaminophen just received FDA approval in the USA it was available in other parts of the world. NSAIDS are useful in reducing the amount of opioid requested by the patient and thus decrease opioid side effects ^[70–72]. A single 30 mg intravenous dose has the efficacy of 10 mg of intravenous morphine. Although ketorolac may be as safe as other NSAIDs in the treatment of postoperative pain and studies exist providing evidence of safe use of NSAIDS after otolaryngologic procedures, ENT and plastic surgery may pose an increase in postoperative bleeding risk, especially when postoperative thromboprophylaxis is utilized ^[35,73]. Gastrointestinal and renal complications are rare. Ketorolac has been reported to cause acute bronchospasm in

Table 22.2 Opioid conversion table				
Drug	IV dose equianalgesic to morphine 10 mg IV	Oral dose equianalgesic to morphine 10 mg PO	Oral to IV equianalgesic dose	
Morphine	10	10	10:3.3	
Fentanyl	0.1	-	-	
Meperidine	75	100	10:2.5	
Hydromorphone	1.5	2.5	10:2	
Methadone	2	6.7 (acute dosing)	10:1 (acute dosing)	
		1 (chronic dosing)	10:2.5 (chronic dosing)	
Codeine	120	66.7	10:6	
Oxycodone	-	6.7	-	

aspirin-intolerant patients and up to 10% of asthmatics may be sensitive to aspirin and NSAID therapy ^[73].

Ketamine

The addition of a sub-anesthetic dose (0.1–0.3 mg/kg/h) intravenous ketamine, an *N*-methyl-D-aspartic acid (NMDA) receptor antagonist, results in superior analgesia, significant morphine sparing, less sedation, decreased nausea/vomiting, and reduced need for physician intervention to manage post-operative pain. It is useful as an adjunct for patients on chronic opioids in whom pain is poorly controlled in spite of high-dose opioid therapy. Side effects of low-dose ketamine (hallucinations) are usually mild, but experienced anesthesia personnel should be involved in managing the drug ^[74–79]. Subhypnotic doses (<5 mcg/kg/min) have been reported to provide effective analgesia and sedation in a small number of patients inadequately controlled with high doses of opioids and benzodiazepines in the critical care unit ^[80, 81].

Lidocaine

Intravenous lidocaine has been shown to improve pain scores in the initial postoperative period, decrease perioperative opioid requirements, and reduce incidence of postoperative ileus. Typically, infusions are initiated at 1.5–3 mg/kg/h after an initial bolus of 1.5–2 mg/kg. Postoperative infusions can be continued, albeit at lower doses. Monitoring for neurologic and cardiovascular side effects of systemic local anesthetics is imperative throughout the duration of the infusion ^[82, 83].

Postoperative Oral Analgesics

Patients tolerating oral medications may be transitioned to oral analgesics. An opioid equipotency table (Table 22.2) should be employed to calculate the appropriate oral dose based on preceding 24 h IV PCA requirements. Patients in mild to moderate pain may be managed with NSAIDS (unless contraindicated), acetaminophen, or combination drugs (i.e., Tylenol #3—acetaminophen/codeine, Percocet—acetaminophen/oxycodone, etc.).

Tramadol, a weak non-opioid mu receptor agonist and SNRIs, and the newer tapentadol, a strong non-opioid mu receptor agonist and norepinephrine reuptake inhibitor, are effective for postoperative pain ^[84, 85].

Patients with moderate to severe pain may require more significant doses of strong opioids (i.e., morphine, hydro-morphone, oxycodone, etc.). Many medications are available as liquid suspensions if swallowing pills is painful after otolaryngologic procedures ^[86].

Chronic Pain Management

Background

Chronic pain usually refers to pain lasting more than 3 or 6 months after the initiation of pain; however, alternatively it may be defined as "pain that extends beyond the expected period of healing" ^[87]. Chronic pain is highly prevalent in the general population and has serious impact on worker disability, health-care consumption, and quality of life ^[88, 89]. Almost half of patients with chronic pain are inadequately managed and few are managed by pain medicine specialists ^[90].

The World Health Organization (WHO) developed a three-step pain treatment guideline for cancer patients known as the "Analgesic Ladder" (Fig. 22.8). Although the "ladder" provides the majority of cancer pain patients with adequate relief, it has not been well validated for non-cancer pain patients. The WHO ladder concept recommends the use of non-opioid medications (NSAIDS/acetaminophen) with or without the use of adjuvants (anticonvulsants/ antidepressants) first. If inadequate the addition of weak opiates (e.g., codeine, hydrocodone) for mild to moderate pain with or without non-opioids or adjuvants should follow.

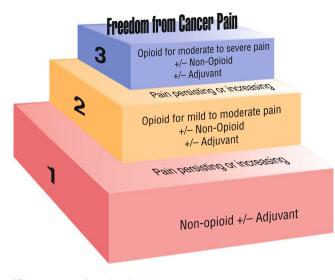


Fig. 22.8 WHO analgesic ladder

For moderate to severe pain strong opiate/opioids (i.e., morphine, hydromorphone, oxycodone, methadone, etc.) are employed with or without non-opioids or adjuvants^[91]. When pain cannot be controlled with systemic analgesics invasive techniques such as intrathecal pumps and neural blockade techniques should be considered ^[92].

Agents and Analgesics

Opiates and Opioids

The role of opioid therapy for treatment of acute (postoperative) and malignant pain is well established, but treatment of chronic nonmalignant pain with opioids remains controversial. These agents can be administered subcutaneously, parentally, intravenously and into the central nervous system. A systematic review and meta-analysis found that the use of opioids in patients with chronic back pain, compared to placebo and non-opioid analgesics, did not result in significant improvement in pain scores ^[93, 94]. Opioids may be effective for intermediate term management of neuropathic pain, although pain control is comparable to non-narcotic gabapentin and adverse events were common ^[95]. Some evidence does exist, however, for longer term use of opioids to treat nonmalignant pain ^[96].

Rates of addiction appear to be higher in patients prescribed opioids for nonmalignant pain than for cancer pain and aberrant drug taking behaviors may occur in up to 24% of patients ^[94, 97]. Patients should be closely monitored, especially immediately after initiation of therapy, as the risk for an adverse event is greatest shortly after starting medications ^[98]. Patients receiving higher doses are at increased risk

for overdose. In a review of malpractice claims associated with medication management for chronic pain, death was the most common outcome ^[99]. Doses should be increased in a goal-directed manner, with increases dependent on clear functional improvement ^[100].

In addition to direct physiologic side effects (i.e., nausea, vomiting, constipation, respiratory depression, etc.) and the aforementioned risks of addiction and overdose, chronic opioid therapy may also cause hypogonadism, decreased immune function, disruption of sleep patterns, and, paradoxically, opioid-induced hyperalgesia [101-104]. Overall, the decision to begin long-term opioid therapy must be weighed carefully and guidelines established for chronic opioid therapy should be followed closely ^[105]. Equianalgesic tables may be utilized for opioid conversion; however, due to tremendous inter-patient variability and inconsistency of published conversions, clinical judgment must be used and individual patient characteristics considered when applying any table. In order to account for incomplete cross tolerance, a 25-50% dose reduction is commonly employed when rotating from one opioid to another [106]. Table 22.2 lists generally accepted opioid conversions.

Tramadol

Tramadol (Ultram) is thought to provide analgesia through weak mu receptor agonism and inhibition of serotonin and norepinephrine uptake. It is effective in the treatment of acute postsurgical pain, neuropathic pain, and pain associated with osteoarthritis. Effectiveness of sustained release preparations is equivalent to those providing immediate release [85, 107-109]. Tapentadol (Nucynta), a close chemical relative of tramadol, is a centrally acting mu-opioid receptor agonist with selective norepinephrine reuptake inhibitor activity. It has been shown to be as effective as oxycodone in the management of acute and chronic pain with a decreased rate of gastrointestinal side effects. At this time, however, tapentadol is indicated only for the treatment of acute pain [110, 111]. A small risk of seizure and serotonin syndrome, a potentially life-threatening syndrome associated with excessive serotonin, exists with both of these medications, particularly in patients taking antidepressants, neuroleptics, or other drugs that decrease seizure threshold [111, 112]. Symptoms such as agitation, tachycardia, hyperpyrexia, labile blood pressure, hyperreflexia, hallucinations, and loss of coordination are associated with the syndrome.

Anticonvulsant Agents

Gabapentin has strong evidence supporting its widespread use and is the most commonly used drug for neuropathic pain. It has been used effectively in management of diabetic peripheral neuropathy and post-herpetic neuralgia ^[113]. Although the mechanism of action is not completely understood, it is thought to antagonize voltage-gated calcium channels and centrally modulate GABA activity ^[114]. The initial dose is usually 300 mg at night and is increased with a maximum daily dose of 3,600 mg until analgesic efficacy or intolerable side effects are reached. Fatigue and sedation are most common side effects and can be minimized by slow titration of medication dose ^[115].

Pregabalin has a mechanism of action similar to gabapentin and is also effective in the treatment of neuropathic pain ^[116]. Combined with tricyclic antidepressants [dual reuptake inhibitors of serotonin and norepinephrine (SNRIs)] and topical lidocaine, these agents make up the first-line treatment of neuropathic pain ^[117].

Carbamazepine, a sodium and calcium channel blocker that modulates descending inhibition of pain pathways, is the drug of choice for the treatment of trigeminal neuralgia. Oxcarbazepine provides a good alternative treatment ^[118]. Interestingly, Lamotrigine, another sodium and calcium channel blocker has not been found to be an effective treatment of neuropathic pain ^[119]. Conflicting data exists on the use of topiramate for neuropathic pain, but it has shown to be useful in treatment of chronic back pain and as a prophylactic therapy for migraine headaches. Due to need for serum monitoring and the existence of safer and more effective therapies, phenytoin is rarely used for the treatment of pain ^[120–122].

Antidepressants

Antidepressants have been shown to be effective adjuvants for the treatment of chronic pain while improving mood, and pain initiated sleep disruption. Tricyclic antidepressants (TCA), along with serotonin norepinephrine reuptake inhibitors (SNRI) and gabapentinoids (e.g., gabapentin and pregabalin), are first-line agents used for various neuropathic pain syndromes ^[117].

TCAs (i.e., imipramine, desipramine, nortriptyline, etc.) are thought to exert their analgesic action by serotonin and norepinephrine reuptake inhibition and by independently relieving depressive symptoms and improving sleep in patients with chronic pain. Adverse side effects associated with TCAs include orthostatic hypotension, dizziness, urinary retention, dry mouth, blurry vision, acute angle closure glaucoma, and cardiac conduction disturbances ^[123].

The SNRIs, duloxetine and venlafaxine, have been successfully used in the treatment of neuropathic pain, as well as musculoskeletal pain ^[117, 124]. These medications appear to have similar efficacy to TCAs ^[125].

Benzodiazepines

Routine use of benzodiazepines in chronic pain patients should be avoided due to a paucity of evidence for their use and a higher incidence of overdose in this population ^[100, 102]. Diazepam may be useful in treating painful muscle spasm; however, other antispasticity agents (i.e., baclofen, tizanidine, etc.) or antispasmodic agents (i.e., cyclobenzaprine, etc.) are readily available and may be just as effective for this indication ^[126].

Nonselective Nonsteroidal Anti-inflammatory Drugs (nsNSAIDS)

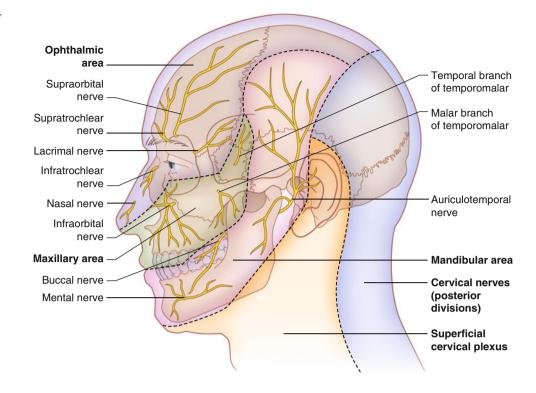
nsNSAIDS (ibuprofen, naproxen, etc.) inhibit both isoforms of cyclooxygenase (COX-1 and COX-2) thereby impairing transformation of arachidonic acid to prostaglandins, prostacyclin, and thromboxanes. Selective COX-2 (coxibs) inhibiting drugs were created to minimize side effects of nsNAIDSs; however, all but one (celecoxib) have been removed from the market due to increased rates of adverse cardiovascular events associated with their use.

A meta-analysis of all trials comparing NSAIDs found a dose-related increase in the risk of adverse cardiovascular events with both coxibs and high doses of nsNSAIDS^[127]. The rate for hospitalization for heart failure for older patients without history of heart failure and mortality in patients with preexisting heart failure were significantly greater with coxib and high-dose nsNSAID use [128-130]. NSAIDS can also cause gastrointestinal (GI) ulceration, hypertension, edema, and renal failure (especially when taken with ACE inhibitors and diuretics) ^[131-134]. Concomitant use with other antiplatelet therapies is not recommended and GI prophylaxis with a proton pump inhibitor should be initiated in patients at risk for GI bleeding ^[135]. Overall, careful patient selection, utilization of the minimal effective doses, and limiting treatment duration are key in the successful treatment of pain with NSAIDS.

Chronic Pain Syndromes of the Head and Neck Patient

Craniofacial Pain

Multiple mechanisms, such as inflammation, nociceptor activation, and tissue injury are responsible for the induction of central and peripheral craniofacial pain. Injury to the central nervous system leads to central pain, while damage or loss of primary afferent fibers results in peripheral



neuropathic pain ^[136]. Therapy reflects the multiple etiologies for chronic pain syndromes and will include the use of medications, nerve stimulation, and regional nerve blockade. Most chronic pain patients will also need intervention to counter the physical and psychiatric impact of chronic pain.

Five general causes of central facial pain are described in The International Classification of Headache Disorders ^[137].

Anesthesia dolorosa presents as painful anesthesia or hypesthesia in the distribution of the occipital or trigeminal nerves (Fig. 22.9). The most frequent cause is iatrogenic, occurring after rhizotomy or thermocoagulation performed to treat trigeminal neuralgia. Either TCAs (amitriptyline) or gabapentin can be helpful in its treatment ^[138, 139].

Central post-stroke pain is a unilateral dysesthesia and altered sensation that is not attributable to trigeminal nerve pathology. Rather, the etiology is a lesion of the trigeminothalamic pathway, thalamus, or thalamocortical projection. Tricyclic antidepressants are first-line agents, although gabapentin does show therapeutic potential ^[140].

Multiple sclerosis may result in severe unilateral or bilateral facial pain that is attributed to a demyelinating lesion of the central connections of the trigeminal nerve. Carbamazepine is the drug of choice; however, evidence for its use is weak ^[141].

Persistent idiopathic facial pain is not attributed to another disorder and is a diagnosis of exclusion. Either TCAs or topiramate may be considered for management ^[142, 143].

Burning mouth syndrome is an oral burning sensation without an identifiable pathology. All potential medical or dental causes of mouth pain must be ruled out before establishing this diagnosis. TCAs, clonazepam, gabapentin, and cognitive behavioral therapy may be employed in its treatment [144].

In addition to pharmacologic and behavioral therapy, transcutaneous electric nerve stimulation and gasserian ganglion stimulation have been used with some success for the treatment of central craniofacial pain. Deep brain stimulation and other surgical therapies are used as last resorts in patients with severe disability and failure of other treatments ^[145, 146].

Peripheral Causes of Facial Pain (Neuralgias)

Neuralgia is pain in the distribution of a particular nerve. This pain is often maximal at onset and described as sharp, lancinating, burning, or electric.

Glossopharyngeal neuralgia is characterized by paroxysmal, severe, stabbing pain affecting the tonsillar fossa, ear, tongue, or the angle of the jaw. Triggers include chewing, touch, swallowing, coughing, speaking, or yawning. Secondary causes include demyelinating lesions, cerebellopontine angle tumor, peritonsillar abscess, carotid aneurysm, and Eagle syndrome (lateral compression of CN IX against an ossified stylohyoid ligament) ^[137, 147]. Vascular compression of CN IX and X can occur at the nerve root entry by the vertebral artery or posterior inferior cerebellar artery. Magnetic Resonance Imaging is indicated to rule out a mass lesion, vascular pathology, or other pathologies (i.e., multiple sclerosis etc.). Plain skull films may reveal an ossified stylohyoid ligament (consistent with *Eagle syndrome*) ^[148–150].

The pain of trigeminal neuralgia (TN) tends to occur in paroxysms and is maximal at or near onset. The pain has been described as "electric shock-like" or "stabbing." Increasing evidence suggests that many cases are caused by vascular compression of the trigeminal nerve proximally to its exit from the brain stem ^[151, 152]. Up to 5% of patients with multiple sclerosis will develop TN. Available evidence shows that carbamazepine is the drug of choice^[153,154]. Oxcarbazepine can be used if carbamazepine is not well tolerated [155]. Gabapentin has not been successful in the management of TN [111]. Blockade of the mandibular nerve with alcohol has been shown to provide long-lasting pain relief for the treatment of trigeminal neuralgia^[156]. A sphenopalatine ganglion (SPG) block is indicated for TN, as well as for treatment of headache (radiofrequency ablation of SPG), and other types of facial pain [157-159]. Percutaneous, controlled radiofrequency trigeminal rhizotomy (RF-TR) is another minimally invasive, low-risk technique with a high rate of efficacy, which may be repeated if pain recurs [160]. Both microvascular decompression and gamma knife radiosurgery may be employed in the treatment of TN, although the former is more likely to result in complete pain relief [161].

Cancer Pain

At least 50% of patients with head and neck cancer experience significant pain with approximately 10% experiencing severe pain. Pain in patients who have cancer may be due to direct effects of the tumor (i.e., invasion of bone, compression of nerves, etc.), by complications of therapy (i.e., radiation fibrosis, chemotherapy-induced neuropathy, etc.), or it can be unrelated to the disease or its treatment.

Due to the association with recurrence and survival, the complaint of pain within the first year of treatment for head and neck cancer is an important symptom that should be appropriately monitored, evaluated, and managed during routine follow-up ^[162].

Head and neck cancer patients have considerable psychiatric morbidity, which must be addressed in order to effectively manage malignancy related pain. A third of the patients have a possible or probable mood disorder during the first year of diagnosis ^[163]. Anxiety levels are highest during diagnosis and decreased during the course of treatment, while depressive symptoms develop during treatment and either increase or remain unchanged over time ^[163, 164]. Use of prophylactic antidepressants has been advocated in this population ^[165]. This coupled with an aggressive application of the WHO Ladder is the mainstay of head and neck cancer pain management; however, a variety of procedures may be employed for patients who continue to experience pain

Table 22.3 Head and neck nerve blocks

HeadFaceTrigeminal nerve blockContents of orbit, sphenoid sinus, eyelids, anterior 2/3 of scalpOphthalmic nerve blockForeheadSupraorbital nerve blockUpper jaw, maxillary antrum, distribution of infraorbital nerveMaxillary nerve blockLower eyelid, upper lip, temple, lateral aspect of noseInfraorbital nerve block, Mental nerve blockLower jaw, teeth, anterior tongue, floor mouthMandibular nerve block, Mental nerve blockNose, palateSphenopalatine nerve blockPosterior third of tongue, soft palate, parotid glandGlossopharyngeal nerve Block Third occipital nerve block, Third occipital nerve block, Cheadache)Shoulder and neckCervical plexus block, cervical paravertebral block, cervical facet (medial branch) block, cervical epidural injection	Area of pain	Nerve block
Contents of orbit, sphenoid sinus, eyelids, anterior 2/3 of scalpOphthalmic nerve blockForeheadSupraorbital nerve blockUpper jaw, maxillary antrum, distribution of infraorbital nerveMaxillary nerve blockLower eyelid, upper lip, temple, lateral aspect of noseInfraorbital nerve blockLower jaw, teeth, anterior tongue, floor mouthMandibular nerve block, Mental nerve blockNose, palateSphenopalatine nerve blockPosterior third of tongue, soft palate, parotid glandGreater occipital nerve block, Third occipital nerve block, Third occipital nerve block, crvical paravertebral block, cervical paravertebral block, cervical facet (medial branch) block,	Head	
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Upper jaw, maxillary antrum, distribution of infraorbital nerveMaxillary nerve blockLower eyelid, upper lip, temple, lateral aspect of noseInfraorbital nerve blockLower jaw, teeth, anterior tongue, floor mouthMandibular nerve block, Mental nerve blockNose, palateSphenopalatine nerve blockPosterior third of tongue, soft palate, parotid glandGlossopharyngeal nerve BlockNeckScalp, back of neckScalp, back of neckGreater occipital nerve block, theadache)Shoulder and neckCervical plexus block, cervical paravertebral block, cervical facet (medial branch) block,	· •	Ophthalmic nerve block
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floor mouthMental nerve blockNose, palateSphenopalatine nerve blockPosterior third of tongue, soft palate, parotid glandGlossopharyngeal nerve BlockNeckScalp, back of neckGreater occipital nerve block, Third occipital nerve block (headache)Shoulder and neckCervical plexus block, cervical paravertebral block, cervical facet (medial branch) block,		Infraorbital nerve block
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palate, parotid gland Image: Parotic gland Neck Scalp, back of neck Scalp, back of neck Greater occipital nerve block, Third occipital nerve block (headache) Shoulder and neck Cervical plexus block, cervical paravertebral block, cervical facet (medial branch) block,	Nose, palate	Sphenopalatine nerve block
Scalp, back of neckGreater occipital nerve block, Third occipital nerve block (headache)Shoulder and neckCervical plexus block, cervical paravertebral block, cervical facet (medial branch) block,		Glossopharyngeal nerve Block
Third occipital nerve block (headache) Shoulder and neck Cervical plexus block, cervical paravertebral block, cervical facet (medial branch) block,	Neck	
paravertebral block, cervical facet (medial branch) block,	Scalp, back of neck	Third occipital nerve block
eer rieur epitaliur injeenen	Shoulder and neck	paravertebral block, cervical
Larynx, trachea Laryngeal nerve block, deep cervical plexus block	Larynx, trachea	

Adapted from Otolaryngol Clin North Am, 42(1), Mehio AK, Shah SK, Alleviating head and neck pain, pp 143–59, Copyright 2009, with permission from Elsevier

despite conservative treatment or who have intolerable treatment side-effects.

Blockade of the mandibular nerve (with and without an indwelling catheter), sphenopalatine (pterygopalatine) ganglion (and maxillary nerve), trigeminal ganglion, glossopharyngeal nerve, and the occipital nerve (and neurolysis of these structures) may be performed for the treatment of intractable cancer pain ^[166–171]. A combination of a mandibular nerve and sphenopalatine ganglion blocks, as well as intrathecal infusion of local anesthetic or opioid has been reported ^[172–175]. Table 22.3 couples areas of pain with possible interventions.

Other Pain Syndromes of the Head and Neck

Headache

Headache syndromes include migraine, tension headache, trigeminal autonomic cephalgias [cluster headache, paroxysmal hemicrania, short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing

	Migraine headache	Tension headache	Cluster headache
Location	Unilateral or bilateral	Bilateral	Always unilateral, around the eye or temple
Characteristics	Gradual onset; crescendo pattern; pulsating; aggravated by routine physical activity	Pressure or tightness which waxes and wanes	Pain begins quickly, reaches a crescendo within minutes; pain is deep, continuous, excruciating, and explosive in quality
Duration	4 h to 3 days	Variable	30 min to 3 h
Associated symptoms	Nausea, vomiting, photophobia, phonophobia; aura (usually visual, but can involve other senses or cause speech or motor deficits)	None	Tearing, conjunctival injection; stuffy nose; rhinorrhea; pallor; sweating; Horner's syndrome; sensitivity to alcohol

 Table 22.4
 Common headache syndromes

(SUNCT), short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms (SUNA)], and chronic daily headache (including medication overuse head-ache). Other causes of headache include brain tumors, cerebral vein thrombosis, giant cell arteritis, intracranial hypotension (either spontaneous or after a dural puncture), posttraumatic headache, ruptured intracranial aneurysm, and sinus headache ^[137]. While a thorough discussion of the topic of headache is beyond the scope of this chapter, sinus headache ache and contact point headache are of special interest and will be discussed. Features of common headache syndromes are summarized in Table 22.4.

Sinusitis appears to be an uncommon cause of chronic headaches and many patients initially diagnosed with sinus headache actually suffer from migraine ^[176, 177]. True acute sinus headache occurs in conjunction with acute sinusitis, fever, and purulent discharge. If these features are not present, migraine accompanied with sinus pain should be considered.

Sinus pain or headache usually presents as a pressure-like or dull bilateral periorbital sensation that is associated with nasal congestion. In patients with middle or inferior turbinate hypertrophy, deviated septum, or unilateral sinus disease, the pain may be unilateral. It is unusual for sinus headache to present with nausea, photophobia, or hearing sensitivity. Severity or location of mucosal disease on imaging studies does not correlate with sinus related pain [178]. In 2005 multidisciplinary guidelines were published to assist in differentiation of migraine and sinus headache. These guidelines suggest that: a stable pattern of recurrent headaches that interfere with daily function is most likely migraine, recurrent self-limited headaches associated with rhinogenic symptoms are most likely sinus. Prominent rhinogenic symptoms with headache as one of several symptoms should be evaluated carefully for otolaryngologic conditions (nasal endoscopy preferred or alternatively with magnetic resonance imaging (MRI) or CT. Headache associated with fever and purulent nasal discharge is likely rhinogenic in origin. Referral to a headache specialist should be considered for

new-onset headache, frequent (more than once a week) headache, headache associated with neurologic symptoms or signs, or headache that does not respond adequately to conventional therapy. Patients with migraine with no evidence of infection should be given a trial of migraine-specific medication and seen for a follow-up evaluation, and patients with noninfectious rhinogenic symptoms who have headache as a minor symptom should be given nasal glucocorticoids and/or selective nasal antihistamines ^[179].

Contact point headaches are likely due to intranasal contact between mucosal surfaces that results in referred pain in the distribution of the trigeminal nerve. In subjects with primary headaches, contact points may be associated with therapeutic failure. For selected patients with refractory headaches, demonstrable contact points, and positive response after topical anesthesia, surgical approach toward the triggering factor may be useful ^[180].

Neck Pain

Cervical spine disorders may result in neck pain, extremity pain, and neurologic dysfunction. Axial neck pain may be due to cervical strain, spondylosis, intervertebral disc disease (may be most common), cervical facet syndrome, whiplash injury, myofascial pain, and diffuse skeletal hyperostosis. However, these etiologies should be considered only after rheumatologic (i.e., rheumatoid arthritis, etc.), neurologic (i.e., Chiari malformation, etc.), visceral (i.e., esophageal or biliary disease, apical lung tumor, etc.), oncologic (bony metastasis), infectious (i.e., pharyngeal abscess, meningitis, Lyme disease, etc.) vascular (vertebral or carotid artery dissection), cardiac (angina or myocardial infarction) etiologies of neck pain have been ruled out. Examination should document range of motion, provocative maneuvers, and neurologic examination for radicular and upper motor signs. Plain films should be ordered for neck trauma, new symptoms in patients older than 50, or constitutional symptoms. CT or MRI should be ordered for patients with

objective neurologic impairment, dramatic bony tenderness with impaired mobility, or persistent symptoms despite 6 weeks of conservative therapy. Electromyographic (EMG) testing can distinguish peripheral nerve from radicular pathology and should be considered when pain is greater in the extremities than in the neck ^[181].

Most mild-to-moderate axial neck pain will improve within 2-3 weeks. Initial treatments may include acetaminophen or NSAIDS, posture modification, and home exercise. Mild opioid analgesics (e.g., codeine, hydrocodone) or tramadol may be used for short-term treatment of severe pain. Low-dose tricyclic antidepressants may be prescribed at nighttime for pain that interferes with sleep. Severe muscle spasm can be treated with muscle relaxants (i.e., tizanidine, etc.). Cervical collars may be used to alleviate severe pain, but should not be used for any longer than 3 h at a time for a period of less than 3 weeks, as greater use may result in muscle atrophy. Physical therapy may be employed for pain that does not resolve with home exercise and mild analgesics. Spinal traction is not recommended for the treatment of neck pain. Spinal manipulation is not recommended for the treatment of neck pain, as it does not appear to be effective and can result in rare, but devastating complications. Trigger point injections, TENS, and cervical medial branch blocks (and percutaneous radiofrequency neurotomy) may be useful in patients who do not respond to more conservative measures. Surgery is generally not recommended for patients with neck pain who do not exhibit myelopathic or radiculopathic signs and/or symptoms [182].

Cervical radiculopathy is neck and extremity pain resulting from nerve root compression. Nonoperative management is similar to that of axial neck pain. In addition, epidural steroid injections and spinal cord stimulation may provide pain relief in patients with radiculopathic neck pain ^[183].

A thorough workup with the goal of identifying the underlying mechanisms of head and neck pain is an essential element in its treatment. A multidisciplinary approach utilizing medications, interventions, and psychiatric support should be employed in the management of these, often challenging to treat, pain syndromes.

Conclusion

Acute and chronic pain remains significant sources of postoperative morbidity. Identification of patients most at risk and aggressive multimodal, multidisciplinary strategies are vital to improving patient outcomes. Ultimately, close collaboration between otolaryngologists and pain specialists is the critical component in establishing comprehensive, effective care.

Clinical Insights

For the Otolaryngologist

- 1. Consider administering acetaminophen, COX-2 selective inhibitors (celecoxib, meloxicam) gabapentinoids, or clonidine 1–2 h prior to surgery.
- 2. Prior to incision infiltrate wound with local anesthetic±clonidine
- 3. Employ multimodal analgesia postoperatively— NSAIDS, acetaminophen, opioids. Using tramadol (or tapentadol) may be associated with a decreased incidence of nausea and vomiting.
- 4. Inpatients with moderate-severe levels of pain should receive an IV PCA (not nursing administered boluses).
- 5. Patients not responding to basic analgesic regimens or those with a history of chronic pain (especially those on chronic opioid therapy) should be seen by a specialist pain medicine service. A preoperative evaluation of patients with chronic pain by a pain specialist should be considered.
- 6. Preparation of children and caregivers for the surgical experience may decrease postoperative pain.

Clinical Pearls

For the Anesthesiologist

- 1. Administer dexamethasone IV prior to incision.
- 2. Administer acetaminophen perioperatively.
- 3. Administer ketorolac, if agreed upon by surgeon, at the end of the procedure.
- 4. Utilize regional anesthetic blocks to decrease anesthetic requirements, postoperative pain, and opioid requirements (as well as their side effects).
- 5. Consider intraoperative IV lidocaine infusion, ketamine infusion, and/or methadone for opioid tolerant patients, those with significant preoperative pain, and extensive surgeries.
- 6. Consider postoperative low dose ketamine in patients with poorly controlled pain (especially patients with chronic pain on opioid therapy)
- 7. Preparation of children and caregivers for the surgical experience may decrease postoperative pain.

References

- Freburger JK, Holmes GM, Agans RP, et al. The rising prevalence of chronic low back pain. Arch Intern Med. 2009;169(3):251–8.
- Salmon L. Pain prevalence in a French teaching hospital. J Pain Symptom Manage. 2002;24(6):586.
- Sommer M, et al. Prevalence and predictors of postoperative pain after ear, nose, and throat surgery. Arch Otolaryngol Head Neck Surg. 2009;135(2):124–30.
- Merskey H, Bogduk N. Classification of chronic pain: descriptions of chronic pain syndromes and definitions of pain terms. 2nd ed. Seattle: IASP Press; 1994.
- Harden RN. Chronic neuropathic pain. Mechanisms, diagnosis, and treatment. Neurologist. 2005;11:111.
- Jamison RN, Gracely RH, Raymond SA, et al. Comparative study of electronic vs. paper VAS ratings: a randomized, crossover trial using healthy volunteers. Pain. 2002;99:341.
- Cork CR, et al. A comparison of the verbal rating scale and the visual analog scale for pain assessment. Internet J Anesthesiol. 2004;8(1).
- Herr K, Spratt KF, Garand L, et al. Evaluation of the Iowa pain thermometer and other selected pain intensity scales in younger and older adult cohorts using controlled clinical pain: a preliminary study. Pain Med. 2007;8:585.
- Voepel-Lewis T, et al. A comparison of the clinical utility of pain assessment tools for children with cognitive impairment. Anesth Analg. 2008;106:72–8.
- Kelly DJ, Ahmad M, Brull SJ. Preemptive analgesia I: physiological pathways and pharmacological modalities. Can J Anaesth. 2001;48:1000.
- Melzack R. Evolution of the neuromatrix theory of pain. The Prithvi Raj lecture: presented at the Third World Congress of World Institute of Pain. Pain Pract. 2005;5(2):85–94.
- Apfelbaum JL, Chen C, et al. Postoperative pain experience: results from a national survey suggest postoperative pain continues to be undermanaged. Anesth Analg. 2003;97:534–40.
- de Beer Jde V, Winemaker MJ, Donnely GA, et al. Efficacy and safety of controlled-released oxycodone and standard therapies for postoperative pain after knee or hip replacement. Can J Surg. 2005;48:277.
- Recard A, Duchene D, White PF, et al. Efficacy and safety of fasttrack recovery strategy for patients undergoing laprascopic nephrectomy. J Endourol. 2005;19:1165.
- Watcha MF, Issioui T, Klein KW, White PF. Costs and effectiveness of rofecoxib, celecoxib, and acetaminophen for preventing pain after ambulatory otolaryngologic surgery. Anesth Analg. 2003;96:987.
- Polomano RC, Rathmel JP, Krenzischek DA, et al. Emerging trends and new approaches to acute pain management. J Perianesth Nurs. 2008;23:43.
- Kehlet H, Jensen TS, Woolf CJ. Persistent postsurgical pain: risk factors and prevention. Lancet. 2006;367(9522):1618–25.
- Hudcova J, McNicol E, Quah CS, et al. Patient controlled opioid analgesia versus conventional opioid analgesia for controlling postoperative pain. Cochrane Database Syst Rev. 2006.
- Roth W, Kling J, Gockel I, et al. Dissatisfaction with postoperative pain management: a prospective analysis of 1071 patients. Acute Pain. 2005;7:75.
- Kehlet H, Dahl JB. The value of "multimodal" or "balanced analgesia" in postoperative pain treatment. Anesth Analg. 1993;77:1048.
- Moiniche S, Kehlet H, Dahl JB. A qualitative and quantitative systematic review of preemptive analgesia for postoperative pain relief: the role of timing of analgesia. Anesthesiology. 2002;96:725.
- Dahl J, Kehlet, H. Preventive Analgesia. Current Opinion in Anaesthesiology 2011;24(3): p 331–8.

- Naja MZ, El-Rajab M, Kabalan W, et al. Pre-incisional infiltration for pediatric tonsillectomy: a randomized double-blind clinical trial. Int J Pediatr Otorhinolaryngol. 2005;69:1333.
- Ong CK, Lirk P, et al. The efficacy of preemptive analgesia for acute postoperative pain management: a meta-analysis. Anesth Analg. 2005;100:757.
- 25. Updike GM, Manolitsas TP, Cohn DE, et al. Pre-emptive analgesia in gynecological surgical procedures: preoperative wound infiltration with ropivacaine in patients who undergo laparotomy through a midline vertical incision. Am J Obstet Gynecol. 2003;188:901.
- Leung CC, Chang YM, Ngai SW, et al. Effect of pre-incision skin infiltration on post-hysterectomy pain – a double-blind randomized controlled trial. Anaesth Intensive Care. 2000;28:510.
- Grainger J, Saravanappa N. Local anaesthetic for post-tonsillectomy pain: a systematic review and meta-analysis. Clin Otolaryngol. 2008;33(5):411–9.
- Giannoni C, et al. Ropivacaine with or without clonidine improves pediatric tonsillectomy pain. Arch Otolaryngol Head Neck Surg. 2001;127:1265–70.
- Akkurtb CO, et al. Ropivacaine compared to bupivacaine for posttonsillectomy pain relief in children: a randomized controlled study. Int J Pediatr Otorhinolaryngol. 2006;70(7):1169–73.
- Uruga B, et al. Effects of intramuscular and peritonisllar injection of tramadol before tonsillectomy: a double blind, radomized, placebo-controlled clinical trial. Int J Otorhinolaryngol. 2008;72(2): 241–8.
- Onal A, et al. Preliminary findings for preemptive analgesia with inhaled morphine: efficacy in septoplasty and septorhinoplasty cases. Otolaryngol Head Neck Surg. 2006;135(1):85–9.
- Afman CE, et al. Steroids for post-tonsillectomy pain reduction: meta-analysis of randomized controlled trials. Otolaryngol Head Neck Surg. 2006;134(2):181–6.
- Battacharya D, et al. Single dose IV dexamethasone with preemptive transdermal diclofenac patch reduces opioid requirement and postoperative morbidity following tonsillectomy. J Anaesth Clin Pharmacol. 2009;25(1):29–32.
- 34. Ibrahim AS. Ketamine does not reduce postoperative morphine consumption after tonsillectomy in children. Clin J Pain. 2008;24(5):395–8.
- 35. Jeyakumar A, Brickman TM, Williamson ME, et al. Nonsteroidal anti-inflammatory drugs and postoperative bleeding following adenotonsillectomy in pediatric patients. Arch Otolaryngol Head Neck Surg. 2008;134:24.
- 36. Lee H, et al. The preemptive analgesic effect of ketorolac and propacetamol for adenotonsillectomy in pediatric patients. Korean J Anesthesiol. 2009;57(3):308–13.
- Norman PH, Daley MD, Lindsey RW. Preemptive analgesic effects of ketorolac in ankle fracture surgery. Anesthesiology. 2001;94:599–603.
- Ong KS, Seymour RA, Chen FG, Ho VC. Preoperative ketorolac has a preemptive effect for postoperative third molar surgical pain. Int J Oral Maxillofac Surg. 2004;33:771–6.
- Romej M, et al. Effect of preemptive acetaminophen on postoperative pain scores and oral fluid intake in pediatric tonsillectomy patients. AANA J. 1996;64(6):535–40.
- Cardwell M, Siviter G, Smith A. Non-steroidal anti-inflammatory drugs and perioperative bleeding in paediatric tonsillectomy. Cochrane Database Syst Rev. 2005;CD003591.
- Hurley RW, Cohen SP, Williams KA, et al. The analgesic effects of perioperative gabapentin on postoperative pain: a meta-analysis. Reg Anesth Pain Med. 2006;31:237.
- 42. Tiippana EM, Hamunen K, Vesa KK, Kalso E. Do surgical patients benefit from perioperative gabapentin/pregabalin? A systematic review of efficacy and safety. Anesth Analg. 2007;104(6): 1545–56.

- 43. Remy C, Marret E, Bonnet F. State of the art of paracetamol in acute pain therapy. Curr Opin Anaesthesiol. 2006;19(5):562–5.
- 44. Hidalgo MP, Auzani JA, Rumpel LC, et al. The clinical effect of small oral clonidine doses on perioperative outcomes in patients undergoing abdominal hysterectomy. Anesth Analg. 2005; 100:795.
- 45. White PF. The changing role of non-opioid analgesic techniques in the management of postoperative pain. Anesth Analg. 2005; 101:S5.
- 46. Kain ZN, Mayes LC, Caldwell-Andrews AA, et al. Preoperative anxiety, postoperative pain, and behavioral recovery in young children undergoing surgery. Pediatrics. 2006;118:651.
- Prasad KC, Shanmugam VU. Major neck surgeries under regional anesthesia. Am J Otolaryngol. 1998;19(3):163–9.
- McCartney CJ, Brull R, Chan VW, et al. Early but no long-term benefit of regional compared with general anesthesia for ambulatory hand surgery. Anesthesiology. 2004;101:461.
- 49. Anderson MB, Davidov T, et al. Postoperative recovery advantages in patients undergoing thyroid and parathyroid surgery under regional anesthesia. Semin Cardiothorac Vasc Anesth. 2010; 14(1):49–50.
- Rathmell JP, Pollack GJ. Nerve blocks of the head and neck and airway blocks. In: Hadzic A, editor. Textbook of regional anesthesia and acute pain management. Columbus: McGraw-Hill; 2006. p. 319.
- 51. Voronov P, Cote CJ, et al. Postoperative pain relief in infants undergoing myringotomy and tube placement: comparison of a novel regional anesthetic block to intranasal fentanyl – a pilot analysis. Paediatr Anaesth. 2008;18(12):1196–201.
- Mariano ER, Watson D. Bilateral infraorbital nerve blocks decrease postoperative pain but do not reduce time to discharge following outpatient nasal surgery. J Can Anesth. 2009;56:584–9.
- 53. Higashizawa T, et al. Effect of infraorbital nerve block under general anesthesia on consumption of isoflurane and postoperative pain in endoscopic endonasal maxillary sinus surgery. J Anesth. 2001;15:136–8.
- 54. Rajamani A, et al. A comparison of bilateral infraorbital nerve block with intravenous fentanyl for analgesia following cleft lip repair in children. Pediatr Anesth. 2006;17(2):133–9.
- 55. Belvis D, Voronov P, Suresh S. Head and neck blocks in children. Tech Reg Anesth Pain Manag. 2007;11(4):208–14.
- Kumar C, Dowd T. Ophthalmic regional anaesthesia. Curr Opin Anaesthesiol. 2008;21(5):632–7.
- 57. Lee LA, Posner KL, Cheney FW, et al. Complications associated with eye blocks and peripheral nerve blocks: an American society of anesthesiologists closed claims analysis. Reg Anesth Pain Med. 2008;33(5):416–22.
- Plantevin F, Pascal J, Morel J, et al. Effect of mandibular nerve block on postoperative analgesia in patients undergoing oropharyngeal carcinoma surgery under general anaesthesia. Br J Anaesth. 2007;99:708.
- 59. Kodama Y, et al. Placement of mandibular nerve block using computed tomography to locate the foramen ovale in a patient with severe dislocation after segmental mandibulectomy. Br J Oral Maxillofac Surg. 2009;47(5):407–8.
- 60. Simon F, Reader A, et al. A prospective, randomized single-blind study of the anesthetic efficacy of the inferior alveolar nerve block administered with a peripheral nerve stimulator. J Endod. 2010;36(3):429–33.
- Fassiadis N, Zayed H, et al. Carotid endarterectomy under local anaesthetic supplemented by an inferior alveolar nerve block. Ann R Coll Surg Engl. 2007;89(1):75.
- 62. Pandit JJ, Bree S, et al. A comparison of superficial versus combined (superficial and deep) cervical plexus block for carotid endarterectomy: a prospective, randomized study. Anesth Analg. 2000;91(4):781–6.
- 63. Voronov P, Suresh S. Head and neck blocks in children. Curr Opin Anaesthesiol. 2008;21(3):317–22.

- 64. Shih ML, et al. Bilateral superficial cervical plexus block combined with general anesthesia administered in thyroid operations. World J Surg. 2010;34(10):2338–43.
- Dionigi G, Bacuzzi A, Rovera F, et al. Shortening hospital stay for thyroid surgery. Exp Rev Med Dev. 2008;5:85.
- Ballantyne JC, LaForge KS. Opioid dependence and addiction during opioid treatment of chronic pain. Pain. 2007;129:235.
- 67. Grass JA. Patient-controlled analgesia. Anesth Analg. 2005;101: 44–61.
- 68. Hutchison RW, Chon EH, Tucker WF, et al. A comparison of a fentanyl, morphine and hydromorphone patient-controlled intravenous delivery for acute postoperative analgesia: a multicenter study of opioid-induced adverse reactions. Hosp Pharm. 2006;41:659.
- 69. Jacobi J, Fraser GL, Coursin DB, et al. Clinical practice guidelines for the sustained use of sedatives and analgesics in the critically ill adult. Crit Care Med. 2002;30:119.
- Lowder JL, Shackelford DP, Holbert D, et al. A randomized, controlled trial to compare ketorolac tromethamine versus placebo after cesarean section to reduce pain and narcotic usage. Am J Obstet Gynecol. 2003;189:1559.
- Ng A, Temple A, Smith G, Emembolu J. Early analgesic effects of parecoxib versus ketorolac following laprascopic sterilization: a randomized controlled trial. Br J Anaesth. 2004;92:846.
- Pavy TJ, Paech MJ, Evans SF. The effect of intravenous ketorolac on opioid requirement and pain after cesarean delivery. Anesth Analg. 2001;92:1010.
- Forrest JB, et al. Ketorolac, diclofenac, and ketoprofen are equally safe for pain relief after major surgery. Br J Anaesth. 2002;88(2): 227–33.
- Subramaniam K, et al. Ketamine as an adjuvant analgesic to opioids: a quantitative and qualitative systematic review. Anesth Analg. 2004;108:631.
- Himmelseher S, Durieux ME. Ketamine for perioperative pain management. Anesthesiology. 2005;102:211.
- Pyati S, Gan TJ. Perioperative pain management. CNS Drugs. 2007;21:185.
- 77. Webb AR, Skinner NS, et al. The addition of a small-dose ketamine infusion to tramadol for postoperative analgesia: a double blinded, placebo-controlled, randomized trial after abdominal surgery. Anesth Analg. 2007;35:199.
- Buvanendran A, Kroin JS. Useful adjuvants for postoperative pain management. Best Pract Res Clin Anaesthesiol. 2007;21:31.
- Bell RF, Dahl JB, Moore RA, Kalso EA. Perioperative ketamine for acute postoperative pain. Cochrane Database Syst Rev. 2006;(1):CD004603.
- Edrich T, Friedrich AD, et al. Ketamine for long-term sedation and analgesia of a burn patient. Anesth Analg. 2004;99:893.
- De Pinto M, Jelecic J, Edwards WT. Very-low-dose ketamine for the management of pain and sedation in the ICU. J Opioid Manag. 2008;4:54.
- Marret E, Rolin M, et al. Meta-analysis of intervenous lidocaine and post-operative recovery after abdominal surgery. Br J Surg. 2008;95:1331.
- McKay A, Gottschalk A, et al. Systemic lidocaine decreased the perioperative opioid analgesic requirements but failed to reduce discharge time after ambulatory surgery. Anesth Analg. 2009;109:1805.
- 84. Daniels SE, et al. A randomized, double-blind, phase III study comparing multiple doses of tapentadol IR, oxycodone IR, and placebo for postoperative (bunionectomy) pain. Curr Med Res Opin. 2009;25(3):765–76.
- Rahimi SY, et al. Postoperative pain management with tramadol after craniotomy: evaluation and cost analysis. J Neurosurg. 2010;112(2):268–72.
- 86. Yener M, Erdogan G, Ozdemir I. Adjuvant use of liquid alginate suspension for post-tonsillectomy morbidity: double-blind ran-

domized clinical trial of efficacy. Otolaryngol Head Neck Surg. 2009;140(5):652–6.

- Turk DC, Okifuji A. Pain terms and taxonomies. In: Bonica's management of pain. 3rd ed. Lippincott Williams & Wilkins. 2001; p. 18–25.
- Stovner LJ, Hagen K. Prevalence, burden, and cost of headache disorders. Curr Opin Neurol. 2006;19:281.
- Loeser JD. Economic implications of pain management. Acta Anaesthesiol Scand. 1999;43:957.
- Breivika H, Collettb B, et al. Survey of chronic pain in Europe: prevalence, impact on daily life and treatment. Eur J Pain. 2006;10(4):287–333.
- Vargas-Schaffer G. Is the WHO analgesic ladder still valid? Twenty four years of experience. Can Fam Physician. 2010;56(6):514–7.
- 92. Swarm RA, Karanikolas M, Cousins MJ. Anaesthetic techniques for pain management. In: Doyle D, Hanks G, MacDonald N, editors. Oxford textbook of palliative medicine. 3rd ed. Oxford: Oxford University Press; 2003. p. 378.
- Ballantyne JC, Mao J. Opioid therapy for chronic pain. N Engl J Med. 2003;349:1943.
- 94. Martell BA, O'Connor PG, Kerns RD, et al. Systematic review: opioid treatment for chronic back pain: prevalence, efficacy and association with addiction. Ann Intern Med. 2007;146:116–27.
- Eisenberg E, McNicol ED, Carr DB. Efficacy and safety of opioid agonists in the treatment of neuropathic pain of nonmalignant origin. JAMA. 2005;293:3043.
- 96. Trescot AM, et al. Opioids in the management of chronic noncancer pain: an update of American society of the interventional pain physicians' (ASIPP) guidelines. Pain Physician. 2008; 11:S5–62.
- Hojsted J, Sjogren P. Addiction to opioids in chronic pain patients: a literature review. Eur J Pain. 2007;11(5):490–518.
- Dunn KM, Saunders KW, Rutter CM, et al. Opioid prescriptions for chronic pain and overdose: a cohort study. Ann Intern Med. 2010;152:85.
- Fitzgibbon DR, Rathmell JP, Michna E, Stephens LS, Posner KL, Domino KB. Malpractice claims associated with medication management for chronic pain. Anesthesiology. 2010;112:948–56.
- McLellan AT, Turner BJ. Chronic noncancer pain management and opioid overdose: time to change prescribing practices. Ann Intern Med. 2010;152:123.
- Daniell HW. Hypogonadism in men consuming sustained-action oral opioids. J Pain. 2002;3(5):377–84.
- 102. Moore JT, Kelz MB. Opiates, sleep, and pain: the adenosinergic link. Anesthesiology. 2009;111(6):1175–6.
- Angst M, Clark D. Opioid-induced hyperalgesia: a qualitative systematic review. Anesthesiology. 2006;104:570–87.
- 104. Chang G, Chen L, Mao J. Opioid tolerance and hyperalgesia. Med Clin North Am. 2007;91:199.
- 105. Choul R, Fanciullo GJ, et al. Clinical guidelines for use of chronic opioid therapy in chronic noncancer pain. Anesthesiol Clin. 2009;10(2):113–30.
- 106. Shaheen PE, Walsh D, Lasheen W, et al. Opioid equianalgesic tables: are they all equally dangerous? J Pain Symptom Manage. 2009;38:409–17.
- 107. Furlan AD, Sandoval JA, Mailis-Gagnon A, Tunks E. Opioids for chronic noncancer pain: a meta-analysis of effectiveness and side effects. Can Med Assoc J. 2006;174:1589.
- Duhmke RM, Cornblath DD, Hollingshead JR. Tramadol for neuropathic pain. Cochrane Database Syst Rev. 2004;CD003726.
- Mariconti P, Collini R. Tramadol SR in arthrosic and neuropathic pain. Minerva Anestesiol. 2008;73(3):63–8.
- 110. Afilalo M, et al. Efficacy and safety of tapentadol extended release compared with oxycodone controlled release for the management of moderate to severe chronic pain related to osteoarthritis of the

knee: a randomized, double-blind, placebo- and active-controlled phase III study. Clin Drug Investig. 2010;30(8):489–505.

- Nucynta[®] Package Insert. Ortho-McNeil-Janssen Pharmaceuticals, Inc. 2009.
- 112. Ultram ER[®] Package Insert. Ortho-McNeil-Janssen Pharmaceuticals, Inc. 2009.
- Wiffen PJ, McQuay HJ, Edwards JE, et al. Gabapentin for acute and chronic pain. Cochrane Database Syst Rev. 2005;CD005452.
- 114. Sarantopoulos C, McCallum JB, Kwok WM, et al. Gabapentin decreases membrane calcium currents in injured as well as in control mammalian primary afferent neurons. Reg Anesth Pain Med. 2002;27:47.
- 115. Backonja M, Glanzman RL. Gabapentin dosing for neuropathic pain: evidence from randomized, placebo-controlled clinical trials. Clin Ther. 2003;25:81.
- 116. Dworkin RH, Corbin AE, Young Jr JP, et al. Pregabalin for the treatment of postherpetic neuralgia: a randomized, placebo-controlled trial. Neurology. 2003;60:1274.
- 117. Dworkin RH, et al. Recommendations for the pharmacological management of neuropathic pain: an overview and literature update. Mayo Clin Proc. 2010;85(3):s3–14.
- 118. Gronseth G, Cruccu G, et al. Practice parameter: the diagnostic evaluation and treatment of trigeminal neuralgia (an evidencebased review). Neurology. 2008;71:1183–90.
- 119. Wiffen PJ, Rees J. Lamotrigine for acute and chronic pain. Cochrane Database Syst Rev. 2007;CD006044.
- Dworkin RH, et al. Pharmacologic management of neuropathic pain: evidence based recommendations. Pain. 2007;132:237–51.
- 121. Muehlbacher M, Nickel MK, et al. Topiramate in treatment of patients with chronic low back pain: a randomized, double-blind, placebo-controlled study. Clin J Pain. 2006;22(6):585–97.
- Mullners WM. Anticonvulsants in migraine prophylaxis: a cochrane review. Cephalagia. 2008;28(6):585–97.
- Richelson E. The pharmacology of antidepressants. Mayo Clin Proc. 2001;76:511.
- 124. Kroenke K, Bair MJ, Damush TM, et al. Optimized antidepressant therapy and pain self-management in primary care patients with depression and musculoskeletal pain: a randomized controlled trial. J Am Med Assoc. 2009;301:2099.
- 125. Sindrup SH, Bach FW, Madsen C, et al. Venlafaxine versus imipramine in painful polyneuropathy: a randomized, controlled trial. Neurology. 2003;60:1284.
- See S, Ginzburg R. Skeletal muscle relaxants. Pharmacotherapy. 2008;28(2):207–13.
- 127. Kearney PM, et al. Do selective cyclo-oxygenase-2 inhibitors and traditional non-steroidal anti-inflammatory drugs increase the risk of atherothrombosis? Meta-analysis of randomized trials. Br Med J. 2006;332:1302–8.
- 128. Solomon SD, Wittes J, Finn PV, et al. Cardiovascular risk of celecoxib in 6 randomized placebo controlled trials: the cross trial safety analysis. Circulation. 2008;117:2104.
- 129. Mamdani M, Juurink DN, Lee DS, et al. Cyclo-oxygenase-2 inhibitors versus non-selective non-steroidal anti-inflammatory drugs and congestive heart failure outcomes in elderly patients: a population-based cohort study. Lancet. 2004;363:1751.
- 130. Zgislason GH, Rasmussen JN, Abildstrom SZ, et al. Increased mortality and cardiovascular morbidity associated with use of nonsteroidal anti-inflammatory drugs in chronic heart failure. Arch Intern Med. 2009;169:141.
- Higuchi K, Umegaki E, Watanabe T, et al. Present status and strategy of NSAIDs-induced small bowel injury. J Gastroenterol. 2009;44(9):879–88.
- 132. Chan CC, Reid CM, Aw TJ, et al. Do COX-2 inhibitors raise blood pressure more than nonselective NSAIDs and placebo? An updated meta-analysis. J Hypertens. 2009;27:2332.

- 133. Mc T. Diuretics, ACE inhibitors and NSAIDs the triple whammy. Med J Aust. 2000;172(4):184–5.
- 134. Wolfe F, Zhao S, Pettitt D. Blood pressure destabilization and edema among 8538 users of celecoxib, rofecoxin and nonselective nonsteroidal anti-inflammatory drugs (NSAID) and nonusers of NSAID receiving ordinary care. J Rheumatol. 2004;31:1143.
- 135. Bhatt D, et al. ACCF/ACG/AHA 2008 expert consensus document on reducing the gastrointestinal risks of antiplatelet therapy and NSAID use. J Am Coll Cardiol. 2008;52:1502–17.
- 136. Rowbotham MC. Mechanisms of neuropathic pain and their implications for the design of clinical trials. Neurology. 2005; 65:S66.
- 137. Headache classification subcommittee of the International Headache Society. The International Classification of Headache Disorders. 2nd ed. Cephalagia. 2004;24(1):S9.
- Finnerup NB, Otto M, McQuay HJ, et al. Algorithm for neuropathic pain treatment: an evidence based proposal. Pain. 2005; 118:289.
- 139. Rozen TD. Relief of anesthesia dolorosa with gabapentin. Headache. 1999;39:761.
- 140. Frese A, Husstedt IW, Ringelstein EB, Evers S. Pharmacologic treatment of central post-stroke pain. Clin J Pain. 2006;22:252.
- 141. Crayton H, Heyman RA, Rossman HS. A multimodal approach to managing the symptoms of multiple sclerosis. Neurology. 2004;63:S12.
- 142. Agostoni E, Frigerio R, Santoro P. Atypical facial pain: clinical considerations an different diagnosis. Neurol Sci. 2005;26(2):71.
- 143. Volcy M, Rapoport AM, Tepper SJ, et al. Persistent idiopathic facial pain responsive to topiramate. Cephalalgia. 2006;26:489.
- 144. Zakrzewska JM, Forsell H, Glenny AM. Interventions for the treatment of burning mouth syndrome. Cochrane Database Syst Rev. 2005;CD002779.
- 145. Tasker RR, Watson CPN. The treatment of central pain. In: Noseworthy JH, editor. Neurological therapeutics: principles and practice. London: Martin Dunitz; 2003. p. 214.
- 146. Taub E, Munz M, Tasker RR. Chronic electrical stimulation of the gasserian ganglion for the relief of pain in a series of 34 patients. J Neurosurg. 1997;86:197.
- Rozen TD. Trigeminal neuralgia and glossopharyngeal neuralgia. Neurol Clin. 2004;22:185.
- 148. Fini G, Gasparini G, Filippini F, et al. The long styloid process syndrome or Eagle's syndrome. J Craniomaxillofac Surg. 2000;28:123.
- 149. Hiwatashi A, Matsushima T, Yoshiura T, et al. MRI of glossopharyngeal neuralgia caused by neurovascular compression. Am J Roentgenol. 2008;191:578.
- Carrieri PB, Montella S, Petracca M. Glossopharyngeal neuralgia as onset of multiple sclerosis. Clin J Pain. 2009;25(8):737–9.
- Nurmikko TJ, Eldridge PR. Trigeminal neuralgia pathophysiology, diagnosis and current treatment. Br J Anaesth. 2001;87: 117–32.
- Love S, Coakham HB. Trigeminal neuralgia: pathology and pathogenesis. Brain. 2001;124:2347–60.
- 153. Wiffen PJ, McQuay HJ, Moore RA. Carbamazepine for acute and chronic pain. Cochrane Database Syst Rev. 2005;CD005451.
- Zakrzewska JM, Lopez BC. Trigeminal neuralgia. Clin Evid. 2005;1669–77.
- 155. Canavero S, Bonicalzi V. Drug therapy of trigeminal neuralgia. Expert Rev Neurother. 2006;6:429–40.
- 156. Kyung RH, Kim C. The long-term outcome of mandibular nerve block with alcohol for the treatment of trigeminal neuralgia. Anesth Analg. IARS 2010.
- 157. Day M. Sphenopalatine Ganglion Analgesia. Curr Rev Pain. 1999;3:342–7.

- 158. Pipolo C, Bussone G, Leone M, et al. Sphenopalatine endoscopic ganglion block in cluster headache: a reevaluation of the procedure after 5 years. Neurol Sci. 2010;31(1):197–9.
- 159. Narouse S, Kapural L, Casanova J, Mekhail N. Sphenopalatine ganglion radiofrequency ablation for the management of chronic cluster headache. Headache. 2009;49:571–7.
- 160. Kanpolat Y, Savas A, Bekar A, Berk C. Percutaneous controlled radiofrequency trigeminal rhizotomy for the treatment of idiopathic trigeminal neuralgia: 25-year experience with 1600 patients. Neurosurgery. 2001;48:524–32.
- 161. Brisman R. Microvascular decompression vs. gamma knife radiosurgery for typical trigeminal neuralgia: preliminary findings. Stereotact Funct Neurosurg. 2007;85:94–8.
- 162. Scharpf J, Hynds Karnell L, et al. The role of pain in head and neck cancer recurrence and survivorship. Arch Otolaryngol Head Neck Surg. 2009;135(8):789–94.
- 163. Hammerlid E, Ahlner-Elmqvist M, Bjordal K, et al. A prospective multicentre study in Sweden and Norway of mental distress and psychiatric morbidity in head and neck cancer patients. Br J Cancer. 1999;80:766.
- 164. Verdonck-de Leeuw IM, de Bree R, Keizer AL, et al. Computerized prospective screening for high levels of emotional distress in head and neck cancer patients and referral rate to psychosocial care. Oral Oncol. 2009;45:e129.
- 165. Lydiatt WM, Denman D, McNeilly DP, et al. A randomized, placebo-controlled trial of citalopram for the prevention of major depression during treatment for head and neck cancer. Arch Otolaryngol Head Neck Surg. 2008;134:528.
- 166. Varghese BT, Koshy RC. Endoscopic transnasal neurolytic sphenopalatine block for head and neck cancer pain. J Laryngol Otol. 2001;115(5):385–7.
- 167. Bhatnagar S, Mishra S. Interventional pain management in cancer patients: experience from tertiary care center in India. Am Soc Clin Oncology (ASCO) Annu Meet Proc. 2008; 26(15):20739.
- 168. Koizuka S, Saito S, et al. Percutaneous radio frequency mandibular nerve rhizotomy guided by CT fluoroscopy. Am J Neuroradiology. 2006;27:1647–8.
- 169. Koizuka S, Saito S, et al. Technical communication: percutaneous radiofrequency mandibular nerve rhizotomy guided by high-speed real-time computed tomography fluoroscopy. Anesth Analg. 2010;111(3):763–7.
- 170. Kohase H, Umino M, Shibaji T, et al. Application of a mandibular nerve block using an indwelling catheter for intractable cancer pain. Acta Anaesthesiol Scand. 2004;48(3):382–3.
- 171. Gangi A, Dietemann JL, Schultz A, et al. Interventional radiologic procedures with CT guidance in cancer pain management. Radiographics. 1996;16:1289–304.
- 172. Mehio AK, Shah SK. Alleviating head and neck pain. Otolaryngol Clin North Am. 2009;42:143–59.
- 173. Vargese BT, Koshy RC, Sebastian P, Joseph E. Combined sphenopalatine ganglion and mandibular nerve neurolytic block for pain due to advanced head and neck cancer. Palliat Med. 2002;16(5):447–8.
- 174. Lundborg C, Dahm P, Nitescu P, et al. High intrathecal bupivacaine for severe pain in the head and neck. Acta Anaesthesiol Scand. 2009;53(7):908–13.
- 175. Baker L, Balls J, Regnard C, Pridie A. Cervical intrathecal analgesia for head and neck/upper limb cancer pain: six case reports. Palliat Med. 2007;21(6):543–5.
- 176. Andersen PE, Cohen JI, Everts EC, et al. Intrathecal narcotics for relief of pain from head and neck cancer. Arch Otolaryngol Head Neck Surg. 1991;117(11):1277–80.

- 177. Schreiber CP, Hutchinson S, Webster CJ, et al. Prevalence of migraine in patients with a history of self-reported or physician-diagnosed "sinus" headache. Arch Intern Med. 2004;164:1769.
- 178. Cady RK, Dodick DW, Levine HL, et al. Sinus headache: a neurology, otolaryngology, allergy, and primary care consensus on diagnosis and treatment. Mayo Clin Proc. 2005;80:908.
- 179. Tarabichi M. Characteristics of sinus-related pain. Otolaryngol Head Neck Surg. 2000;122:842.
- 180. Levine HL, Setzsen M, Cady RK, et al. An otolaryngology, neurology, allergy, and primary care consensus on diagnosis and

treatment of sinus headache. Otolaryngol Head Neck Surg. 2006;134:516.

- 181. Behin F, Behin B, Bigal ME, Lipton RB. Surgical treatment of patients with refractory migraine headaches and intranasal contact points. Cephalagia. 2005;25(6):439–43.
- 182. Marion DW. Evaluation of the patient with neck pain and cervical spine disorders. In: Basow DS, editor. UpToDate. Waltham: UpToDate. 2010.
- 183. Marion, DW. Treatment of neck pain. In: Basow DS, editor. UpToDate. Waltham: UpToDate. 2010.
- 184. Van Zundert J. Cervical radicular pain. Pain Pract. 2010;10(1):1-17.

Postoperative Care

Jody Jones and Brigid Flynn

Introduction

Although many otolaryngologic procedures are done in the ambulatory setting and postoperative care and monitoring will focus on discharge readiness, patients' status post surgery on the head and neck is predisposed to numerous complications in the immediate postoperative period that are unlike complications occurring in other surgical patients. In addition to the commonly encountered postoperative problems such as nausea, vomiting, and pain that affect patient satisfaction, these patients are at risk for critical, lifethreatening postoperative sequelae requiring superior clinical acumen and expedited care in the post-anesthesia care unit (PACU).

There are numerous reasons why the postoperative care of the otolaryngologic patient may be challenging. (1) The anatomy involved in ear, nose, and throat surgery either directly involves or is intimately related to the structures and thus the integrity of the airway. (2) Endocrine dysfunction following surgery can disrupt key elements in the body and have devastating effects. (3) The areas involved in these surgeries are highly vascularized thus increasing the risk of lifethreatening bleeding and edema. (4) The innervation of the ear, nose and throat can easily be disrupted during surgery leading to severe neurologic sequelae. In addition to the discussion of basic PACU discharge criteria for the ambulatory and in patient, this chapter will elucidate etiologies and treatments of both common and life-threatening postoperative complications associated with common ear, nose, and throat surgeries.

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The PACU

The PACU is an area designated for the monitoring of patients in the immediate postoperative period after general or regional anesthesia and monitored anesthetic care (MAC). According to published standards, patients are routinely observed during this period characterized by the potential for an abrupt onset and rapidly progressing change in clinical status by specially trained physicians and nurses ^[1–4]. Frequent evaluations seek out postoperative complications and assess the degree of recovery from anesthetic and surgical derangements with the goal of a safe transfer to a less acute inpatient setting or discharge to home in the case of outpatient surgery (Tables 23.1–23.2). Guidelines and standardized scoring algorithms such as the Aldrete post-anesthesia recovery score have been developed to define PACU discharge suitability (Table 23.3) ^[4,5].

Depending on the center, outpatients may be discharged home from the PACU, called phase 1, or transferred to the less acute phase 2 recovery area and discharged from there. The existence of separate phase 1 and 2 recovery areas is predicated on the idea that outpatient recovery can be divided into two phases: phase 1, the immediate postoperative intensive care level for patients during awakening from anesthesia and continues until standard PACU criteria are met; and phase 2, a lower level of care that ensures the patient is ready to go home ^[1]. Stable patients, status post small peripheral procedures may be discharged directly from the OR to the phase 2 area by specific order of the attending anesthesiologist after adequate anesthetic recovery has been established. ^[1, 4]. Table 23.4 is an example of a phase 2 discharge-specific recovery scoring system ^[6].

Tonsillectomy and Adenoidectomy Patients

Tonsillectomy and adenoidectomy are among the most commonly performed surgical procedures in the USA and often are performed in an ambulatory setting ^[7, 8]. Common indications of combined tonsillectomy and adenoidectomy

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Table 23.1 Summary of recovery and discharge criteria [4]

General principles

Medical supervision of recovery and discharge is the responsibility of the supervising practitioner

The recovery area should be equipped with appropriate monitoring and resuscitation equipment

Patients should be monitored until appropriate discharge criteria are satisfied

Level of consciousness, vital signs, and oxygenation (when indicated) should be recorded at regular intervals

A nurse or other individual trained to monitor patients and recognize complications should be in attendance until discharge criteria are fulfilled

An individual capable of managing complications should be immediately available until discharge criteria are fulfilled

Guidelines for discharge

Patients should be alert and oriented. Patients whose mental status was initially abnormal should have returned to their baseline

Vital signs should be stable and within acceptable limits. Discharge should occur after patients have met specified criteria. Use of scoring systems may assist in documentation of fitness for discharge. Outpatients should be discharged to a responsible adult who will accompany them home and be able to report any postprocedure complications. Outpatients should be provided with written instructions regarding postprocedure diet, medications, activities, and a phone number to be called in case of emergency

Each patient care facility should develop suitable recovery and discharge criteria. The table lists some of the basic principles that might be incorporated in these criteria

Table 23.2 Summary of recommendations for discharge [4]

Requiring that patients urinate before discharge

The requirement for urination before discharge should not be part of a routine discharge protocol and may only be necessary for selected patients

Requiring that patients drink clear fluids without vomiting before discharge

The demonstrated ability to drink and retain clear fluids should not be part of a routine discharge protocol but may be appropriate for selected patients

Requiring that patients have a responsible individual accompany them home. As part of a discharge protocol, patients should routinely be required to have a responsible individual accompany them home

Requiring a minimum mandatory stay in recovery

A mandatory minimum stay should not be required. Patients should be observed until they are no longer at increased risk for cardiorespiratory depression. Discharge criteria should be designed to minimize the risk of central nervous system or cardiorespiratory depression after discharge

Table 23.3 The post-anesthetic recovery/Aldrete score ^[5], modified

Table 23.5 The post-anesthetic recovery/Addrete score	Score				
Consciousness					
Alert	2				
Drowsy	1				
Unresponsive	0				
Circulation					
±20 mmHg preanesthetic level	2				
±20–50 mmHg preanesthetic level	1				
±>50 mmHg preanesthetic level	0				
Oxygenation					
SaO ₂ >92%	2				
$SaO_2 > 90\%$ w/supplemental O_2	1				
$SaO_2 < 90\%$ w/supplemental O_2	0				
Respiration					
Able to breathe deeply, cough freely	2				
Dyspnea, shallow or limited breathing	1				
Apnea	0				
Activity					
Moves four extremities on command	2				
Moves 2 extremities on command	1				
Unable to move extremities on command	0				
Maximum total score	10				

A score of 9 or greater indicates adequate anesthetic recovery for discharge

include recurrent tonsillitis, especially if complicated by recurrent febrile seizures or heart valve lesions, chronic tonsillitis, peritonsillar abscess, heroic snoring, obstructive sleep apnea, and adenotonsillar hypertrophy complicated by dysphagia, sleep abnormalities, or occlusive craniofacial growth abnormalities ^[9–11].

Despite the frequent performance of these procedures, post-anesthetic management of these patients is not necessarily straightforward. Tonsillectomy surgery itself is an independent risk factor for postoperative complications in children. The patient is potentially a small child with a history of obstructive airway symptoms, poor intravenous access, and the potential for blood contamination of the lower airway. Chronic upper airway obstruction is associated with systemic hypertension, pulmonary hypertension potentially complicated by cor pulmonale, altered ventilation-perfusion relationships, and malignant dysrhythmias ^[10, 12–14].

The complication rate of adenotonsillectomy is approximately 8%. The majority of these complications involve postoperative bleeding and the development of stridor in children under the age of 4 ^[13, 15]. Post-anesthetic management is often

 Table 23.4
 Fast-track criteria to determine whether outpatients can be transferred directly from the OR to the phase 2 unit ^[6]

	Score ^a			
Level of consciousness				
Awake and oriented	2			
Arousable with minimal stimulation	1			
Responsive only to tactile stimulation	0			
Physical activity				
Able to move all extremities on command	2			
Some weakness in movement of extremities	1			
Unable to voluntarily move extremities	0			
Hemodynamic stability				
Blood pressure within 15% of baseline MAP value	2			
Blood pressure within 15-30% of baseline MAP value	1			
Blood pressure <30% below baseline MAP value	0			
Respiratory stability				
Able to breathe deeply	2			
Tachypnea with good cough	1			
Dyspneic with weak cough	0			
Oxygen saturation status				
Maintains value >90% on room air	2			
Requires supplemental oxygen (nasal prongs)	1			
Saturation <90% with supplemental oxygen	0			
Postoperative pain assessment				
None or mild discomfort	2			
Moderate to severe pain controlled with IV analgesics	1			
Persistent severe pain	0			
Postoperative emetic symptoms				
None or mild nausea with no active vomiting	2			
Transient vomiting or retching	1			
Persistent moderate to severe nausea and vomiting	0			
Maximal total score	14			

^aA minimal score of 12 with no score less than 1 in any individual category is required for a patient to be fast-tracked (i.e., bypass the post-anesthesia care unit) after general anesthesia

complicated by postoperative nausea and vomiting (PONV) occurring in up to 70% of patients secondary to passive intraand postoperative drainage of blood into the stomach ^[3, 10, 12, 16, 17]. Less frequently encountered is post-obstructive pulmonary edema, which may develop after relief of a long-standing compensated upper airway obstruction.

Postoperative Hemorrhage

The management of postoperative hemorrhage is the most significant problem encountered in the PACU after these procedures. Primary post-tonsillectomy hemorrhage occurs within 24 h (usually presenting in less than 6 h) and can potentially lead to hypovolemic shock and or require reoperation ^[7, 8, 10, 13, 14, 18]. The ability to swallow blood may conceal a slow continuous bleed in the postoperative patient. The incidence of postoperative hemorrhage within the first 24 h is approximately 12.3% in children younger than 3 years of age and 7.9% in older patients. The incidence of post-tonsillectomy bleeding requiring treatment is much smaller, ranging from 2% to 10%; the incidence of subsequent reoperation has been reported to be as high as 5.5%. Factors that contribute to postoperative bleeding include a history of coagulopathy and high postoperative blood pressure ^[10, 13, 14, 19].

Once postoperative hemorrhage is recognized or suspected, a rapid and coordinated treatment effort is required since it is difficult to accurately determine the blood volume lost or the hemodynamic reserve of the patient. Blood-stained saliva may be considered an indication of bleeding potential and suggest the need for more frequent assessment [15, 18]. Other signs and symptoms of significant blood loss include tachycardia, frequent swallowing, irritation, pallor, anuria, and nausea and vomiting. Hypotension is a late sign ^[10, 14, 15, 18]. In addition to management strategies, the PACU team is responsible for coordinating the communication with other members of the care team including members of the otolaryngologic and OR anesthesia teams. The operating room and blood bank must also be notified in case the patient needs to return for reexploration or needs blood for transfusion respectfully.

Medical management is complicated by hypovolemia and potential hemodynamic instability. The risk of aspiration is increased by ingested blood, which is itself emetogenic. Lastly, but critically important is the recognition that there is a high likelihood of difficult laryngoscopy due to visual impairment secondary to copious bloody secretions and airway edema. Resuscitation is performed with the provision of supplemental oxygen and the administration of intravenous fluids through two secure large-bore intravenous lines. The patient may be placed in a sitting or a lateral decubitus position. The latter minimizes the possibility of aspiration and swallowing of blood by allowing it to flow away from the hypopharynx ^[14, 15, 18]. Relevant laboratory investigations include a complete blood count and coagulation studies. Hypotension may be managed with vasopressors if fluid resuscitation is not adequate [10, 14, 15, 18].

Airway management may become necessary after adequate (or failure of) fluid resuscitation. It is prudent to avoid multiple intubation attempts. Cricothyroidotomy equipment should be available in case of failure. Rapid sequence induction must be performed in this context as the application of positive pressure ventilation may insufflate a full stomach or force blood into the tracheobronchial tree. It is also prudent to use an endotracheal tube one size smaller than that which was used originally due to the high likelihood of airway edema. The presence of oozing blood and clots may obscure laryngoscopic view and the airway itself may be edematous secondary to obstructed venous and lymphatic drainage and prior surgical manipulation. Suction should be rapidly available, but clearing the oropharynx may require digital manipulation. It is recommended that two suction sources (double suction setup) with hard tip yankauer suction be available should one prove to be inadequate or clot during airway management. Intubation attempts should cease and cricothyroidotomy should commence if hypoxia develops ^[10, 14, 15, 18]. If awake fiberoptic intubation is attempted, a double suction setup (i.e., one suction port on the fiberscope and one for a hard Yankauer suction) is strongly recommended.

Postoperative Nausea and Vomiting

Postoperative nausea and vomiting (PONV) can be described as a triad of symptoms and signs: (1) nausea, the subjective unpleasant sensation associated with the awareness of the urge to vomit; (2) vomiting, the phenomenon of expulsion of gastric contents from the mouth; and (3) retching, the labored spasmodic rhythmic contractions without the expulsion of gastric contents ^[8]. Patient, anesthesia and medications, and surgery-specific risk factors affect the incidence of PONV. Risk factors for PONV of particular relevance to post-tonsillectomy patients include the presence of blood passively drained into the stomach intraoperatively, a lack of a smoking history, the use of volatile agents during anesthetic maintenance, opioid exposure, pharyngeal location of surgery, and high levels of postoperative anxiety or pain ^[3, 8, 12, 16, 17, 20].

Without prophylaxis, greater than 70% of patients will experience PONV after adenotonsillectomy ^[3]. PONV is among the most common postoperative complications associated with these procedures and is the commonest cause of delayed discharge and overnight admission in planned ambulatory cases [8, 13, 17]. PONV is also associated with increased rates of pulmonary aspiration and patient discomfort^[3]. A number of prophylactic interventions are employed pre- or intraoperatively to reduce the incidence of PONV such as the use of multimodal pharmacologic therapy with actions on the chemoreceptor trigger zone. Pharmacologic interventions may include: dexamethasone, a serotonin receptor antagonist such as ondansetron or granisetron, and/or dopamine antagonists such as droperidol [8, 12, 17, 21]. Table 23.5 lists various antiemetics by mechanism of action along with associated side effects. Selection of less emetogenic anesthetic maintenance techniques (e.g., TIVA with propofol) and/or minimization of the use of opioids may be warranted [3, 8, 16, 21-24] and can be accomplished with the use of multimodal analgesic techniques.

Table 23.5	Antiemetics	used for postoperative nausea and vomiting
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Mechanism and agents	Side effects	
Anticholinergic	Dry mouth, blurry vision, dilated pupils, sedation, urinary retention, drowsiness	
Scopolamine (Transderm Scop)		
Serotonin receptor antagonists	QT prolongation, headache, diarrhea, constipation, malaise	
Ondansetron (Zofran)		
Granisetron (Kytril)		
Dolasetron (Anzemet)		
Dopamine antagonists	Sedation, extrapyramidal effects, QT prolongation, neuroleptic malignant syndrome, blood dyscrasias	
Droperidol (Inapsine)		
Metoclopramide (Reglan)		
Prochlorperazine (Compazine)		
Promethazine (Phenergen)		
Chlorpromazine (Thorazine)		
Antihistamines	Sedation, urinary retention	
Dimenhydrinate (Dramimine)		
Cyclizine (Marezine)		
Diphenhydramine (Benadryl)		
Meclizine (Antivert)		
Steroids	Hyperglycemia, possible poor wound healing	
Dexamethasone		
Methylprednisolone (Medrol)		
Unknown mechanism		
Trimethobenzamide (Tigan)	Extrapyramidal effects, hypotension, blood dyscrasias, blurry vision, sedation, headaches	

Postoperative prophylactic maneuvers include the maintenance of normovolemia with intravascular fluids being continued until the patient is able to tolerate fluids by mouth and the minimization of opioid administration ^[8, 12, 17]. However, a subset of patients who have received antiemetic prophylaxis will still require rescue therapy in the PACU. One should investigate the presence of contributing factors such as continued bleeding with subsequent swallowing of blood, excessive opioid use, or dehydration before administering additional antiemetic agents. Patients who experience PONV within 6 h of surgery despite receiving appropriate prophylaxis should be administered a "rescue" antiemetic agent that possesses activity via a different mechanistic pathway ^[3, 8, 17].

Thyroidectomy Patients

Typical indications for thyroidectomy are thyroid malignancy, goiter, refractory hyperthyroidism, and Graves' disease. Thyroidectomy is described as a safe, but challenging procedure that demands complete anatomic knowledge of the visceral neck. This includes familiarity with variations concerning relationships of the important structures contained therein, specifically the nerves and the parathyroid glands ^[25–27].

With good surgical technique, major complications are uncommon, but include laryngeal nerve damage leading to vocal cord paralysis or voice changes (1–2.3%, unilateral), hematoma development within the neck with possible airway obstruction (1–3%), and hypocalcemia (2–8%) secondary to inadvertent removal or damage of the parathyroid glands ^[7, 25, 26, 28]. It is important to maintain vigilance in the acute postoperative period for signs of bilateral recurrent laryngeal nerve damage, rapidly expanding neck hematoma, and severe hypocalcemia as each of these complications necessitates emergent corrective therapy ^[25, 26, 28].

Laryngeal Nerve Injuries

The larynx is innervated by paired superior and recurrent (or inferior) laryngeal nerves. The superior laryngeal nerve provides motor innervation to the cricothyroid muscles and sensation to the mucosa above the vocal cords. The recurrent laryngeal nerves innervate the intrinsic muscles of the larynx except the cricothyroid muscles and provide sensory innervation to the mucosa at and below the vocal cords ^[3, 12, 26, 29].

The recurrent laryngeal nerve, due to its anatomical relationships and variable course, is at risk of iatrogenic damage during thyroidectomy ^[26]. The nerve courses upward from the mediastinum in the tracheoesophageal groove keeping a close relationship with the thyroid gland and its surrounding structures. Numerous variations of the nerve's course, its branching pattern, and relationships to other anatomical structures have been documented ^[26, 28]. Subsequently, the most common neural injury after thyroid surgery is unilateral damage to a recurrent laryngeal nerve ^[25, 26, 28].

Unilateral recurrent nerve injury affects the excursion of the ipsilateral vocal cord, usually rendering it paralyzed in a paramedian position. This may not be apparent immediately after extubation, but usually presents as voice changes, aphonia, or less commonly, with symptoms of dysphagia or aspiration. The diagnosis may be confirmed with video or fiberoptic flexible laryngoscopy ^[25]. Unilateral vocal cord paralysis usually does not present an acute problem in the PACU and is managed expectantly on an outpatient basis ^[25, 30]. Bilateral recurrent laryngeal nerve injury is usually recognized at extubation or immediately in the postoperative period ^[25]. This is a potential airway emergency as the paralyzed vocal cords may potentially juxtapose under the influence of the subatmospheric pressure generated at the glottis by inspiration. The rapid development of stridor and respiratory distress are common presenting signs. The diagnosis can be confirmed with flexible fiberoptic laryngoscopy but the need for immediate reintubation or emergent tracheotomy assumes primacy should the patient develop clinically significant airway compromise. Bilateral recurrent laryngeal nerve damage also causes loss of sensation at and below the vocal cords, increasing the risk of aspiration ^[25, 29, 30].

The external branch of the superior laryngeal nerve maintains a close relationship with the superior thyroid vessels and may also be damaged during dissection of the superior thyroid pole and ligature of the superior thyroid pedicle. This injury causes paralysis of the cricothyroid muscle and subsequent inability to lengthen and stiffen the ipsilateral vocal cord. Subsequently, voice pitch modulation may be impaired, a change likely to be of little consequence in most patients ^[26, 28].

Hypocalcemia

Hypocalcemia is the most common complication associated with thyroidectomy, reported to occur in up to 8% of procedures. This is usually, but not always, due to inadvertent removal or devascularization with subsequent necrosis of the parathyroid glands. These glands are very small, usually are four in number, range between 6 and 8 mm in size, and are variably found on, near, or in (7%) the thyroid gland ^[25, 26, 28]. It is theorized that a transient drop in serum calcium levels occurs in all patients after thyroidectomy secondary to a short-lived parathyroid insufficiency induced by surgical manipulation. The mechanism of post-thyroidectomy hypocalcemia in the context of retained parathyroid glands is poorly understood ^[25].

Most post-thyroidectomy hypocalcemia is asymptomatic. Serum calcium, albumin, and magnesium levels are drawn periodically in the postoperative period to assist in monitoring and diagnosis. Post-thyroidectomy hypocalcemia is considered present when calcium treatment is triggered by the demonstration of symptoms of hypocalcemia; asymptomatic post-thyroidectomy hypocalcemia is not routinely treated unless the rate of decline of serum calcium is excessive (Table 23.6)^[25].

Symptoms that will be seen in the post-anesthetic care unit can manifest within 1–3 h after surgery and reflect the increased neuronal activity induced by hypocalcemia. These include perioral numbness, tinnitus, and muscle cramping.

Table 23.6 Clinical manifestations of hypocalcemia

Psychiatric	
Seizures	
Anxiety	
Neuromuscular	
Chvostek's sign	
Trousseau's sign	
Circumoral or acral (e.g., fingers, toes) parasthesias	
Muscle stiffness, myalgias	
Cardiovascular	
Prolonged QT interval	
Hypotension	
Autonomic	
Bronchospasm	
Diaphoresis	

Chvostek's (the elicitation of facial spasms through percussion of the facial nerve at the stylomastoid process) and Trousseau's signs (the elicitation of carpopedal spasm after 3 min of brachial artery occlusion) may be appreciated but are rarely pursued. Notably, these signs may be elicited in a small number of patients preoperatively. As the laryngeal muscles are particularly sensitive to hypocalcemia, the patient may demonstrate stridor, a sign of potential laryngospasm. Severe untreated hypocalcemia leads to seizures and the development of cardiac dysrhythmias ^[1, 3, 7, 25, 31, 32].

Moderately symptomatic hypocalcemia in the postoperative period may be treated with a gram of intravenous calcium gluconate given over a 10- to 30-min period ^[25, 26]. Oral supplementation with calcium carbonate may be employed if tolerated and in the absence of severe symptoms with the goal of delivering one to two grams of elemental calcium daily in 4–6 divided doses. In the presence of severe symptoms or with failure of initial calcium therapy, one may initiate an intravenous infusion of calcium chloride at a rate of 1–2 mg/kg/hour until symptoms resolve or calcium levels normalize.

Post-thyroidectomy hypocalcemia can be defined as either transient or permanent based on the duration of need for calcium replacement. Permanent hypocalcemia (treatment duration greater than one year) is very uncommon ^[25–27, 31, 32].

Postoperative Hemorrhage

The incidence of postoperative hematoma following thyroidectomy is reported to be from 1% to 3% with less than 1% requiring surgical intervention ^[26]. However uncommon, it should be remembered that an expanding cervical hematoma is an airway emergency as the associated mass effect

may cause tracheal obstruction. Venous and lymphatic congestion from the hematoma will contribute to airway obstruction and airway management difficulties by causing airway edema and anatomic distortion. Compression of the trachea occurs on the posterior membranous portion of the trachea due to lack of cartilaginous protection. The most common signs and symptoms of a neck hematoma include sensations of pain or pressure, dysphagia, respiratory distress, and increased drainage volume from a surgical drain left in place [1,7,25,28,29].

Suspicion of an expanding wound hematoma necessitates reopening and evacuation of the wound preferably in the operating room, but may be done at the bedside in emergent situations. In an emergency, wound decompression assumes primacy even before attempts at securing the airway because performing a tracheotomy or cricothyroidotomy may not be possible due to blatant hematoma interference and subsequent distortion of anatomical structures which may also obstruct the passage of an endotracheal tube. Once the hematoma is evacuated, a definitive airway should be secured. The patient may then be moved into the operating room for surgical reexploration and revision ^[1, 7, 25, 26, 28, 29].

Parathyroidectomy Patients

Parathyroidectomy is the only definitive treatment for primary hyperparathyroidism. The surgical procedure involved in removing the parathyroid glands is known to be a safe, but sometimes difficult, operation. In fact, the most frequent complication is failure to remove all parathyroid tissue due to the anatomical challenges involved in locating the parathyroid glands. Despite this, experienced surgeons are able to cure more than 95% of patients with parathyroid disease. Perioperative morbidity is uncommon, occurring in less than 1% of patients ^[31, 33].

As previously stated, the most common complication associated with primary parathyroid surgery is failure to cure the disease, which occurs in less than 5% of patients. Other complications include permanent hypoparathyroidism with subsequent hypocalcemia, recurrent laryngeal nerve damage with subsequent vocal cord paralysis (less than 1%), and wound hematoma. Reoperations are less successful, offering an 80% chance of cure with increased incidences of complications (hypocalcemia, 25%; recurrent laryngeal nerve injury, 1%)^[33]. Though the incidence rates of these specific complications may differ, the presentations and treatments of these complications due to parathyroidectomy are the same as those occurring in the context of thyroidectomy. The reader is referred to that section for a more detailed discussion concerning the presentation and treatment of these complications.

Laryngeal Nerve Injuries

The incidence of recurrent laryngeal nerve injury in the context of parathyroid surgery is less than 1%. Unilateral recurrent laryngeal nerve injury will present either shortly after extubation in the operating room or in the post-anesthetic care unit. The most common presentation is hoarseness or a change in voice quality. Unilateral vocal cord injury does not necessitate any immediate or emergent intervention aside from referral to an otolaryngologist ^[29, 30, 32].

Bilateral recurrent laryngeal nerve damage is rare in initial parathyroid exploration. However, reexploration carries a 6.6% risk of permanent vocal cord paralysis with 20% of these complications requiring subsequent tracheostomy ^[25, 32, 33].

Hypocalcemia

As is the case with thyroidectomy, temporary hypocalcemia is common after parathyroidectomy and can be symptomatic or asymptomatic. Permanent hypocalcemia is rare, having a 0.5% incidence. Measurement of serum calcium is not routinely performed in initial explorations, but may be considered in reexplorations as symptomatic hypocalcemia is more frequently encountered in this context ^[25, 31–33].

Postoperative Hemorrhage

The incidence of wound hematoma after initial parathyroid exploration is 0.3%, but, as in thyroidectomy, its development is potentially catastrophic. Reexploration is obligatory and may be performed at the bedside in an emergency, though this is rarely necessary ^[25, 32, 33].

Neck Dissection Surgery

Neck dissection surgery is the systematic removal of the cervical lymph nodes. It is usually employed to excise metastatic head, neck, or thyroid cancer and is the standard treatment for cervical metastases ^[3, 7, 34, 35]. Unfortunately, radical neck dissection surgery is associated with significant morbidity, which is the reason behind the development of more conservative approaches ^[36]. Although more conservative approaches are termed modified radical, selective, and extended neck dissections and are defined by the extent of anatomical resection involved ^[1, 3, 7, 36, 37], they are still associated with significant postoperative morbidity. Please refer to Chap. 15 on Head and Neck Cancer Surgery for a detailed discussion of the surgical procedures and anatomic structures involved.

Postoperative complications associated with neck dissections include cranial nerve, laryngeal nerve, and brachial plexus damage, postoperative hematoma formation, chylous leak, and carotid artery exposure and rupture ^[3, 34, 35]. Neck dissection in the context of thyroidectomy carries the additional risk of hypocalcemia and an increased incidence of recurrent laryngeal nerve damage relative to dissections performed for non-thyroidal malignancies ^[34, 35].

Post-anesthetic management may be further complicated by patient history of exposure to radiation therapy of the head and neck causing stiff, noncompliant tissues, long-term cigarette smoking with underlying lung disease, and alcohol abuse with subsequent dependence or malnutrition ^[1, 29]. It must also be noted that patients may have had a tracheostomy placed pre- or intraoperatively and drains are usually present to decompress the surgical wound as these interventions are associated with postoperative bleeding, especially if tissues are more friable due to radiation exposure.

Carotid Rupture

Carotid artery rupture is the most severe presentation of a collection of clinical signs termed the *carotid blowout syndrome or CBS*. There are three presentations of this syndrome: type I, threatened; type 2, impending; and type 3, acute carotid blowout. *Threatened blowout* is defined as the presence of carotid exposure or radiologic evidence of rupture. *Impending blowout*, also called a sentinel hemorrhage, is characterized by an episode of transcervical hemorrhage that may resolve spontaneously or under exogenously applied pressure. It is often self-limited and may precede acute carotid blowout by moments or months. *Acute carotid blowout* [38, 39].

Carotid blowout is the most feared complication of head and neck procedures because it is rapidly fatal, with reports estimating a 40% mortality rate and a 60% neurologic morbidity rate ^[1, 7, 36, 38, 39]. The incidence of CBS after neck dissection is 3–4%. Patient-specific risk factors of carotid arterial exposure and rupture include previous radiation therapy, malnutrition, infection, and history of diabetes. Radiation therapy has been demonstrated to cause obliteration of the vasa vasorum and ultimately weaken arterial walls, predisposing them to extravasation ^[1, 38, 39].

Historically, management of an acute carotid blowout consisted of neck exploration and carotid artery ligation. Therapeutic interventions utilized presently employ endovascular techniques and angiography with the aim of achieving hemostasis and obviating emergent reoperation ^[38, 39].

The management of postoperative carotid blowout syndrome is dictated first by the clinical stability of the patient and then by the type of blowout encountered. Control of the airway, the achievement of hemostasis with focused pressure, and fluid resuscitation after the attainment of large-bore intravenous or central venous access are most important. The practice of placing multiple dressings over a bleeding wound is inappropriate in an acute carotid blowout as pressure diffusely applied will not stop arterial bleeding. Focal pressure such as that provided by a gloved finger is more effective and is less likely to interfere with intubation if needed. Should emergent intubation commence, a targeted history regarding prior neck radiation and airway difficulties will direct the PACU physician toward an awake versus a rapid sequence intubation. At least 4 units of blood should be requested from the blood bank and blood pressure should be supported with vasopressors if needed. Care must be targeted to prevent hypertension especially during airway management, which would increase arterial bleeding or convert a threaten blow out to an overt one. Diagnostic or therapeutic interventions are attempted only after cardiovascular and respiratory stability are achieved [36, 38, 39].

The discovery of an exposed carotid artery in a stable patient should trigger immediate surgical notification. Management options at this point include wound management and transfer to angiography for further investigation. A threatened carotid blowout, or sentinel bleed, and acute carotid blowout are managed endovascularly with stenting, occlusion, or embolization if the patient is stable enough to transfer to an interventional radiology department. Ligation is a modality of last resort, performed only in the context of a stabilized patient who has suffered an acute carotid blowout, but is unable to access or tolerate any endovascular therapy ^[7, 38, 39].

Nerve Injury

A number of cranial nerves are at risk during neck dissection. Facial palsy can be seen postoperatively as a result of damage of branches of the facial nerve [Cranial Nerve (CN) VII]. This may manifest as an inability to depress the lower lip and facial droop. Damage of the vagus nerve (CN X) can cause vocal cord paralysis, hoarseness, and dysphagia. Bilateral damage of its laryngeal branches can increase aspiration risk and cause upper airway obstruction requiring emergent (possibly surgical) airway intervention. The reader is directed toward Laryngeal Nerve Injuries under Thyroidectomy for a more detailed consideration of this situation. Spinal accessory (CN XI) resection performed inadvertently or as part of a radical dissection denervates the trapezius muscle and causes "shoulder syndrome," a condition characterized by impaired abduction, abnormal scapular rotation, and pain and stiffness. Accessory nerve damage also causes weakness of the ipsilateral sternocleidomastoid ^[7, 36, 37]. Hypoglossal nerve (CN IX) damage creates difficulty speaking and eating and may result in asymmetric tongue protrusion on physical examination ^[3, 7, 36, 37].

Other neural structures at risk during neck dissection include the phrenic nerve, the sympathetic chain, and the brachial plexus. Neck dissection below the deep layer of the deep cervical fascia puts the phrenic nerve at risk. Unilateral injury is likely to go unnoticed and only recognized by the presence of a raised hemidiaphragm on a chest roentgenogram, but bilateral injury impairs spontaneous breathing and will result in atelectasis, respiratory compromise, and hypoxemia. Disruption of the sympathetic chain will cause a unilateral Horner's syndrome consisting of ptosis, miosis, and anhidrosis ^[3, 7, 36, 37].

As stated above, neck dissections that include a thyroidectomy are associated with an increased incidence of recurrent laryngeal nerve damage. Please refer to Laryngeal Nerve Injuries under Thyroidectomy. Bilateral laryngeal nerve palsy resulting in airway obstruction is rare in this context ^[34, 35].

Hematoma

Postoperative neck hematoma commonly results from poor intraoperative hemostasis, loss of a ligature, drain occlusion, hypertension, excessive coughing, bucking, and agitation. Postoperative care should focus on relief of anxiety, pain, PONV, and coughing. Immediate reopening of the wound and decompression of the neck is indicated should signs of airway obstruction develop ^[7]. The postoperative management of a neck hematoma is discussed in more detail under Thyroidectomy.

Chylous Fistula

The thoracic duct runs into the posterior mediastinum and opens up at the junction of the left jugular and subclavian veins. The incidence of chylous leak is between 1% and 2% following neck dissections. Caused by intraoperative injury of the thoracic duct, it often presents within 24 h of surgery and after the resumption of oral intake. The typical presentation consists of an increased surgical drain output of a milky quality. Reexploration is indicated when drainage is excessive (greater than 600 ml/day) or if a leak is noted immediately postoperatively ^[7, 34, 35].

Hypocalcemia

Neck dissection undertaken in the context of thyroid malignancy may be complicated by postoperative hypocalcemia. The reader is referred to the relevant section under Thyroidectomy.

Tracheotomy and Tracheostomy

A tracheotomy is a procedure that involves opening the trachea either surgically or via a percutaneous procedure. A tracheostomy is a tracheotomy performed with the intent of creating a permanent opening and involves the exteriorization of the trachea to the cervical skin ^[7,40]. Nonemergent indications of tracheostomy include partial upper airway obstruction, the need to provide mechanical ventilation when a translaryngeal approach to the respiratory tree is not possible, severe obstructive sleep apnea, and an inability to clear pulmonary secretions. Emergent surgical airway management usually entails cricothyroidotomy or tracheotomy. Though technically more difficult, the latter is preferred when subglottic obstruction is likely to be encountered, as in with the pediatric airway which is most narrow at the level of the cricoid cartilage ^[7,40].

It is estimated that the incidence of postoperative complications associated with tracheostomy ranges from 5% to 40% ^[41]. Complications may be divided by their typical time of onset into early (intraoperative and immediate postoperative) and late ^[41]. Early complications likely to be seen in the PACU include hemorrhage, subcutaneous emphysema possibly complicated by pneumomediastinum or pneumothorax, cannula obstruction with secretions and blood, and cannula dislodgement. Late complications may present in postoperative patients who have chronically placed cannulae. These include tracheal stenosis, tracheo-innominate artery fistula, and tracheoesophageal fistula ^[7,40,41].

Careful attention in the PACU is essential to the shortand long-term viability of the airway ^[7, 40]. Formal recommendations concerning the care of tracheostomized patients are available and are directly relevant to the immediate postoperative period ^[40]. Most importantly, otolaryngologists must be present during the manipulation of a fresh tracheostomy tube (within 5–7 days of placement) since doing so risks the loss of the surgical airway and collapse of the cutaneo-tracheal tract or the inadvertent creation of a false tract. To avoid the need to manipulate a fresh tracheostomy, tracheostomy tubes have dual cannulae, such that the inner cannula may be removed, replaced, or cleaned of secretions and/ or blood clot while leaving the outer cannulae intact. ^[40].

As the filtration and humidification functions of the upper airway have been bypassed, all tracheostomized patients should be administered humidified air in the PACU. Provision of dry air retards muciliary transport and thickens airway secretions, creating distal mucous plugs and increasing the risk of pneumonia. Cuffed tubes should be inflated and cuff pressures should be maintained between 20 and 25 mmHg. Excessive pressure promotes tissue ischemia; however, inadequate pressure encourages the drainage of upper respiratory tract secretions into the bronchial tree. It is suggested that suctioning through the tracheostomy tube be performed as needed, rather than at a fixed frequency, and deep suctioning, which involves advancing the aspiration catheter until distal resistance met, be minimized to reduce airway mucosal damage and irritation ^[40].

Postoperative Hemorrhage

The incidence of continued minor bleeding after tracheostomy is approximately 4%, though major hemorrhage during or immediately after the procedure is uncommon ^[7, 40, 41]. Meticulous hemostasis is generally achieved before leaving the operating room. The immediate postoperative patient is often hypotensive, so bleeding may not become apparent in some cases until after intravascular volume and blood pressure are restored or coughing begins. Minor oozing is common and may be managed conservatively at the bedside with packing ^[41].

Subcutaneous Emphysema

Positive pressure ventilation or coughing against a tightly sutured or packed tracheostomy wound can cause dissection of the tissue planes and subcutaneous emphysema. The incidence has been reported to be as high as 9% in adults with the incidence of the development of associated pneumothorax of 4%. When subcutaneous emphysema is encountered in the PACU, a chest radiograph should be obtained to rule out pneumothorax. The emphysema should resolve spontaneously over days and does not necessarily mandate intervention as tracheal compression due to subcutaneous air is exceedingly uncommon ^[7, 40, 41].

Cannula Obstruction, Malposition, or Dislodgement

Cannula obstruction and malposition are not frequent complications, having incidence rates of 2.5% and 7% respectively, but they are the most frequent causes of tracheostomy-related death. A tracheostomy tube may become obstructed by mucous, blood clots, the surrounding soft tissues, or impingement of the tube against the tracheal wall. Inability to clear the tube with a suction catheter necessitates changing of the inner cannula. Should ventilation remain inadequate, the entire tracheostomy tube may be replaced ^[7, 40-42].

Primary cannula malpositioning occurs when a cannula is improperly placed intraoperatively or after a replacement. Translaryngeal intubation must be performed if the tract cannot be immediately reestablished [40, 41]. Confirmation of proper tube placement with an X-ray postoperatively is especially important in children because pneumothorax in this context is the most frequently fatal post-pediatric tracheostomy complication ^[42]. The incidence of tracheostomy tube dislodgement is affected by a number of factors inclusive of tube length, body habitus and neck thickness of the patient, the presence of edema, and the method used to secure the tube. Tracheostomy tubes should be tied tight enough to allow only a finger between the tie and the neck and should be sutured to the patient's peristomal skin. The need for frequent repositioning of the patient also increases the risk of dislodgement ^[40, 41]

Tracheoesophageal Fistula

Tracheoesophageal fistula is usually an iatrogenic complication due to erosion of the posterior tracheal wall by the tracheostomy cuff. The presence of a nasogastric tube will predispose to its development. It is rare (1% incidence) but often leads to aspiration pneumonia with an 80% mortality rate. The presence of a fistula should be suspected when a patient coughs while swallowing saliva. A history of aspiration or pneumonia may be suggestive. An endoscopic evaluation is necessary should this diagnosis be suspected. Upon confirmation, definitive surgical repair is mandatory ^[40, 41].

Conclusion

Ear, nose, and throat surgeries are unlike other general surgical procedures. Most of these surgeries occur in close proximity to the airway affecting the patient's ability to breathe effectively postoperatively. Additionally, the increased vascularity of tissues in the otolaryngologic area creates not only a bleeding risk, but also increases airway edema with potential airway obstruction. Lastly, many otolaryngologic procedures involve vital endocrine organs that if altered can have substantial systemic effects. Due to these and other concerns, the postoperative care of otolaryngologic patients warrants knowledge of possible sequelae, vigilance in recognition, and prompt treatment if a morbidity should occur in the PACU.

References

- Morgan GE, Mikhail MS, Murra MJ. Clinical anesthesiology, vol. xiv. 4th ed. New York: Lange Medical Books/McGraw Hill Medical Pub. Division; 2006. p. 1105.
- Pritchard V, Eckard JM. Standards of nursing care in the postanesthesia care unit. J Post Anesth Nurs. 1990;5(3):163–7.
- Stoelting RK, Miller RD. Basics of anesthesia, vol. xii. 5th ed. Philadelphia: Churchill Livingstone; 2007. p. 697.
- American Society of Anesthesiologists Task Force. Practice guidelines for postanesthetic care: a report by the American Society of Anesthesiologists Task Force on Postanesthetic Care. Anesthesiology. 2002;96(3):742–52.
- Aldrete JA. The post-anesthesia recovery score revisited. J Clin Anesth. 1995;7(1):89–91.
- White PF, Song D. New criteria for fast-tracking after outpatient anesthesia: a comparison with the modified Aldrete's scoring system. Anesth Analg. 1999;88(5):1069–72.
- Lalwani A. Current diagnosis and treatment in otolaryngology: head and neck surgery, vol. xix. New York: Lange Medical Books/ McGraw Hill; 2004. p. 1056.
- Steele SM, Nielsen KC, Klein SM. The ambulatory anesthesia and perioperative analgesia, vol. xxi. New York: McGraw-Hill/Medical Pub. Division; 2005. p. 580.
- Darrow DH, Siemens C. Indications for tonsillectomy and adenoidectomy. Laryngoscope. 2002;112(8 Pt 2 Suppl 100):6–10.
- Smith SL, Pereira KD. Tonsillectomy in children: indications, diagnosis and complications. ORL J Otorhinolaryngol Relat Spec. 2007;69(6):336–9.
- Bhattacharyya N. Ambulatory pediatric otolaryngologic procedures in the United States: characteristics and perioperative safety. Laryngoscope. 2010;120(4):821–5.
- Miller RD. Miller's anesthesia, 2 vols. 6th ed. New York: Elsevier/ Churchill Livingstone. 2005; xviii, 3203 pp.
- Brigger MT, Brietzke SE. Outpatient tonsillectomy in children: a systematic review. Otolaryngol Head Neck Surg. 2006; 135(1):1–7.
- Windfuhr JP, Schloendorff G, Baburi D, et al. Serious posttonsillectomy hemorrhage with and without lethal outcome in children and adolescents. Int J Pediatr Otorhinolaryngol. 2008;72(7): 1029–40.
- Frost EAM, Goldiner PL. Postanesthetic care, vol. xiii. Norwalk Conn: Appleton and Lange; 1990. p. 318.
- Czarnetzki C, Elia N, Lysakowski C, et al. Dexamethasone and risk of nausea and vomiting and postoperative bleeding after tonsillectomy in children: a randomized trial. JAMA. 2008;300(22): 2621–30.
- 17. Fujii Y. Current management of vomiting after tonsillectomy in children. Curr Drug Saf. 2009;4(1):62–73.
- Cohen D, Dor M. Morbidity and mortality of post-tonsillectomy bleeding: analysis of cases. J Laryngol Otol. 2008;122(1):88–92.
- Marret E, Flahault A, Samama CM, et al. Effects of postoperative, nonsteroidal, antiinflammatory drugs on bleeding risk after tonsillectomy: meta-analysis of randomized, controlled trials. Anesthesiology. 2003;98(6):1497–502.

- Pavlin DJ, Chen C, Penaloza DA, et al. Pain as a factor complicating recovery and discharge after ambulatory surgery. Anesth Analg. 2002;95(3):627–34.
- 21. Bolton CM, Myles PS, Nolan T, et al. Prophylaxis of postoperative vomiting in children undergoing tonsillectomy: a systematic review and meta-analysis. Br J Anaesth. 2006;97(5):593–604.
- Afman CE, Welge JA, Steward DL. Steroids for post-tonsillectomy pain reduction: meta-analysis of randomized controlled trials. Otolaryngol Head Neck Surg. 2006;134(2):181–6.
- Gupta A, Wu CL, Elkassabany N, et al. Does the routine prophylactic use of antiemetics affect the incidence of postdischarge nausea and vomiting following ambulatory surgery?: a systematic review of randomized controlled trials. Anesthesiology. 2003;99(2): 488–95.
- Habib AS, White WD, Eubanks S, et al. A randomized comparison of a multimodal management strategy versus combination antiemetics for the prevention of postoperative nausea and vomiting. Anesth Analg. 2004;99(1):77–81.
- Fewins J, Simpson CB, Miller FR. Complications of thyroid and parathyroid surgery. Otolaryngol Clin North Am. 2003;36(1): 189–206.
- Cernea CR, Brandao LG, Hojaij FC, et al. How to minimize complications in thyroid surgery? Auris Nasus Larynx. 2001;37(1):1–5.
- Efremidou EI, Papageorgiou MS, Liratzopoulos N, et al. The efficacy and safety of total thyroidectomy in the management of benign thyroid disease: a review of 932 cases. Can J Surg. 2009;52(1):39–44.
- Herranz-Gonzalez J, Gavilan J, Matinez-Vidal J, et al. Complications following thyroid surgery. Arch Otolaryngol Head Neck Surg. 1991;117(5):516–8.
- 29. Benumof J. Airway management: principles and practice, vol. xv. St. Louis: Mosby; 1996. p. 974.

- Rubin AD, Sataloff RT. Vocal fold paresis and paralysis. Otolaryngol Clin North Am. 2007;40(5):1109–31.
- Mittendorf EA, Merlino JI, McHenry CR. Post-parathyroidectomy hypocalcemia: incidence, risk factors, and management. Am Surg. 2004;70(2):114–9.
- Carty SE. Prevention and management of complications in parathyroid surgery. Otolaryngol Clin North Am. 2004;37(4): 897–907.
- Kearns AE, Thompson GB. Medical and surgical management of hyperparathyroidism. Mayo Clin Proc. 2002;77(1):87–91.
- Cheah WK, Arici C, Ituarte PH, et al. Complications of neck dissection for thyroid cancer. World J Surg. 2002;26(8):1013–6.
- 35. Shaha AR. Complications of neck dissection for thyroid cancer. Ann Surg Oncol. 2008;15(2):397–9.
- Chummun S, McLean NR, Ragbir M. Surgical education: neck dissection. Br J Plast Surg. 2004;57(7):610–23.
- Aiken RD. Neurologic complications of head and neck cancers. Semin Oncol. 2006;33(3):348–51.
- Powitzky R, Vasan N, Krempl G, et al. Carotid blowout in patients with head and neck cancer. Ann Otol Rhinol Laryngol. 2010;119(7):476–84.
- Cohen J, Rad I. Contemporary management of carotid blowout. Curr Opin Otolaryngol Head Neck Surg. 2004;12(2):110–5.
- 40. De Leyn P, Bedert L, Delcroix M, et al. Tracheotomy: clinical review and guidelines. Eur J Cardiothorac Surg. 2007;32(3):412–21.
- Goldenberg D, Ari EG, Golz A, et al. Tracheotomy complications: a retrospective study of 1130 cases. Otolaryngol Head Neck Surg. 2000;123(4):495–500.
- Kremer B, Botos-Kremer AI, Eckel HE, et al. Indications, complications, and surgical techniques for pediatric tracheostomies–an update. J Pediatr Surg. 2002;37(11):1556–62.

Medico-Legal Considerations and Patient Safety

Elizabeth A.M. Frost

Introduction

A shared airway, presence of the elements of fire, risk of bleeding, likelihood of comorbidities such as obstructive sleep apnea, intraoperative position changes, long and complex surgeries combine to increase the incidence of perioperative complications during otolaryngology procedures. Not uncommonly, these complications may be interpreted as malpractice and the case becomes entangled in the medicolegal system. Understanding this system and developing strategies to avoid poor or even catastrophic outcomes are essential to the entire otolaryngology team.

An Overview

Approximately 60,000 medical malpractice claims are ongoing at any one time in the USA, a number affecting about 10% of the physician population. The average doctor is sued at the rate of 8–12% per year. Approximately 3% of malpractice claims relate to anesthesia. The commonest malpractice claims made against anesthesiologists are:

- Dental damage
- Death
- Nerve damage
- Brain damage
- Tissue injury
- Surgical complications
- Vision loss
- Burns
- Infection
- Retained instruments

Claims for brain death related to anesthesia decreased between 1975 and 2000^[1]. Indeed over 20 years, anesthesia-

related deaths have dropped from 1:10,000 anesthetics delivered to 1:400,000 for outpatient procedures. However, the number of malpractice claims continues to rise at a rate of 4% annually. Payouts and claims made vary widely according to region and practice. For example, over a 10-year period the overall incidence of malpractice claims against the Department of Anesthesia at the University of Chicago, an academic center, indicated an occurrence rate of 0.038%. While national data pointed to airway issues being more common, this study indicated that 23% cases involved regional techniques and 17% were dental injuries compared to 6% concerning airway problems ^[2]. Other studies have shown a higher incidence of severity in nonoperating room anesthesia claims and more substandard care than is seen in operating rooms where the commonest mechanism of injury was oxygenation/ventilation errors [3]. Claims associated with monitored anesthetic care tended to involve older and sicker patients with oversedation leading to respiratory depression, most commonly during elective eye surgery (21%) or facial plastic surgery (26%)^[4]. More than 40% of claims involved death or permanent brain damage. Anesthesia malpractice is currently the 12th highest medical specialty when it comes to the percentage of physicians that has paid claims for malpractice. Systems errors (related to equipment, etc.) contributed to 30% of settled claims between 2004 and 2006 ^[1]. In departments using anesthesia information systems (AIS), out of 41 cases filed, 30 were dropped and 11 went to settlement or litigation ^[5]. Of 21 respondents, 24 viewed AIS as valuable or essential for risk management.

The Medico-Legal System

While the overall injury rate in hospitals is about 4%, only 1:8 patients file a claim ^[6]. Should an adverse event occur, a patient might seek advice from an attorney. The lawyer will then consider the case and may immediately decide not to pursue it. Or he/she may seek the advice of an "expert" by sharing hospital records. This individual may opine that the standard of care was met (in which case the case may be

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discontinued unless the lawyer chooses to seek other opinions), or point to errors resulting in the damage implicating the anesthetic care team or other health-care workers. The "expert" may write an opinion letter (differs by state) and a claim is filed. If intent to sue is filed, then an immediate action is indicated or a lawsuit will almost certainly result. After a suit is filed, discovery begins in earnest, more extensive records are obtained, going back for many years, and the depositions of many individuals sought, including anesthetic care providers (everyone involved in the cases, even those relieving for only a few minutes) surgeons, any physicians listed as part of the patient's care, nurses, family members, actuaries, and usually the patient if he/she is still alive. Summaries are prepared. The insurance carrier may elect to settle the case if it appears to be financially advantageous. As such action means that the anesthesiologist will be reported to the National Practitioner Data Bank, the decision may be appealed. After distillations have been made a judge becomes involved. Mediation is attempted and the case may be dismissed (summary judgment), settled or go for trial.

It is important to realize that the medico-legal system is a business, influenced by societal norms and pressures. Attorneys work to obtain a favorable outcome for their clients. Experts on both sides are paid (on average \$250–600 per hour): the plaintiff attorney is usually paid only if the case is settled (customarily 1/3 of the settlement plus all expenses) or if the client is wealthy with independent funds and the defense attorney is paid in any case by the insurance carrier. In almost identical cases, the verdict may be diametrically opposed.

The ASA Closed Claims Project

Begun in 1985 under the auspices of the Committee on Professional Liability, the ASA closed claims project is a study of malpractice claims. Two basic aspects: damaging events and adverse outcomes characterize claims. The damaging event is the specific incident (e.g., loss of airway) that leads to an adverse outcome or injury (e.g., hypoxic encephalopathy). There are more adverse outcomes than damaging events, as the latter may not be identified. The project indicated that three adverse outcomes constitute 58% of claims paid out; namely death (29%), nerve damage (19%), and permanent brain injury (10%) ^[7]. Payment for permanent brain injury is the highest, ranging from \$7,650 to \$46,400,000.

The project has three associated registries, established in response to recurring claims and in an attempt to identify common causes that might be eliminated and thus prevent damage. They are:

 Pediatric perioperative cardiac arrest and death (POCA) registry (from 1994 to 2005, a total of 373 cases of anesthesia-related cardiac arrests were reported in children).

- 2. Postoperative visual loss (POVL) registry (174 cases reported to date).
- 3. Anesthesia awareness registry (196 potential subjects contacted the registry and 41 medical records collected).

As of December 2008, there are 8,954 claims in the database. Eighty-two ASA members are on the active reviewer list and 20–25 ASA reviewers travel a total of 50–60 days annually reviewing files. Twenty-two insurance carriers who insure 13,000 anesthesiologists (there are 40,000 ASA members) participate. Most cases involve healthy adults undergoing non-emergency surgery under general anesthesia (93% age>16, 78% non-emergency, 64% ASA 1 or 2, 63% general anesthesia, 59% female)^[7].

At the time the project was initiated, professional liability insurance was high (average \$36, 224) and often difficult to obtain ^[6, 7]. The intention of the closed claims project was to identify causes and thereby reduce the insurance problem for anesthesiologists. As of 2009, average insurance premiums for anesthesiologists are \$21,480 with the lowest rates of around \$7,000 in states including MN, SD, WI, and NE.

The project consists of an in-depth investigation of closed insurance claims resulting from anesthetic mishaps. Claims for dental injury, a very common and in most cases minor injury, are excluded. Also, cases in which the sequence of events and/or nature of injury cannot be reconstructed are excluded. Thus, in most cases, files are collected from mishaps resulting in lawsuits as files in these cases contain the most extensive information. The database also contains a narrative summary of each case describing the sequence of events and adding pertinent information. The closed claims project is located at the Department of Anesthesiology and Pain Management, University of Washington, Seattle.

Otolaryngology Cases

Mishaps in otolaryngology cases may result in loss of the airway and hypoxia. Many other cases in the database also list airway and hypoxia as crucial events. Thus, it is difficult to identify cases where the mishap related directly to the particular type of surgery. Nevertheless, certain hazards are unique to surgeries involving the head and neck. Most importantly, they include:

- 1. Problems associated with a shared airway
- Loss of the airway, postoperatively and related to intraoperative position change
- 3. Fire hazard
- 4. Bleeding of the airway, especially postoperatively
- 5. Complication related to comorbidities that may have prompted the surgery
- 6. Long and complex surgeries
- 7. Medication errors

The Shared Airway

Both otolaryngologists and anesthesiologists depend on access to the oral cavity during oral and laryngeal surgery. Space is limited and compromises must be made. Situations when problems may arise include:

- Compression of the endotracheal tube by a tongue speculum and/or retractor
- Disconnection of the endotracheal tube
- · Obstruction of the pharynx by packing
- Inadequate view of the operative site because of the presence of the endotracheal tube
- Excessive tongue or jaw pressure
- Positioning of the anesthesiologist at the side of the patient and draping making direct contact and visualization of the endotracheal tube impossible
- Extension of the head by the surgeon, causing the endotracheal tube to move up and out of the trachea.

Case

A 64-year-old man was scheduled for biopsy and excision of a lesion of the tongue. His past medical history was significant for smoking and hypertension. He had a relatively small mouth opening and a complete set of implanted teeth. General anesthesia was induced with fentanyl 50 mcg, midazolam 2 mg, propofol 150 mg, and succinylcholine 100 mg. Intubation of the trachea was successful. Bilateral breath sounds were confirmed with auscultation during manual bag ventilation. Shortly after induction, the anesthesiologist turned to complete the record as surgery commenced. Approximately 8 min later bradycardia was noted by both anesthesiologist and surgeon. At this point, the anesthesiologist realized that he had failed to begin automatic ventilation and the surgeon saw that the tongue depressor had not only obstructed the endotracheal tube but also caused disconnection of the tube from the breathing circuit. Alarms were not triggered earlier as the ventilator was not connected. The patient survived but suffered hypoxic brain injury.

Analysis

As with most cases, several factors combined to cause this complication. Failure to reconnect the ventilator on this older machine meant that the apnea alarm would not sound. The anesthesiologist was distracted to complete the record and did not observe the flattening of the capnograph wave. The small mouth and full set of teeth reduced the size of the oral cavity and excessive pressure was placed on the endotracheal tube causing it to move and become disconnected from the breathing circuit, secured to the table.

Prevention

Prevention as in many scenarios depends on vigilance and communication. It is easy to forget a crucial move such as turning the ventilator back on after checking correct tube placement. Had the tube not become obstructed and then disconnected it is possible that apneic oxygenation would have sustained the patient until the effects of succinylcholine had worn off. Had the breathing circuit not been so tightly attached to the table, it might not have become disconnected. Had the team communicated on the small size of the oral cavity, awareness might have been raised and less pressure applied to the endotracheal tube. (Liability, anesthesiologist, surgeon; settled pre trial).

Recently attention was drawn to four cases of necrosis of the jaw occurring shortly after general anesthesia with endotracheal intubation^[8]. In each case within a few hours or days, patients complained of mouth soreness that intensified. Examination revealed exposed bone of the posterior mandible and later sequestra formation. The authors concluded that the lesions were associated with localized oral trauma and/or pressure to the thin mucosa over the myohyoid ridge area during intubation and laryngoscopy causing periosteal damage, compromised blood supply and bone necrosis. They advised increased awareness of the complication and securing of the endotracheal tube in the midline position with better support of the breathing circuit to prevent gravitational pull. However, an editorial comment in the same journal advises against a rush to assume causality [9]. Rather, limited necrosis in this area is seen fairly commonly in dental and oral surgery practice and may occur "spontaneously" and without a recognized temporal relationship to surgery. The condition is associated with any mild trauma, viral infection, aphthous ulcers, radiation therapy, and medications that inhibit angiogenesis (bisphosphonates). Better clinical definition of cases after surgery or those labeled "spontaneous" and a clearer understanding of etiologies and strategies for prevention or management are required.

Fire and Burns

Burn injury continues as a significant cause of injury and a source of liability for the anesthesia care team. In otolaryngology procedures, the risk of causing burns is considerable, both from use of cautery (19% of fires, 27 claims made) and LASER (3 claims) ^[10, 11].

Case

A 74-year-old woman was scheduled for excision of a 2 cm basal cell carcinoma of her right cheek. She had been a heavy smoker for years and her room air saturation was 88%. A non-rebreathing mask was put over her nose and mouth and O_2 supplied at 8 l flow for 10 min by a nurse anesthetist. The area was cleaned with DuraPrep[®] and draped sterilely. Shortly thereafter, the O_2 was turned off and the surgery started. Almost immediately a pop was heard and flames were seen coming from under the drapes. The surgeon pulled the drapes off and stamped on them. The mask and O_2 tubing had ignited. The patient suffered extensive facial burns and died shortly thereafter from complications related to sepsis. There was no evidence of required fire training within either the departments of surgery, anesthesia or the hospital.

Analysis

There was no documentation of planning by the anesthesia care team in this case. Even though the oxygen was turned off as surgery commenced, there was insufficient time to dissipate the gas. Closed space oxygen build up, combined with restrictive draping and a combustible mask and use of a flammable prep solution made ideal conditions for a fire when cautery was used.

Prevention

A plan should have been in place to minimize the use of O_2 in this case. The patient was accustomed to a low ambient O_2 . Nasal cannula should have been in place and O_2 given as required to maintain adequate oxygenation SPO₂ around 90%. No draping was indicated. More time should have been allowed for the prepping solution to dry. Better communication with the surgeon was necessary before use of the cautery. An annual fire awareness program should be in place and adhered to closely. (Liability: the CRNA, supervising anesthesiologist, surgeon, and department of anesthesiology for failure to educate the CRNA, the hospital for failure to educate the prep nurse. Settled pre trial.)

The closed claims project of the ASA has concentrated on burn injuries and reported significant findings ^[12]. Just over 2% of the total claims in the database relate to burn injuries. Burns occur less in emergency cases and more in monitored anesthetic care (MAC) situations. Burn claims were less severe and payments were frequent but lower than in previous years. While deaths were uncommon, care was more likely to be judged as inappropriate when burns occurred. Fifty eight percent of burns were from warming devices (such as fluid and body heaters...). Thirty one percent were ignited by cautery (usually on the face). Injuries from LASER airway fires were most severe and had the highest payments. By 2004 the proportion of claims from cautery fires had increased since 1994 (56% vs 16%) of all burns (p<0.01), probably related to publication and dissemination of information regarding the hazards associated with the use of all warming devices. One death was reported following a LASER burn in the airway. Two airway fires resulted in permanent disabling injuries. Payment was made more often in burn claims (72%) than in other claims in the ASA database, although the payment was generally lower (usually surface burns from warming devices). Payment was made for 100% of airway fires, and these had the highest payment of all claims ^[11].

In 2008, the ASA presented a Practice Advisory for the Prevention and Management of Operating Room (OR) Fires ^[12]. Advisories are based on a synthesis of scientific literature and analysis of expert opinions, clinical feasibility data, and open forum commentary and consensus surveys. As such, they are not intended as mandates of absolute standards but rather convey general practice and recommendations that may be adopted, modified, or rejected. The advisory notes that while the incidence of operating room fires is difficult to determine, based in part on a lack of required national reporting, some estimates suggest that 50-200 fires occur annually in operating rooms in the USA. As many as 20% of incidents may result in serious injury or death. Some deaths may not occur immediately but may be due to resultant sepsis or multiple organ failure days to months later [13]. Fire requires three factors, the "fire triad":

- 1. An ignition source
- 2. An oxidizing agent and
- 3. Fuel

In the OR, the ignition source is the cautery or LASER and can also include heated probes, drills, and fiberoptic and light cables. Oxidizers include oxygen and nitrous oxide. An oxidizer-enriched atmosphere exists in closed or semi-closed breathing circuits and in the patient's airway or locally when the configuration of drapes traps oxygen. Fuel sources include endotracheal tubes, drapes, sponges, alcohol containing solutions, oxygen masks and cannulae and hair among several other materials. DuraPrep[®], a widely used alcohol-based prep solution has been associated with surgical fires, when it was not dried sufficiently, especially in hirsute patients ^[14].

Risk factors and prevention were explored recently by questionnaires sent to otolaryngologists ^[15]. Twenty five percent of the respondents had witnessed at least one fire on the OR. The electrocautery had ignited the fire in 59%, the LASER in 32%, the light cord in 7%, and the anesthesia

machine in 1%. Common fuels were the endotracheal tube (31%), drapes (18%), and a flash fire (no substrate identified, 11%). The highest risks occurred during endoscopic airway surgery, oropharyngeal surgery, and tracheostomy.

The report of the ASA made several recommendations:

- 1. All anesthesiologists should have fire safety education, specifically for OR fires with emphasis on the risk created in an oxidizer-enriched atmosphere.
- 2. OR fire drills and simulation training can improve staff response and drills should be conducted with the entire OR team during periods of dedicated educational time rather than during patient care.
- 3. To the extent that it is medically appropriate, several principles should apply including continuous collaboration with the procedure team to minimize the presence of an oxidizer-enriched atmosphere in proximity to an ignition source, configuration of drapes to decrease accumulation of oxygen and nitrous oxide, allowing adequate time for the drying of alcohol containing prepping solutions and moistening of sponges.
- 4. Management of OR fires include early recognition, halting the procedure, turning off fuel sources, attempting to extinguish the fire, following evacuation procedures, and providing post fire patient care. If a fire is not immediately extinguished, a CO₂ fire extinguisher should be used, the fire alarm activated and the patient evacuated.

In essence, the anesthesiologist should have a higher tolerance for a lower SPO_2 , using as low inspired oxygen as is compatible with patient safety, communication with the surgeon should precede use of electrocautery, and prepping solutions should be dried and the use of drapes minimized.

The optimal amount of oxygen and its delivery have been examined. At a flow of 21 per minute oxygen concentration exceeded 23% within only a few centimeters of a nasal cannula ^[16]. Concentration is directly related to the amount of flow, but rarely exceeded 26%. The authors recommended that electrocautery should be at least 10 cm from the oxygen source and O_2 flow should be ≤ 4 l. It is also important to realize that draping might easily displace the nasal cannula, thus altering the distance from the igniting source ^[17]. The Joint Commission recommended "As a general policy, use air or FiO_2 at or <30% for open delivery to prevent surgical fires." One may conclude that 100% oxygen should not be indiscriminately used, although anesthesia machines have a barbed outlet connector that delivers 100% O2. Titration of O₂ concentration is not possible unless a blend of air from the machine is added.

LASERs & LASER-Surgery

LASERs, an acronym for Light Amplification of Stimulated Emission of Radiation may also cause airway fires. In essence, a LASER directs a beam to a biological target. This action results in ionizing radiation in situ, mechanical shock waves and vaporization of tissues by heat. The beam acts both as a scalpel and to coagulate blood vessels. There are many types of LASERs, each with specific indications. Neodymiumdoped yttrium aluminum gradient (Nd-AG) LASER is the most powerful. It allows for a tissue penetration between 2–6 mm and is used for tissue debulking, particularly in the trachea, main-stem bronchi, and upper airway. The energy may be transmitted through a fiberoptic cable placed down the suction port of a fiberoptic bronchoscope. The Nd-YAG LASER can be used in "contact mode" to treat a tumor mass, such as a papilloma. Alternatively, the CO₂ LASER has very little tissue penetration and can be used where great precision is needed. One advantage of the CO LASER in airway surgery is that the beam is absorbed by water, so minimal heat is dispersed to surrounding tissues. It is primarily used for procedures in the oropharynx and in and around the vocal cords. The helium-neon LASER (He-Ne) produces an intense red light and can be used for aiming the CO₂ and Nd-YAG LASER. Because LASERs are capable of igniting airway fires, use of high concentrations of oxygen and nitrous oxide is dangerous. Some patients may not tolerate low concentrations of oxygen (at or just above room air) with resultant desaturation and hypoxemia. In addition, interruptions in ventilation frequently result in hypercarbia and may result in dysrhythmias. LASER surgery of the vocal cords requires the cords be immobile during LASER firing. Adequate muscle relaxation is therefore important. The CO₂ LASER is generally used because of its ability to precisely vaporize tissue. The Nd:YAG LASER coagulates deeper lesions and is used for tumor debulking. Airway management for LASER surgery of the larynx includes endotracheal intubation, intermittent apneic techniques, and jet ventilation. A small-diameter endotracheal tube (5.0-6.0 mm) allows for visualization of the vocal cords. The lowest possible FiO₂ (less than or equal to 0.3 or 0.4) that assures adequate

(continued)

oxygenation is desirable. Nitrous oxide and a high FiO_2 support combustion and should be avoided. Other precautions to prevent airway fires include filling the cuff with methylene blue and normal saline and using a special LASER endotracheal tube such as a Mallinckrodt LASER-Flex ®or Xomed LASER Shield[®]. It should be noted that LASER endotracheal tubes do not provide 100% protection for all LASER types. Several points apply to safety precautions during LASER ablation:

- 1. Factors contributing to the risk of airway fire during LASER surgery include energy level of the LASER, the gas environment of the airway, and the type of endotracheal tube
- 2. A safe gas mixture of 25–30% oxygen and avoidance of nitrous oxide decreases the risk of airway fire during LASER surgery. LASER-resistant endotracheal tubes are designed to prevent fires associated with LASER use. Endotracheal tubes made of polyvinyl chloride, silicone, and red rubber have oxygen flammability indices of 26%. Wrapping the endotracheal tube with reflective tape still imposes a hazard in that kinking of the tube may occur, gaps may be present, and non-LASER-resistant tape may be inadvertently used.
- 3. It is best to use an endotracheal tube that is designed to be resistant to a specific LASER (e.g., CO₂, Nd:YAG, Ar, Er:YAG, KTP). Observation of an indicator dye such as methylene blue from a tracheal cuff alerts the surgeon of contact with the endotracheal tube.
- 4. The anesthesiologist and all members of the operating room team should remain vigilant in recognizing the early signs of airway fire (i.e., unexpected flash, flame, smoke, odors, discolorations of the breathing circuit)
- 5. In the event of an airway fire, the flow of gases should be stopped and the endotracheal tube should be removed immediately followed by removal of burning materials and saline or water poured into the airway.

Of note is that elimination of flammable anesthetic agents has had little effect on OR fires except to change their etiology. Electrocautery, LASER use, oxygenenriched environments can ignite even fire resistant materials, including the patient, and fire triads are many ^[18].

Fluid Replacement

Free flap reconstruction has evolved over the past two decades in the treatment of defects of the head and neck due to recurrent cancers, radiotherapy, among other causes. These surgeries are often extensive and performed in patients with multiple comorbidities. Several risk factors for flap failure have been identified and further examined, including excessive intraoperative fluid resuscitation, concurrent medical conditions, and long duration of surgery ^[19–21]

Case

A 65-year-old male presented with recurrent laryngeal cancer. He had undergone a laryngectomy 2 years previously. He had a long-standing history of hypertension, left bundle branch block, and extensive tobacco use. A stress test indicated ischemic changes. A recent chest X-ray was indicative of pneumonia, likely related to recurrent aspiration. He was scheduled for a graft from the left arm to repair a pharyngoesophageal fistula and also a neck dissection. A verbal preoperative plan, allegedly by the surgeon and anesthesiologist emphasized fluid restriction as tolerated by the patient to minimize edema in the flap. Over the next 12 h, the patient received 1 L of crystalloid and 1 unit of blood. Urine output was 1 L. Vital signs were stable for approximately 8 h. At that point the blood pressure began to decrease and fluid boluses and a vasopressor were administered without improvement. The anesthesiologist recommended termination of the procedure and the surgeon agreed. In the Postanesthetic Care Unit (PACU), the patient developed complete heart block and a pacemaker was inserted. Massive fluid resuscitation was undertaken despite the finding of a central venous pressure of 10 useful. A few days later he sustained pulmonary emboli and died. Expert witness for the plaintiff argued that the patient was allowed to become severely hypovolemic and thus acidotic which caused the heart block and subsequent poor outcome. The surgeon later denied that she asked for fluid restriction, although it had been her practice to restrict fluids during free flap reconstruction in previous cases.

Analysis

The care rendered by the anesthesiologist could be supported by literature review. The patient was assessed as ASA 4 indicating severe disease, including long-standing heart and lung disease. Nursing documentation indicated that the anesthesiologist was present throughout but there was no formal note from the surgeon regarding preoperative decision-making. The chart was meticulously maintained.

Prevention

It is possible that this outcome could not have been prevented intraoperatively. The large fluid resuscitation postoperatively and failure to maintain anticoagulant prophylaxis may have been more contributory. The extensive pretrial hearings could have been shortened had documentation regarding conversations between the surgeon and the anesthesiologist regarding a fluid plan and extent of procedure been available. (Liability: case released before trial. No liability determined.)

Several factors have been identified by large-scale studies as contributory to poor outcome after major surgery, especially in high-risk patients. One of the most important has challenged our time honored practice of intraoperative fluid management and the existence of "a 3rd space" that must be taken into account. ^[22]. Even during long surgeries, large fluid replacement is not necessary and may result in free flap loss, postoperative acute respiratory distress syndrome and even multiple organ failure and increased mortality ^[22–24]. Rather, intraoperative fluid optimization may better be managed by using stroke volume variation or echocardiography. Both anesthesiologists and surgeons must be involved to ensure appropriate fluid management and application of optimal surgical techniques.

Pediatrics

A previous review of closed pediatric malpractice claims from the 1970 to 1980s indicated that 43% of injuries were associated with respiratory events, usually inadequate oxygenation with 89% deemed preventable ^[25]. With changes to monitoring standards, the hoped was that such errors would decline.

Case

A 15-year-old boy with a BMI of 47 underwent a tonsillectomy and adenoidectomy without apparent anesthetic difficulty. In the Postanesthetic Recovery Room (PACU), he was slow to awaken and was given naloxone. He responded immediately. He was discharged from anesthetic care after about 2 h. Monitoring was reduced to q 2 h. The ward bed was not ready and he remained in the PACU for a further 3 h. During transfer, the attendant noted he was sleepy and gave him oxygen. In the ward, the family remarked that he was difficult to arouse and was told that he had "had a hard day and needed rest." The pulse oximeter was alarming and was turned off. The family went home. Three hours later the patient was found unresponsive and in cardiac arrest. Reintubation was difficult and resuscitation delayed. He expired 2 days later.

Analysis

The family reported obstructive sleep apnea in retrospect. The anesthetic team noted sensitivity to narcotics. The nurses in the PACU failed to reassess the patient prior to discharge. The nurses on the ward did not question the cause of the sleepiness or apparent alarming of the oximeter, believing it to be machine malfunction.

Prevention

Attention to hospital policy and nursing protocols is essential. Alarmed monitors should not be disabled. A more comprehensive preoperative history and physical examination should have been obtained. (Liability: hospital at trial.)

About 8% of the current closed claims database contains claims for children (16 years and under), of which about 60% are age 3 or younger and sicker (ASA 3-5). One third of claims were associated with dental, ear, nose, and throat and maxillofacial procedures. Cardiovascular (26%) and respiratory (23%) events were most common in these claims ^[26]. Equipment and medication problems accounted for nearly one third of claims. From 2001 to 2003 there was a decrease in the proportion of claims for death and brain damage for inadequate ventilation/oxygenation, however, claims for death (41%) and brain damage (21%) remained the dominant injury in children. The major source of injury was cardiorespiratory events. Of the respiratory events, laryngospasm followed by aspiration, premature extubation, and inadequate ventilation were most common. Less common were malignant hyperthermia (MH) associated with halothane and/or succinylcholine, or medication errors. Of the aspiration events, the majority was associated with aspiration of adenoidal tissue or blood immediately after extubation. One case reported aspiration of blood after discharge. One emergency esophageal intubation postoperatively was not recognized when the capnography was not turned on and the flat line was presumed to be due to machine failure. The most common cardiovascular events included congenital cardiac anomalies (both known and unknown), inadequate fluid resuscitation and blood loss. Common equipment-related claims were due to burns from warming blankets or electrocautery. Medication related problems were mainly due to overdose.

Another analysis reviewed the characteristics and outcomes of claims after tonsillectomy. Data were drawn from two sources: Part 1, a private liability insurance company in New York State and Part 2, the public records of state and federal court trials including claims from 1979 to 2007. Awards were adjusted for inflation and reported in 2007 dollars. Most deaths were due to airway complication and one third were due to bleeding. In Part 1, cases were most commonly discontinued (44%) or settled (42%). Compensation was made in 48% of cases. In Part 2, death or major injury occurred in 52% with a mean award of \$404,000. Of cases reaching trial, the verdict supported the plaintiff in 60%. Awards against anesthesiologist were more frequent and higher than against surgeons (\$5 m vs \$840,000). Death occurred in 52% of court cases with a mean indemnity of \$3.8 m. Most of these cases were attributed to airway complications. In Part 1, minor or moderate injury was more likely to compensate but at lower sums (65%: \$3.8 m vs \$537,000). Only 28% of claims alleging death were discontinued. In Part 2, most cases were from California and Texas. Specific types of negligence included surgical misadventure (43%, usually bleeding), negligent postoperative care (32%), negligent anesthesia care (17%), lack of informed consent (14%), and failure to diagnose (3%). Two cases involved overdose of vasoconstrictor drugs. Of bleeding claims, only one was intraoperative and the rest occurred at home between postoperative days 0-14 (median day 5). Cases adjudged to be purely due to anesthesia-related complications did not dismiss the surgeon from the lawsuit. Of cases deemed to be anesthesia-related complications, 89% were likely to compensate the plaintiff and produce the highest awards. Cases of postoperative hemorrhage compensated only 50% with lower mean indemnity. The highest award was a jury verdict against an anesthesiologist and surgeon of \$22.5 m to a 7 years old in Texas who experienced periods of hypoxemia of undetermined cause during surgery that led to permanent brain injury.

Thus while awards and verdicts sometimes followed logical patterns, jury or anticipated jury response did not always result in a clearly understandable conclusion.

Comorbidities

Many comorbidities have special implications in disease processes that result in head and neck surgery. Obesity and hypertension are often associated with obstructive sleep apnea (OSA) in patients presenting for uvulopalatopharyngoplasty and children undergoing adenoidectomy. Comorbidity has also been demonstrated between seizures and sleep disorders . There is a higher prevalence of snoring and high-risk OSA in patients diagnosed with acute pulmonary embolism.^[27] Smoking causes obstructive lung disease and oral cancers. The patient with Parkinson's disease frequently has very poor dentition requiring reconstruction as may also occur in epilepsy sufferers maintained for years on dilantin. Asthma has been shown to be associated with nasal polyps requiring surgical treatment.

Case

A 24-year-old male presented for extensive dental reconstruction. Following anesthesia for a fractured arm at age 8 he was slow to awaken and suffered some neurologic deterioration. Some years later he underwent hernia surgery and again a similar pattern emerged. At this point he was severely disabled and unable to eat because of oral pain. Neither anesthesiologist nor surgeon was anxious to proceed given the history. The mother was insistent on surgery for dental rehabilitation and after several cancellations the case proceeded. The anesthesiologist used light inhalation anesthesia, avoiding succinvlcholine. Postoperatively the patient was hypertensive to 180/100 and febrile to 102 °F. Lactic acidosis was marked. He remained in a vegetative state. Plaintiff argued that the patient had sustained malignant hyperthermia (MH) or neurolept malignant syndrome (NMS), not treated by the anesthesiologist or surgeon. A geneticist for the defense diagnosed mitochondrial encephalopathy.

Analysis

Documentation and consultations from the physicians involved were clear and comprehensive. All attempts had been made to obtain earlier records. The anesthetic management met standards of care. Presentation was neither typical for MH nor for NMS. Mitochondrial encephalopathy and lactic acidosis syndrome (MELAS) is rare and the anesthetic implications are poorly understood.

Prevention

Given the paucity of information available about MELAS and the management of the case, it is difficult to imagine how this outcome could have been different. (Liability: none determined at trial, case dismissed.)

Of the comorbidities that present the most problems for anesthetic management, obstructive sleep apnea is probably the most important. A syndrome that is frequently a clinical rather than a laboratory diagnosis, the incidence appears to be increasing as the population becomes more obese. A high prevalence of OSA among patients with head and neck cancer, independent of the size of the lesion, has been shown. An increase in postoperative morbidity was demonstrated in at least one small study of patients with oral cancer and OSA. Implications of OSA for the anesthesiologist, especially as they relate to medication selection and postoperative care are many.

Medication Errors

Drug administration errors continue to be a major source of iatrogenic harm to hospitalized patients and may even be as high as 1:5 doses and account for 38% of drug-related errors.

Case

A 44-year-old woman was scheduled to undergo cosmetic surgery of the face combined with septoplasty under general endotracheal anesthesia. She had a history of hypertension and depression. The surgeon asked the scrub nurse for his standard solution of local anesthetic combined with epinephrine. Shortly after injection of about 25 ml, the anesthesiologist recorded blood pressure of 250/150, tachycardia to 150 beats/minute. Copious amounts of fluid were coming from the endotracheal tube. Diagnosing negative pressure pulmonary edema the anesthesiologist gave succinylcholine and furosemide, followed by a small dose of labetalol. The patient developed ventricular fibrillation and could not be resuscitated. On reviewing the medication injected, the nurse admitted that she did not know that the surgeon's standard solution contained 1:100,000 epinephrine and she had given him the drug undiluted (1:1,000). Neither the anesthesiologist nor the surgeon had checked the drug prior to injection.

Analysis

Failure to ensure that the nurse, who had never worked with this surgeon before, was aware of the protocol and familiar with epinephrine (she had only used it in cardiac arrest situations) was the primary event in causing the fatal outcome. The combination of hypertensive and antidepressant medications made the patient even more susceptible to adverse effects from epinephrine. The anesthesiologist then misdiagnosed the situation and time was lost when a potent vasodilator such as sodium nitroprusside could have been given.

Prevention

Communication with all members of the operating team is vital and adherence to existing protocols essential. It was the practice to check drugs and doses prior to injection by the surgeon, anesthesiologist and circulating nurse but this step was bypassed in this case. (Liability: department of nursing and the hospital, surgeon, anesthesiologist. Settled pre trial.)

Another case that settled quite recently against the anesthesiologist, nurse anesthetist, obstetrician and hospital involved a parturient in whom an epidural bag containing magnesium was hung instead of the intended local anesthetic solution. While not related to otolaryngology, the case underscores the fact that almost identical appearing pharmacy prepared infusions (100 ml bags) may be stored in the same place such as a refrigerator (antibiotics, local anesthetics, vasoactive solutions, paralytics, etc.)

The ASA closed claims project has reviewed medication errors. The proportion of data-based composed drug errors has remained constant over the past two to three decades at about 4%. Categories include:

- 1. Omission...drug not given
- 2. Repetition...extra dose of an intended drug
- 3. Substitution...incorrect or swapped drug
- 4. Insertion...drug not intended at that time
- 5. Incorrect dose
- 6. Incorrect route

Drug errors often result in serious problems including immediate and major physiologic effects in 47 of the cases in the database with 50 deaths (24%) and 70 cases of major morbidity (34%). Epinephrine was involved in 17 cases (8%), 11 of which resulted in death. Epinephrine was likely to be confused with other drugs such as ephedrine, oxytocin, or hydralazine.

Conclusion

Several factors come into consideration in reviewing risks of medico legal consequences of poor outcome after anesthesia in otolaryngology. The standard to which a defendant is held in medical malpractice is that of a "reasonable physician" dealing with a "reasonable patient". Has the practitioner met the standard of care for his/her community? Settling a case prior to trial is often economically more feasible for the insurance carrier but the settlement is reported to the National Practitioner Data Bank necessitating reporting to the hospital when the physician seeks privileges and may impact his/her insurance premiums. Plaintiffs typically seek money for economic losses including pain and suffering. Punitive damages for intentional misconduct are rarely awarded and generally are not covered by insurance. Informed consent requires more than simply signing a form but also should include history and physical examination and a clear understanding that the patient is aware of the risks, consequences, and alternatives for his/her care. And finally, above all, communication and documentation on the part of all the involved health-care workers are essential.

References

- Cheney FW, Posner KL, Lee LA, et al. Trends in anesthesia-related death and brain damage: a closed claims analysis. Anesthesiology. 2006;105(6):1081–6.
- Tung A, Roth S. Anesthesia malpractice in a litigious area: a closed claims study in an academic medical center. In: ASA abstracts A194, Atlanta, October 13, 2007.
- Robbertze R, Posner KL, Domino KB. Closed claims review of anesthesia for procedures outside the operating room. Curr Opin Anaesthesiol. 2006;19(4):436–42.
- Bhananaker SM, Posner KL, Cheney FW, et al. Injury and liability associated with monitored anesthesia care; a closed claims analysis. Anesthesiology. 2006;104(2):228–34.
- Feldman JM. Do anesthesia information systems increase malpractice errors? Results of a survey. Anaesth Analg. 2004;99(3):840–3.
- Caplan RA. Closed claims; medical mistakes. Audio Digest Anesthesiol. 2010;52(5).
- Domino KB, Caplan RA, Lee LA. ASA Closed Claims project and its registries. In: Value to patients and pocketbook ASA refresher course 222, San Diego; October 2010.
- Almazrooa SA, Chen K, Nascimben L, et al. Osteonecrosis of the mandible following laryngoscopy and endotracheal tube placement. Anaesth Analg. 2010;111:437–4.
- 9. Fisher QA. True, true -But how related? Bony necrosis and sequestration in the mandible after endotracheal intubation. Anaesth Analg. 2010;111:272–3.
- 10. Kressin KA, Posner KL, Lee LA, et al. Burn injury in the OR: a closed claims analysis. Anesthesiology. 2004;101:A-1282.
- Yardley IE, Donaldson LJ. Surgical fires, a clear and present danger. Surgeon. 2010;8(2):87–92.
- Practice advisory for the prevention and management of Operating room fires: a report by the American Society of Anesthesiologists task force on Operating Room Fires. Anesthesiology. 2008;108: 786–801.
- Weber SM, Hargunani CA, Wax MK. DuraPrep and the risk of fire during tracheostomy. Head Neck. 2006;28(7):649–52.
- Smith LP, Roy S. Operating room fires in otolaryngology: risk factors and prevention. Am J Otolaryngol. 2011;32(2):109–14.

- Orhan-Sunger M, Komatsu R, Sherman A, et al. Effect of cannula oxygen administration on oxygen concentration at facial and adjacent landmarks. Anaesthesia. 2009;64(5):521–6.
- Reyes RJ, Smith AA, Mascaro JR, et al. Supplemental oxygen: ensuring its safe delivery during facial surgery. Plast Reconstr Surg. 1995;95(5):924–8.
- 17. Lampotang S, Gravenstein N, Paulus DA, et al. Reducing the incidence of surgical fires: supplying the nasal cannulae with sub-100% O_2 gas mixtures from anesthesia machines. Anesth Analg. 2005;101(5):1407–12.
- Rinder CS. Fire safety in the operating room. Curr Opin Anaesthesiol. 2008;21(6):790–5.
- Pattani KM, Byrne P, Boahene K, et al. What makes a good flap go bad? A critical analysis of the literature of intraoperative factors related to free flap failure. Laryngoscope. 2010;120(4):717–23.
- Joo YH, Sun DI, Park JO, et al. Risk factors of free flap compromise in 247 cases of microvascular head and neck reconstruction: a single surgeon's experience. Eur Arch Otorhinolaryngol. 2020;267(10):1629–33.
- Joo YH, Sun DI, Park JO, et al. Factors predicting fistula following radial forearm free flap reconstruction for head and neck cancer. Oral Oncol. 2010;46(9):684–7.
- Chappell D, Jacob M, Hofmann-Kiefer K, et al. A rational approach to perioperative fluid management. Anesthesiology. 2008;109(4): 723–40.
- Namdar T, Bartscher T, Stollwerck PL, et al. Complete free flap loss after extensive hemodilution. Microsurgery. 2010;30(3): 214–7.
- Hughes CG, Weavind L, Banerjee A, et al. Intraoperative risk factors for acute respiratory distress syndrome in critically ill patients. Anaesth Analg. 2010;111(2):464–7.
- Benes J, Chytra I, Altmann P, et al. Intraoperative fluid optimization using stroke volume variation in high risk surgical patients: results of prospective randomized study. Crit Care. 2010; 14(3):R118.
- Jimenez N, Posner K, Cheney FW. An update on pediatric anesthesia liability: a closed claims analysis. Anes Analg. 2007;105:344–50.
- Epstein MD, Segal LN, Ibrahim SM, et al. Snoring and he risk of obstructive sleep apnea in patients with pulmonary embolism. Sleep. 2010;33(8):1069–74.

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