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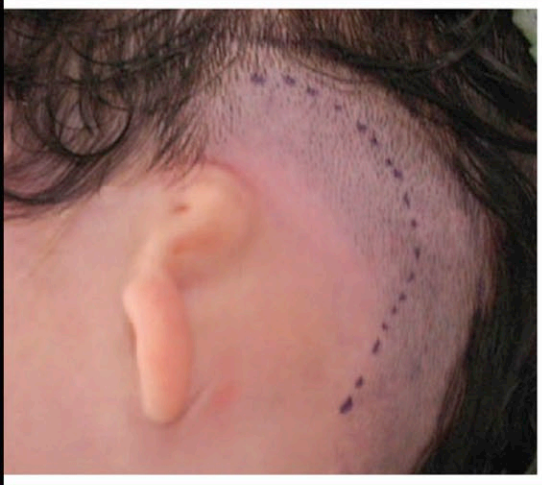
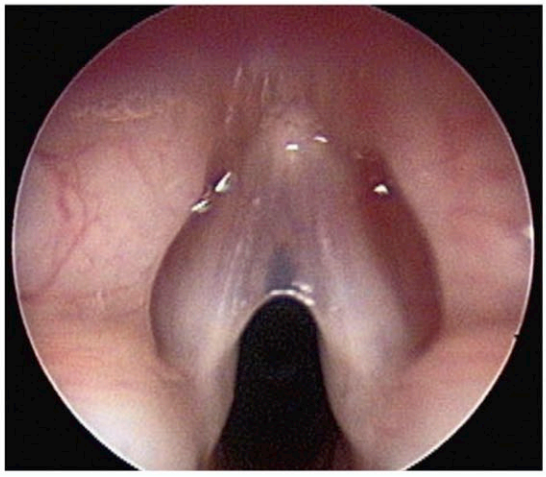
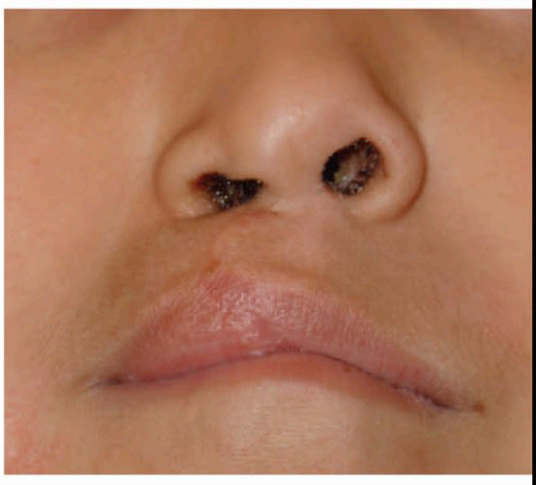

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Preface

For most otolaryngologists, the evaluation and management of children with otolaryngologic disorders constitute an important and often large part of their clinical practice. Yet, recent years have seen great changes in pediatric otolaryngology. To meet this need, we are very pleased to offer the first edition of *Cummings Pediatric Otolaryngology*.

It has been said that pediatric otolaryngology encompasses one third simple problems in healthy children, one third simple problems in children with comorbidities, and one third tertiary clinical problems occurring in healthy and medically complex children. Treatment advances have greatly improved survival for many childhood conditions such as early prematurity, cancer, and congenital heart disease, to name only a few. This text is focused on the latter two categories to assist the practicing otolaryngologist in keeping up to date on these essential topics. Reflecting the growing emphasis on multidisciplinary care of the complex pediatric otolaryngology patient, several chapters feature authors from multiple disciplines, including diagnosis and management of tracheal anomalies and tracheal stenosis, gastroesophageal reflux and laryngeal disease, aspiration and swallowing disorders, and pediatric head and neck malignancies.

This book also features a chapter on pediatric infectious disease, describing a practical approach for common clinical problems, as well as evaluation and management of the infant airway, a topic that is the sine qua non of pediatric otolaryngology.

With increasing recognition of the impact of pediatric sleep disorders on children's health, the chapter on pediatric obstructive sleep apnea is complemented by a chapter on pediatric sleep disorders to address the otolaryngologist's growing need to understand sleep disorders other than obstructive sleep apnea.

Our goal of this first edition of *Cummings Pediatric Otolaryngology* is to provide a lasting resource for students, residents, fellows, and practicing physicians alike and to reflect the dynamic and evolving field of pediatric otolaryngology.

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Contents

1 General Considerations in Pediatric Otolaryngology

J. Scott McMurray

Key Points

- Children are physiologically different from adults. Familiarity with these differences is required for safe management of the medical and surgical problems of children.
- Providing optimal pediatric care requires attention to the child as a patient and to the adults as guardians.
- Involving the child in play may alleviate anxiety regarding procedures in the physical examination.
- With practice, physical examination can be conducted in an expedient fashion and will be relatively painless for the child yet complete for the surgeon.
- Management of the pediatric otolaryngology patient often requires a multidisciplinary team approach.

Pediatric otolaryngology has developed over the years into a formal subspecialty of otolaryngology head and neck surgery, led by surgeons who had specific interests in the care of children. What makes pediatric otolaryngology distinct from its parent discipline of otolaryngology head and neck surgery are the special problems that present in children with an often unique approach to their management.

The specific problems that a pediatric otolaryngologist may encounter include airway disorders that present congenitally or iatrogenically, swallowing disorders that may change with growth and development, head and neck tumors in children and infants, hearing loss that may be congenital or acquired, and other congenital anomalies that may present in the head and neck (Figs. 1-1 through 1-6). As a subspecialty of otolaryngology head and neck surgery, the surgical techniques are not necessarily different, but the differential diagnosis, the approach to the child and parent, and the overall surgical management may diverge significantly. Not all enjoy treating both child and parent, but for those who do, pediatric otolaryngology is exceedingly rewarding. The specific problems that children may present with in the head and neck are often readily correctable, such that children may achieve their full potential as they grow and develop.

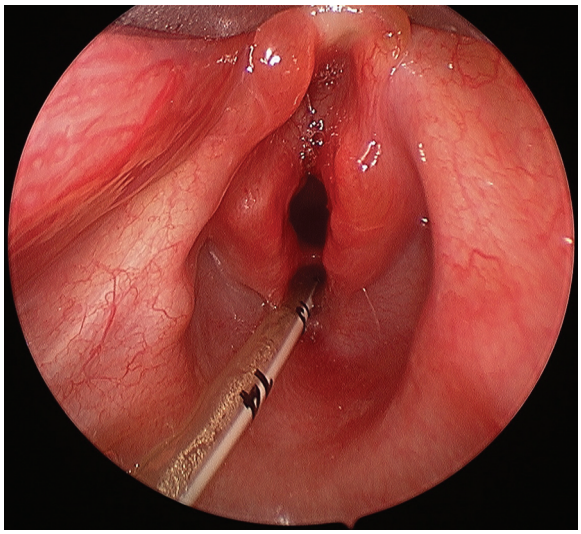

FIGURE 1-1. Telescopic operative laryngoscopy of an infant larynx with a laryngotracheal cleft. A feeding tube can be seen passing into the esophagus with the laryngeal cleft extending inferiorly through the cricoid to the cervical esophagus.

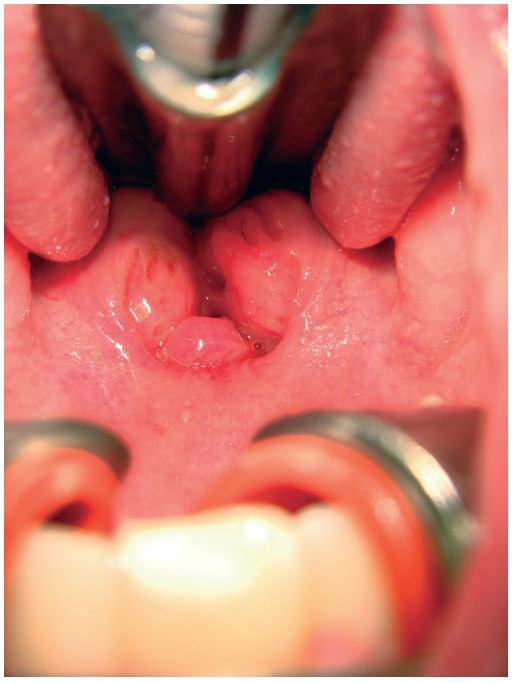

FIGURE 1-2. Oropharyngeal view during tonsillectomy of massive tonsils. These so-called kissing tonsils are causing complete obstruction of the oropharynx. This child had severe obstructive sleep apnea.

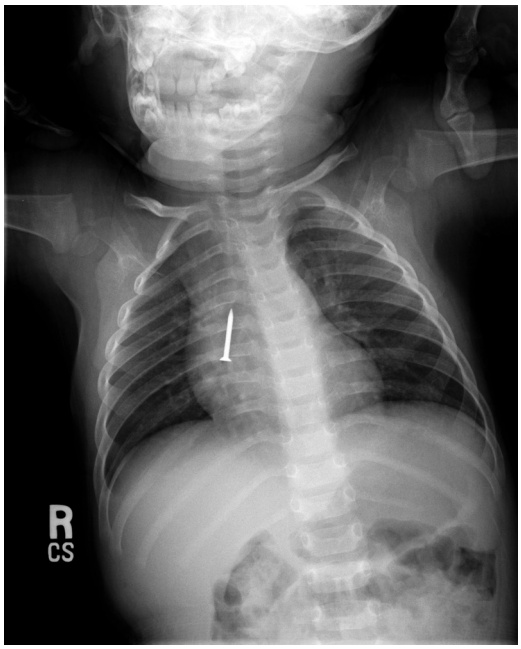

FIGURE 1-3. Chest radiograph of a young child with a radiopaque metallic nail in the right main bronchus.

For many practitioners, there is no greater reward than helping children afflicted with illness. Pediatric otolaryngology has developed as a subspecialty as a bridge between pediatric medicine and the surgical discipline of otolaryngology. Nearly every otolaryngologist will treat children during his or her professional career, and an estimated 25% to 50% of a general otolaryngologist's practice may be related to pediatric problems. Pediatric otolaryngology has therefore become an important part of all training programs in this field.

As the saying goes, children are not simply little adults. Often unique approaches are required for their evaluation, diagnosis, and management. Although the child is of prime concern, the parents—as the child's guardians, who must make decisions regarding the ultimate treatment options—also must be kept informed and must be approached with compassion. This is obvious to nearly everyone who has had to make medical decisions for a loved one, but it may not be as obvious to those

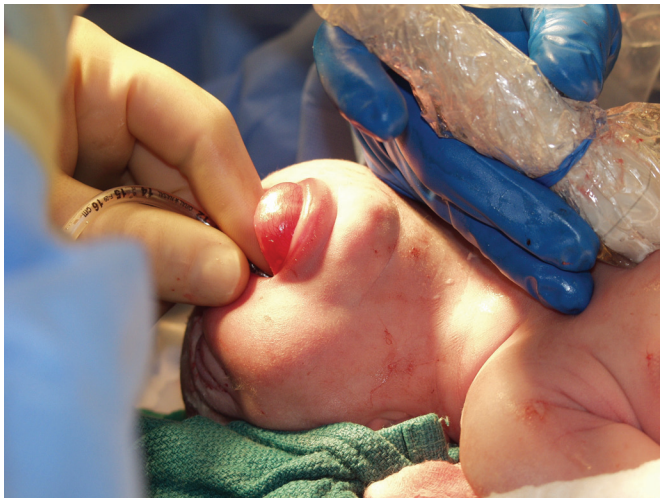

FIGURE 1-5. Intubated newborn with a large lingual cyst. This cyst was identified prenatally by fetal ultrasound, and the airway was secured with the aid of maternal fetal circulation as a bridge during an ex utero intrapartum treatment procedure. The cyst was a bronchogenic duplication cyst and was excised at the time of delivery to avoid tracheotomy.

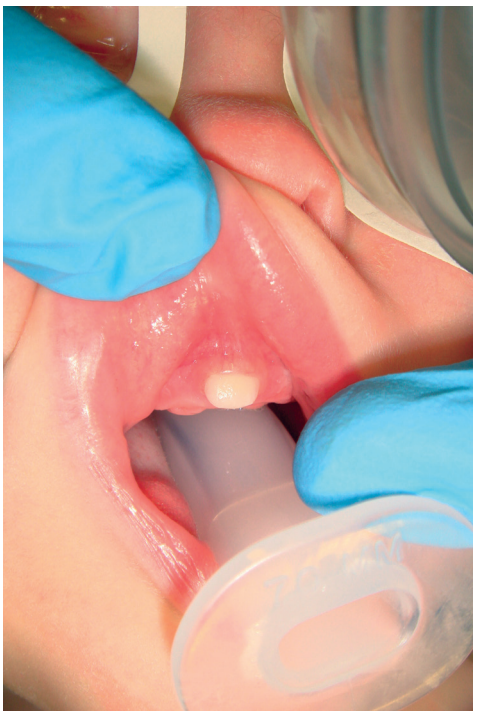

FIGURE 1-4. Photograph of the upper maxilla of a child with a single central mega-incisor, which is associated with piriform aperture stenosis. This child had respiratory distress as a newborn from a narrowed nasal airway at the piriform aperture, and the single central mega-incisor confirmed the diagnosis of piriform aperture stenosis.

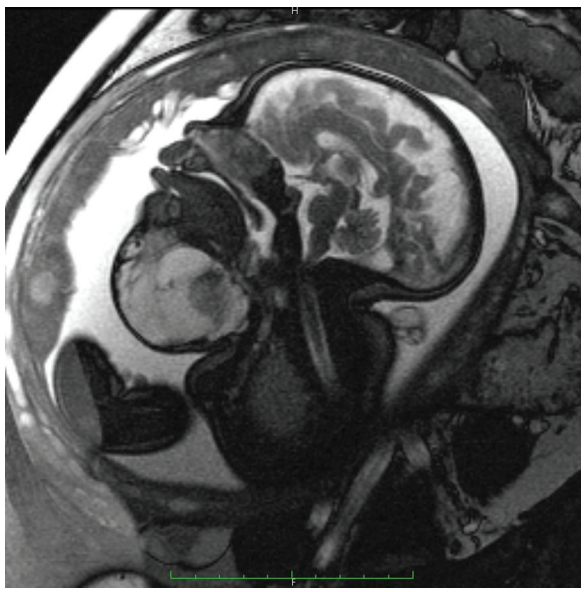

FIGURE 1-6. Fetal magnetic resonance imaging shows a large lymphatic malformation in the floor of the mouth and tongue. The head of the fetus is up with the nose pointing to the left. Planning for the management of the airway by a multidisciplinary team was required. The child was born by an ex utero intrapartum treatment (EXIT) procedure using maternal fetal circulation as a bridge to securing the airway. Although the child was successfully intubated during the EXIT procedure, a tracheotomy was inserted as the diffuse lymphatic malformation was found to also involve the supraglottis, which caused severe airway dysfunction.

who have not. The love for and emotional bond with a child may make even the most calm and rational person unable to comprehend events and medical explanations. It is up to the surgeon to monitor and modify his or her approach and explanations to both the child and the parent. This is not always an easy task, but it is one that is highly rewarding.

Smaller and more premature neonates are surviving, and sicker children are recovering from significant illnesses, as neonatal and pediatric intensive care units and the skills of the physicians who care for children continue to advance. This development has led to a demand for practitioners well versed in the management of disorders encountered in this population—namely, intubation trauma, bronchoesophageal processes, infectious disease, tumors, and congenital malformations. A multidisciplinary approach is often required. Agreement among the pediatric specialists and with the general pediatrician is important and represents a third layer of communication that is required for successful treatment of the child and family.

Physiologic differences among infants, children, and adults have occupied major research efforts and constitute the subject of complete textbooks. This chapter outlines basic differences relevant to the practice of otolaryngology along with some related considerations. In the following discussion, the material presented for each of the major body systems is intended as a general introduction to be used as a starting point for further investigation.

RESPIRATORY SYSTEM

CONTROL OF VENTILATION

Emergence from an underwater world, in which the child relied on placental exchange of gases and nutrition, induces tremendous changes in the neonate's physiology and anatomy. Some of these changes occur rapidly at birth, whereas others proceed more slowly. These significant changes can complicate interpretation of the neonate's problems. In particular, the assessment of the neonate's ventilation may be difficult. Interventions used to obtain measurements, such as placement of a facemask or insertion of a laryngotracheal tube, may induce significant changes in ventilation. Measurements of ventilation to assess respiratory drive also depend on the assumption that the respiratory muscles are capable of converting this drive into work—which is not always the case in infants and neonates.

Muscle fibers may be classified as *type I fibers,* which are slow twitch, highly oxidative, and fatigue resistant, or *type II fibers,* which are fast twitch and easily fatigable. Newborns have a paucity of type I muscle fibers but develop them shortly after birth. The muscle fibers in the diaphragm of a preterm infant are composed of less than 10% of type I fibers, whereas the muscle fibers of a full-term infant may be 30% type I fibers; the percentage of type I fibers increases to 55%, the expected adult level, during the first year of life. Preterm infants are more prone to respiratory fatigue, although this predilection disappears as they reach maturity.

Other subtle differences in sleep patterns also affect control of ventilation. Preterm infants spend as much as 50% to 60% of their sleep state in the rapid eye movement (REM) state. During REM sleep, the intercostal muscles are inhibited, as are most of the other skeletal muscles. This places a greater burden on the diaphragmatic activity. Much of this activity may be wasted because the chest wall can move paradoxically in the very young. This purposeless movement readily leads to hypoventilation, increased respiratory drive, and diaphragmatic fatigue.

Biochemical and reflex controls of ventilation are similar but incompletely developed in full-term neonates when compared with adults. Neonates have a higher basal metabolic rate than that in adults, reflected in higher ventilatory rates relative to body mass at any given partial pressure of carbon dioxide ($PaCO_2$). In term infants and adults, an increased $PaCO_2$ results in a proportionately similar increase in ventilation; however, this concordance in response is not seen in preterm infants. Compared with full-term infants and adults, preterm infants have a blunted response to increases in $PaCO_2$ and an altered response to changes in partial pressure of oxygen. The administration of 100% oxygen *decreases* ventilation in the very young, suggesting the existence of chemoreceptor activity not generally seen in adults.

Gestational age, postnatal age, body temperature, and sleep state modify the ventilatory response of newborns to hypoxia. Preterm and full-term infants less than a week old who are awake and euthermic usually demonstrate biphasic breathing patterns. They often experience tachypnea followed by hypoventilation. Hypothermic infants demonstrate a blunted response to hypoxia with respiratory depression without initial hyperpnea. The central effects of hypoxia on the respiratory center may cause depression of ventilation. Active peripheral chemoreceptors are unable to maintain a significant influence over this response. REM sleep also may decrease the response to hypoxia in these infants, whereas sleep states other than REM sleep are associated with an increase in the ventilatory response to hypoxia. Arousal from sleep during hypoxia is not seen in newborns but develops during the first few weeks of life with further maturation of the chemoreceptors that increases their ventilatory drive to hypoxia. Of interest, a decreased response to hypercarbia associated with hypoxia also occurs in newborns but not in older infants and adults.

Reflexes that arise from the lung and chest wall probably are more important in maintaining ventilation in newborns and in determining the respiratory tidal volume. Periodic breathing is characterized by periods of alternating rapid ventilation followed by apnea and is common in preterm and full-term infants. It is thought to result from dyscoordination of the feedback loops that control ventilation. During the apneic part of periodic breathing, the $PaCO_2$ may increase but changes in heart rate do not. Generally, no serious physiologic consequences are seen, and periodic breathing is considered normal and typically resolves by 6 years of age. Some preterm infants, however, may demonstrate serious and potentially life-threatening episodes of apnea. These episodes may last longer than 20 seconds and are accompanied by bradycardia. Apneic episodes may represent a failed response to hypoxia. Because these episodes are more commonly seen during REM sleep, ventilatory fatigue, as well as impaired chemoreceptor response to hypoxia, may be the cause. Usually, stimulation is all that is required to terminate the apneic event. Aminophylline treatment generally decreases the apneic episodes through central stimulation. Continuous positive airway pressure may also be helpful to decrease apneic episodes by modifying the lung and chest wall reflexes.

LARYNGOSPASM

The primary function of the larynx is to protect the lungs from aspiration. Hence, the laryngeal adductor response is a very strong reflex, such that it may be lethal in some instances. The robustness of the reflex has been shown to change with age and maturity in animal models. When laryngeal adduction is coupled with tachycardia, hypertension, and apnea, it is termed the *laryngeal chemoreflex* (LCR). The LCR can be induced by laryngeal exposure to acid, base, and pressure, but it is most sensitive to water and is ablated by saline. These properties of the LCR may have implications for sudden infant death syndrome (SIDS) because responses evolve with age to hypoxia, hypercarbia, and the laryngeal stimulation that causes the LCR,

which may explain the age pattern seen with SIDS. Deaths attributed to SIDS have dramatically decreased as a result of a change in the recommended sleep position of children—from prone to supine. Elimination of hypercarbia from rebreathing may be the mechanism behind the decrease in SIDS deaths. Hypercarbia is a known potentiator of the LCR. An increased awareness of and more aggressive stance toward the treatment of infant reflux also may have decreased a potential stimulus for the LCR. Decreased bolus size, increased feeding frequency, frequent burping, and positioning to reduce regurgitation have been helpful to decrease the incidence of reflux in infants and perhaps the incidence of apneic spells and infant breathing issues.

LUNG VOLUMES

In proportion to body size, the total lung capacity, functional residual capacity, and tidal volume are roughly equivalent in adults and in infants. In the full-term infant, the total lung volume is approximately 160 mL with functional residual capacity at one half this volume. Tidal volume is approximately 16 mL, and dead space is approximately 5 mL. Because of small lung volumes in infants, any increase in dead space is much more significant compared with adults. In contrast with static lung volumes, however, alveolar ventilation is proportionately much greater in newborns (100 to 150 mL/kg of body weight) than in adults (60 mL/kg of body weight). This higher alveolar ventilation in infants results in a higher alveolar–functional residual capacity ratio, 5:1, compared with 1.5:1 in adults. Consequently, the functional residual capacity is unable to provide the same buffer in infants, such that changes in the concentration of inspired gases are much more rapidly reflected in alveolar and arterial levels. This explains why induction of anesthesia using inhalational techniques is easier in infants than in adults. Along with the higher metabolic rate for body weight seen in infants, this difference also explains why infants have a smaller ventilatory reserve. The time from apnea to oxygen desaturation is much shorter in infants than in adults. Accordingly, surgical maneuvers that require short apneic periods may pose more of a challenge in infants than in adults.

The total surface area of the air-tissue interface of the alveoli in infants is small (2.8 m^2). When this relatively small gas exchange area is combined with the higher relative metabolic rate, infants have a reduced reserve capacity for gas exchange. This difference is of increased importance when congenital defects interfere with lung growth and development or if the lung parenchyma becomes damaged. The remaining healthy lung may not be adequate to sustain life.

RESPIRATORY RATE

The most efficient respiratory rate for newborns has been calculated to be around 37 breaths per minute. This rate is close to that observed for average newborns. Full-term infants are similar to adults in requiring approximately 1% of their metabolic energy to maintain ventilation. The cost of breathing is then 0.5 mL/0.5 L of ventilation. In preterm infants, this nearly doubles to 0.9 mL/0.5 L of ventilation. The cost of respiration also dramatically increases if the lung parenchyma is diseased or damaged by processes other than prematurity. In either case, the result is higher caloric and nutritional demands. Respiratory rate can directly affect the infant's ability to complete the suck-swallow-breathe cycle as well. If gas exchange is poor, ventilation rates may increase. This increase in respiratory rate may not allow adequate time for the suck-swallow portion of feeding. Caloric intake decreases precipitously as the infant expends energy toward breathing over feeding, which may lead to a vicious circle that results in failure to thrive.

TABLE 1-1. Arterial Oxygen Tension (Pa_{O_2}) in Healthy Infants and Children

Age	Pa_{O_2} in Room Air (mm Hg)
0 to 1 week	70
1 to 10 months	85
4 to 8 years	90
12 to 16 years	96

VENTILATION-PERFUSION RELATIONSHIPS

Ventilation and perfusion are imperfectly matched in the neonatal lungs. Some persistent anatomic shunts in the newborn circulatory system, as well as a relatively high closing volume in the lungs, cause this mismatch. The normal arterial oxygen tension in a newborn breathing room air is 50 mm Hg. This increases dramatically during the first 24 hours of life with changes in fetal circulation and maturation of lung parenchyma, and it continues to change slowly during the ensuing months and years (Table 1-1).

CARDIOVASCULAR SYSTEM

NEWBORN HEART AND CARDIAC OUTPUT

The heart of a healthy neonate is much different from that of an adult. The thickness of the right ventricle exceeds that of the left, as seen by the normal right axis deviation on the neonatal electrocardiogram. Shortly after birth, with closure of the fetal circulation, the left ventricle enlarges disproportionately. By the age of 6 months, the adult right/left ventricular size ratio is established.

The newborn myocardium is also significantly different from the adult myocardium. The myocardium of a newborn contains fewer contractile fibers and more connective tissue. Consequently, neonatal ventricles are less compliant at rest and generate less tension during contraction. The usual Starling curves of contractility for adult cardiovascular physiology do not hold true for the neonatal heart. Cardiac output is rate dependent in the neonatal heart, which has reduced compliance and contractility; the low compliance of the relaxed ventricle limits the size of the stroke volume, and therefore increases in preload are not as important in neonatal physiology as is heart rate. Bradycardia invariably equates with reduced cardiac output because the infant heart cannot achieve the increased contractility needed to maintain cardiac output. This distinction is extremely important to recognize during surgical and anesthetic procedures, which may induce bradycardia. Autonomic innervation also is incomplete in the neonatal heart, with its relative lack of sympathetic elements; this relative underdevelopment may further compromise the ability of the less contractile neonatal myocardium to respond to stress.

Heart rate is crucially important in the very young. The normal range for the newborn is 100 to 170 beats per minute, and the rhythm is regular. As the child grows, the heart rate decreases (Table 1-2). Sinus arrhythmia is common in children, but all other irregular rhythms should be considered abnormal. The average newborn systolic blood pressure is 60 mm Hg; the diastolic pressure is 35 mm Hg.

BLOOD VOLUME

Intravascular blood volumes vary tremendously during the immediate postnatal period, related to the amount of blood

TABLE 1-2. Normal Heart Rate for Children by Age

	Heart Rate (Beats/Min)	
Age	**Average**	**Range**
Newborn	120	100-170
1 to 11 months	120	80-160
2 years	120	80-160
4 years	100	80-120
6 years	100	75-115
10 years	90	70-110

transferred from the placenta to the child. "Stripping" the cord after birth or delays in clamping the umbilical cord may increase the blood volume by more than 20%, which results in transient fluid overload and respiratory distress. Conversely, fetal hypoxia during labor causes vasoconstriction and a shift of blood to the placenta. Thus fetal hypoxia may lead to hypovolemia after birth.

Because the total blood volume of an infant is small, relatively minor surgical blood loss may be hemodynamically significant. It has been observed during exchange transfusions that withdrawal of blood parallels a decline in systolic blood pressure and cardiac output. This decline is reversible to normal parameters with replacement of the same blood volume that was removed. Changes in arterial blood pressure with normal heart rates are thus proportional to the degree of hypovolemia. A newborn's ability to adapt the intravascular volume to the available blood volume is limited because of less efficient control of capacitance vessels and to immature or ineffective baroreceptors.

In the infant, systolic arterial blood pressure is closely related to circulating blood volume. Blood pressure is then an excellent guide to the adequacy of blood or fluid replacement during anesthesia, a fact that has been confirmed by extensive clinical experience.

RESPONSE TO HYPOXIA

Because of the relatively high metabolic rate typical of neonates and the relatively low reserve for gas exchange as described earlier, hypoxemia can develop rapidly in neonates, in whom the first sign is bradycardia. During surgery, any unexplained episode of bradycardia should be initially treated with oxygen and increased ventilation. During hypoxemia, pulmonary vasoconstriction and hypertension occur more dramatically in the neonate than in the adult. With a patent foramen ovale and/or a persistent patent ductus arteriosus, the increase in pulmonary vascular resistance may favor a shift into fetal circulation with right-to-left shunting, which compounds the problem. Changes in cardiac output and systemic vascular resistance also differ from those in older children and adults. During hypoxemia in adults, the principal response is systemic vasodilation that, together with an increase in cardiac output, helps to maintain oxygen transport to the tissues. Fetuses and some neonates respond to hypoxemia with systemic vasoconstriction. In the fetus, hypoxemia shifts blood to the placenta to improve gas exchange and oxygenation. After birth, however, hypoxemia may lead to decreased cardiac output, thereby further limiting oxygen delivery and increasing cardiac work. In infants, early and pronounced bradycardia may result from myocardial hypoxia and acidosis.

Neonates exposed to hypoxemia suffer pulmonary and systemic vasoconstriction, decreased cardiac output, and bradycardia. Rapid recognition and intervention are necessary to prevent cardiopulmonary collapse, cardiac arrest, and death.

TABLE 1-3. Ideal Electrolyte Composition for Infants

	Na^+ (mEq/L)	K^+ (mEq/L)
Intracellular	10	150
Extracellular	140	4.5

BLOOD VOLUME AND OXYGEN TRANSPORT

Neonatal blood volume is approximately 80 mL/kg at term and 20% higher in preterm infants. The hematocrit is 60%, and the hemoglobin content is 18 g/100 mL, although these values vary by infant and depend on when the umbilical cord is clamped. Little change in these values occurs during the first week of life, after which the hemoglobin level declines. The hemoglobin level change occurs more rapidly in preterm infants.

Approximately 70% to 90% of the hemoglobin in a full-term infant is the fetal type. Fetal hemoglobin has a higher affinity for oxygen than that of adult hemoglobin. It combines with oxygen more readily but also releases oxygen less efficiently at the tissue level than does adult hemoglobin. The increase in hemoglobin content in neonates is required to overcome this higher affinity of fetal hemoglobin for oxygen. A concentration less than 12 g/100 mL constitutes anemia. Correction of anemia by blood transfusion is indicated if the infant requires oxygen or experiences apnea.

During the first weeks of life, the hematocrit drops as a result of early suppression of erythropoiesis; the fetal type of hemoglobin is replaced with the adult type of hemoglobin with more optimal oxygen-carrying capacity. This physiologic anemia reaches its nadir at 2 to 3 months with a hemoglobin content of 9 to 11 g/100 mL. Provided that nutrition is adequate, the hemoglobin level will then gradually rise over several weeks to 12 to 13 g/100 mL, which is maintained throughout childhood.

SPECIAL CONSIDERATIONS

FLUIDS AND FLUID MANAGEMENT

As in adults, preoperative, intraoperative, and postoperative fluid management is extremely important in children. Some of the physiologic differences outlined earlier make fluid administration even more critical. Because of their small intravascular volume (70 to 80 mL/kg), infants who experience small changes in fluid balance can easily become either dehydrated or overloaded with fluid. Extreme vigilance, early recognition, and tight control are required in managing fluid balance in children. Compartmentalization of total body water changes with age, but intracellular and extracellular electrolyte composition remains stable (Table 1-3). Maintenance fluid requirements for a child can be calculated by relatively simple formulas that vary according to metabolic and physical activity. The calculation of water loss per calorie is described in Table 1-4.

TABLE 1-4. Expected Fluid Losses in Children

System	Fluid Loss (mL/100 cal/day)
Sensible Losses	
Kidneys	55
Insensible Losses	
Lung	15
Skin	30
Total	100

TABLE 1-5. **Children's Maintenance Fluid Intake Calculated by Weight**

Weight	Estimated Fluid Intake
0 to 10 kg	4 mL/kg/hr
11 to 20 kg	40 mL/hr (2 mL/kg/hr)
>20 kg	60 mL/hr (1 mL/kg/hr)

The correspondence of necessary fluid intake proportional to weight is described in Table 1-5.

Complex fluid and electrolyte deficits are beyond the scope of this discussion. In most such cases, consultation with pediatric medical specialists is advised.

PAIN MANAGEMENT

The management of pain in infants and children has undergone tremendous advances in recent years. It had been commonly believed that infants and newborns did not perceive pain because of their immature nervous system and that they would not remember any pain that occurred. However, direct physiologic consequences have been observed in infants in response to pain. Changes in heart rate, blood pressure, and respiration rate have been documented in infants experiencing painful stimuli, and such changes can be physiologically and emotionally deleterious to the child.

The perception of pain depends on both sensory and emotional experiences that may be altered by various psychologic factors. These factors may be specific for each individual patient, based on his or her expectations and past experiences. Efforts to reduce stress, anxiety, and fear will help decrease the apprehension and perception of pain during procedures in the office or in the operating room. In patients of the appropriate age, relaxation techniques such as guided imagery, deep breathing, and hypnosis may diminish the emotional component of pain. An adequate and age-appropriate explanation of expectations also will reduce anxiety, increase cooperation, and decrease perceived pain. The caregiver or parent also should be coached regarding expectations because children often look to the psychological state of a parent for cues. An anxious parent often increases the anxiety of the child. Conversely, a calm and collected parent can help calm the child during uncomfortable procedures.

Nonnarcotic analgesics are helpful for pain management. Acetaminophen in doses that range from 10 to 15 mg/kg every 4 hours is useful. Other nonsteroidal antiinflammatory drugs also are excellent for pain management. They often inhibit platelet function, however, and should be used only at the discretion of the surgeon.

Narcotic analgesics are indicated for moderate to severe pain in all age groups. Optimal use requires consideration of the needs of the individual patient, and neonates require special observation during the administration of narcotics. As noted previously, ventilatory responses to hypoxia and hypercarbia are diminished in this age group. Narcotics may further decrease these responses to potentially life-threatening levels. The metabolism and half-life of narcotics are different in neonates than those in older children and adults, and the permeability of the blood-brain barrier also may be increased in neonates. However, the use of intravenous, intramuscular, and oral narcotics is safe in the appropriately monitored setting and with the proper dose. Unlike adults, who usually self-administer and therefore self-regulate narcotic analgesics, pediatric patients often rely on caregivers to administer pain medication, which can lead to underdosing or overdosing.

SEDATION

More often today, professional certification and credentialing for pediatric sedation are required at institutions that care for children. The development of sedation teams, often staffed by pediatric intensivists, has increased safety and monitoring for these patients. Otolaryngologists can participate in the team effort by assessing the airway of the patient referred for sedation. Occasionally, a general anesthetic procedure with a secured airway is required for the proper level of safety for a sedated patient. Although the otolaryngologist can assess airway safety, the professionals on the sedation team have the final word on whether they are willing to sedate a child. The requirements for certification and the makeup of the sedation team will vary by institution. To achieve a successful outcome, as measured by family satisfaction and patient safety, it is important to adhere to the guidelines of the institution and to work together as a team.

REFERRAL SOURCES

Unlike adults, most pediatric patients have a primary caregiver—the pediatrician or family practice physician, who refers patients for specialty evaluation for problems related to the ears, nose, and throat. Pediatric medicine is a specialty that legitimately considers its practitioners to be advocates for their patients. The child's primary care physician is responsible for his or her overall health and well-being, including preventive medicine such as vaccination. The primary care provider can serve as a tremendous ally to help identify and refer problems early on and to help translate the complex problems related to surgical management. Although every patient encounter requires an element of trust building and rapport development, a long-term relationship usually has already developed between the child's doctor and the family.

The culture of pediatric medicine and its interface with surgery differ somewhat from its counterpart in the typical adult world. Often a multidisciplinary approach is used in the perioperative period, which can enhance the care of the patient, as long as delineation of duties and responsibilities is clear. Open lines of communication are essential.

Although all parties involved must understand that the surgeon is ultimately responsible for the care of any patient undergoing surgery, a decision to proceed should always be accepted by the parents and by the referring physician. Occasionally, expectations for surgery are initiated before the evaluation by the otolaryngologist. In such cases, how a decision is made must be explained clearly and logically; although the surgeon makes the final recommendations for or against surgical management of a problem, all parties must clearly understand the steps that led to the logical decision.

Finally, pediatric otolaryngologists often direct their educational efforts directly to the families of the patients they treat. Education of their colleagues, specifically the primary care physicians who care for these patients, is also very important. Keeping these practitioners up to date through educational forums will maximize their ability to help with perioperative management, and it will increase the visibility of the otolaryngologist's contributions.

PATIENTS

Although the entire family is involved in the care of a child, it is important to focus on the child as the patient. Children experience the same fears of pain and discomfort as adults but may lack the maturity and skills to overcome these fears. Delayed gratification may not be a familiar concept to a child. Whereas adults often bring themselves to the doctor and

therefore have overcome the first hurdle toward obtaining medical care, children are brought by their parents and often have no idea why they are there. Establishing rapport with the child is important for myriad reasons: Not only will this put the child at ease, but it will often put the parents at ease, when they see that the physician has a genuine interest in the well-being of their child. Children take cues from their parents. If the parent is apprehensive, the child will often be as well. It is important to explain what is to be expected during the history and physical examination, not only to the parent but also to the child, on a level that the child can understand. Procedures that may be uncomfortable are best left to the end of the examination to be performed quickly but thoroughly.

Quickness and experience allow for a brief but relatively complete physical examination without undue stress on the child or the parent. Restraining an older child who may have been traumatized by a previous forceful examination is ill advised and could be dangerous. In these cases, sedation or a general anesthetic is appropriate to complete the required examination.

PARENTS

Parents can find themselves in an uncomfortable position of being required to make decisions for their sick child. It is important for the surgeon to be open and honest regarding the options and risks involved as well as potential discomfort. Although there may be a single best option, alternative treatments with respective risks and benefits, as well as expected outcomes, should be explained to the family. The reasons that a surgeon considers one option better than others should be shared, taking time to answer questions. Parents often have questions that may be biased by previous experiences, advice of family members or friends, or exposure to the media.

Often, families have been referred by the primary care physician, who usually has answered many of the relevant questions. Having been referred to a specialist, the parents often identify the otolaryngologist as a person with expertise and added ability. It is important not to be placed in the situation of persuading parents to proceed with surgery; if they continue to have difficulties deciding after all of their questions have been answered, it may be suggested that they contact their primary care physician for another view. Finally, it may be more comfortable for families to defer a decision regarding surgery until they can discuss it at home, with the understanding that they can call if more questions arise and that they will schedule an appointment if they wish to proceed.

CONGENITAL ANOMALIES

Children with developmental or hereditary abnormalities often require a team approach for optimal treatment. For example, children with craniofacial anomalies are optimally cared for by a craniofacial team that consists of at least an otolaryngologist, craniofacial surgeon, dentist, geneticist, audiologist, speech-language pathologist, and social worker. It is important that the participating specialists should speak in a unified voice because conflicting information will only confuse the parents, who are often overwhelmed and experience guilt regarding inheritable defects. Counseling should be available from specialists in genetics when needed.

The goal of caring for children with congenital anomalies or developmental delays is to maximize each child's individual potential. It is important to know what difficulties a child may encounter that result from a particular disorder and to offer advice or techniques proven to help families cope with or manage the disability, which will vary for each child, family, and situation.

Evaluation by a pediatric geneticist and access to genetic counseling is advised by the American College of Medical Genetics for all children with congenital anomalies. Genetics evaluation may be beneficial to diagnose syndromes, monitor for potential associated disorders, and carry out preventive care. The patient may be offered genetic testing, but the decision to pursue genetic testing is an individual one, and counseling should be provided to help interpret the results for the family. For children with multiple anomalies, genetics consultation may be performed at birth, with an opportunity provided for later follow-up as the parents desire. For other disorders such as nonsyndromic sensorineural hearing loss, genetic evaluation is also recommended for all children, but the optimal timing has yet to be determined.

OVERALL ASSESSMENT

HISTORY

In treating children, several sources are available for historic data. A referral letter from the primary care physician often will accompany the child. This aspect of the history is important, but it does not replace the history taken from the parents or the child. Historic data from the primary physician give the added advantage of allowing the specialist physician to verify that the information provided by others and the data gathered during the examination are in agreement. The parents usually are very astute regarding their child's behaviors or symptoms and should therefore be listened to carefully. The child can be questioned as well, although he or she may not understand or be able to identify a problem. Often, chronic symptoms such as velopharyngeal insufficiency may be life-long symptoms and are not seen as problems to the child because the child may never have experienced life in any other way. At times, the difficult part for the pediatric otolaryngologist is to synthesize sometimes conflicting data into a coherent story.

Occasionally, admission to the hospital for observation is required to allow for further collection of data. Symptoms described by the caregiver can be documented and perhaps better understood. Further testing also may document whether the child's safety is compromised because of the severity of symptoms or potential problems.

It also is very important to obtain a general family history in addition to a history of any diseases or illnesses that affect the child's siblings. Information regarding the pregnancy and delivery of the child also is important. Insight into any family stresses or school problems that may influence the child's psychosocial development is also helpful.

PHYSICAL EXAMINATION

Physical examination of a child should be done in a nonthreatening and calm manner. Rapport and trust are the key elements. Engaging the child as an active participant often leads to more cooperation. One technique is to let the child hold or examine the instruments before they are used, which often reduces the child's anxiety. Because many well-child examinations are accompanied by vaccinations, children may unfortunately often equate any visit to the doctor with a shot. The resulting apprehension and perhaps an attempt at maintaining independence or control may underlie seemingly irrational behavior during an examination.

The majority of the head and neck examination should be painless, and otoscopy generally is managed without difficulty; pneumatic otoscopy can be performed but should be explained before proceeding so as not to surprise the child. Explanations build a child's trust in the examining physician. The use of a head mirror or headlamp light during the examination is the surgeon's choice. Equipment that covers the examiner's face

often frightens children, and this can decrease cooperation. Anterior rhinoscopy can be performed with the otoscope and a larger speculum, thereby eliminating the need to introduce a new instrument to the child. Oral examination often can be accomplished without a tongue depressor, although occasionally use of a depressor may be required to view the posterior pharynx. Palpation of the neck can be accomplished without any significant discomfort to the child, and masses or tenderness can be documented.

During the examination, further observations of the child's facial features can be made, which includes assessment of the auricles for shape, pits, or fistulae and of the eyes, for symmetry in shape and alignment without apparent hypertelorism or heterochromia. The nasal airway should be assessed for chronic mouth breathing and to help identify any abnormality of the palate. Listening to the child's breathing for the sounds of stridor or stertor can help identify the level or site of potential airway obstruction. Auscultation of the neck and chest also generally is performed. The otolaryngologist will concentrate the physical examination on the particular area of interest, which is the head and neck; however, a general physical examination should be performed in a brief but complete manner.

EVALUATION OF THE EAR

MICROSCOPIC EXAMINATION OF THE EAR

Otomicroscopy is an invaluable tool for assessing the ears, although children may have anxiety about this portion of the examination. Often, allowing the child to look at the examiner's thumb through the microscope will show that the microscope is simply a tool to obtain a magnified view of the ear. The parent can assist in reminding the child to hold still for the examination while cerumen or debris is removed from the ear canal. With the parent reclined on the examination chair or table, the child may lie on the parent like a table. The parent can then hug the child, to hold the arms and body still, while an assistant helps steady the head. Use of a papoose board is possible in smaller children but often is not successful in older children. Sedation usually is not necessary but may be used in certain cases in accordance with the sedation policies of the institution. Occasionally, a general anesthetic is required to permit a complete and thorough examination.

Although usually not painful, suctioning is loud and can be frightening. The suction device is a very useful tool to quickly clean away debris and drainage, although some clinicians prefer to use cotton-tipped applicators instead. It is important to have a number of techniques in reserve and to use the one that will quickly and effectively accomplish the required task without undue stress and discomfort to the child.

FLEXIBLE ENDOSCOPY

Nasopharyngoscopy is an invaluable tool for the otolaryngologist. With advances in technology, smaller-caliber endoscopes are being developed with improving optics. Although mirror examination is useful, the fiberoptic endoscope allows for a dynamic examination of the nose, velum, pharynx, hypopharynx, and larynx. When combined with video recording, slow-motion replay is possible—an important option if only brief glimpses are obtained in a child who is not able to cooperate fully with the examination.

As in adults, topical anesthesia and nasal decongestion with lidocaine gel or tetracaine spray and oxymetazoline is very useful. Use of cocaine in young children is not advisable because of variable absorption rates. In infants, cocaine can produce unwanted irritability and nervousness. The parents should be counseled that after the topical anesthetic, the child should not eat or drink for approximately 30 minutes. Sedation generally is not necessary for this examination.

Complications from nasopharyngoscopy are rare, although an emergency cart for airway management and the skills to use what it contains are important. Nasal bleeding, if it occurs, often is self-limited or requires little intervention.

NEEDLE BIOPSY

Fine-needle aspiration (FNA) can be performed in children to obtain a biopsy specimen for histopathologic examination. FNA is useful in children, although many head and neck masses typical of children are very different from those encountered in adults and may be more difficult to characterize by FNA. Children usually are very frightened of needles or sharp objects. EMLA cream (Astra Pharmaceuticals, Westborough, MA) or other anesthetic creams that are topically applied can render the introduction of the needle painless.

The site is prepared in a sterile manner. A 22- or 23-gauge needle is attached to a 10-mL syringe and is then placed into the mass while suction is applied. The sample obtained within the needle core is then applied to a glass slide and is placed in a fixative. Optimally, the cytopathologist can attend or perform the procedure to provide immediate feedback regarding the adequacy of the specimen. If the specimen is inadequate and the child appears able to tolerate another attempt, FNA may be repeated using the same anesthetic. An adhesive bandage is applied when the procedure is done. FNA should be no more traumatic than obtaining a blood sample and should be explained in those terms to the parent.

AUDIOLOGY

Every child can be tested for hearing loss. Pediatric otolaryngologists can help to accomplish this goal by providing education, both for families and especially for primary care physicians. It has been shown that early detection and treatment for children with hearing loss leads to dramatic increase in language ability later in life. Audiologists with expertise in pediatric testing are generally able to evaluate children as young as 6 months of age with visual response audiometry, which in experienced hands can be very reliable. Other techniques for younger children or children who are unable to conform to the demands of the task include otoacoustic emissions and auditory brainstem response testing.

Early detection of hearing loss is extremely important to the speech and language development of the child. Currently, newborn hearing screening programs are in place nationwide, many of which use automated auditory brainstem response testing. If a screening test identifies the infant as requiring further investigation, it is essential for the otolaryngologist to provide any necessary referrals and follow-up evaluations. If no action is taken once a child has been diagnosed with hearing impairment, the test is useless and does not benefit the child or the family.

PREPARATION FOR HOSPITALIZATION AND SURGERY

Once the decision to proceed with hospitalization or surgery has been made, the child and parents should be readied emotionally to achieve a successful outcome. The details and expectations should be reviewed with the family by persons familiar with the process. Some institutions and most children's hospitals have programs to introduce the family to the hospital, operating room, and operating room procedures. A sense of

familiarity and an understanding of what to expect will be helpful to all involved. Children can then role play before hospital admission or surgery. With a clear understanding of what is expected, parents also are less apprehensive and will be able to help calm the child.

It is important to clarify arrival times and the nothing-by-mouth requirements to the family. Special films and booklets are valuable tools for education. The parents must be encouraged to be truthful, to the best of their ability, about preparation for the upcoming surgery. It also is important that the surgeon communicate directly with both the parents and the child so that all questions are answered completely.

HOSPITAL

A dedicated children's hospital is the preferred venue for pediatric otolaryngologic surgery because it provides a team of physicians, nurses, and other professionals who can best handle a hospital stay or surgical procedure for the pediatric patient. Children's hospitals constitute an important medical resource, and every surgeon involved in the care of children should support them. Unfortunately, not every community has one. In such instances, it is important to identify the best facilities in the community for children, in which the physicians, nurses, and support staff are capable of and enjoy caring for children. Ensuring an optimal environment for pediatric treatment will encourage the best outcomes and will lead to the greatest satisfaction among patients and their families.

The concept of a team of professionals who deal strictly with children is not new; moreover, a team approach is becoming increasingly accepted as the standard of care. When complications arise, as they inevitably do in the course of even the best medical care, having the support facilities and the personnel available to promptly handle any need is vital for the best outcome possible.

SELECTING ANESTHESIA

Pediatric anesthesia is a subspecialty in itself. Reaching a degree of proficiency to deliver an anesthetic to children of all ages requires considerable training and experience. The younger the patient, the more complex the problems encountered. As in all aspects of medicine, this subspecialty includes practitioners with special interests and expertise in particular fields. Those with expertise in safely delivering anesthesia to children should be sought when their assistance is required, and reliance on these specialists benefits both the patient and the surgeon.

With practice and special expertise, the interested clinician can devise techniques to safely and effectively perform nearly any technique. Local anesthesia as a primary anesthetic technique for pediatric surgery is possible but requires a cooperative child and an adept surgeon. Generally, local anesthetics are used for immediate postoperative pain management after surgery performed with the patient under general anesthesia. Young children generally do not understand the need for painful injections and have short attention spans, which makes it difficult for them to sit still for the procedure. The use of sedation for extended periods is not recommended, unless support personnel are available to monitor the child during the sedation process. Many institutions have strict guidelines and require a certification process for the use of conscious sedation in children, and most invasive or painful procedures require a general anesthetic.

POSTOPERATIVE MANAGEMENT

Probably the most important part of postoperative management is preoperative teaching. If the parent and the child are adequately prepared about the effects of surgery, they are more capable of handling the postoperative period in terms of decreased stress and optimal adherence to the postoperative regimen. Detailed teaching about the expected clinical course and unexpected possibilities is important. Providing written instructions, along with contact telephone numbers if the parents should have questions, also is very helpful. Knowing what to expect is the single most important factor in uneventful recovery from a surgical procedure without multiple phone calls and excessive concern from both patient and physician.

If a child must be hospitalized, it has been shown that the presence of a parent makes the stay less fearsome and stressful for the child. Whenever possible, parents should be encouraged to stay with the child while in the hospital. Arrangements should be made for this to be possible if it is not the norm at the hospital where the child will stay.

More often, short-stay and outpatient procedures are being performed. It is helpful for the child to return to the home setting as soon as possible. Safety is the main concern, however, and monitoring in the hospital should be done as long as necessary.

An awareness that modifications in surgical technique may be required in the management of pediatric problems is important. Dressing changes, suture removal, and postoperative manipulations should be kept to the required minimum so as not to provoke fear and discomfort in children who must undergo minor procedures. Sometimes sedation or a general anesthetic for packing or suture removal, not often required for adults, may be necessary in children. In addition, it is reasonable to plan ahead of time to use absorbable suture material and dressings that need infrequent changing.

A REWARDING EXPERIENCE

The care of children is an extremely rewarding experience. Children may become ill quickly but often recover just as quickly if set on the right pathway. Watching children grow and develop makes for lasting memories and personal satisfaction. For many clinicians, helping children with chronic illness to overcome disabilities and difficulties and watching them reach their full potential as they grow to adulthood have no match for reward.

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Developmental Anatomy

2

Eunice Y. Chen | Kathleen C.Y. Sie

Key Points

- Head and neck anatomy is divided into fascial layers and triangles or levels, which serve as organizational units to manage the volume of anatomic detail in the neck.
- An understanding of the fascial relationships in the neck is important, not only because of the boundary relationships but also because fasciae form planes that provide routes of surgical access or pathways for hemorrhage and infection.
- At approximately 4 to 5 weeks of gestation, the area of the embryo's future face and neck consists of five or six pairs of fingerlike masses of tissue called the *branchial arches.*
- The outer surfaces of the arches, as well as the clefts, are lined by ectoderm; the substance of the branchial arches contains mesoderm and neural crest cells. The pharyngeal pouches are outpouchings from the foregut region and are composed of endoderm.
- Second branchial cleft anomalies are the most common. The fistula tract starts with the external opening in the mid-lower neck at the anterior border of the sternocleidomastoid muscle, and it heads superiorly between the internal and external carotid arteries, passes over cranial nerves IX and XII, and ends in the palatine tonsil bed.
- Third branchial cleft anomalies travel from the medial edge of the sternocleidomastoid muscle, lateral to the common carotid artery, medial and posterior to the internal carotid artery, over cranial nerve XII, under cranial nerve IX, and through the thyrohyoid membrane into the piriform sinus.
- The paired maxillary and mandibular prominences from the first branchial arch along with the unpaired frontonasal prominence grow and come together to form the structures of the face during weeks 4 to 10 of embryonic development.
- Branchial arches 1 through 4 all contribute to the development of the tongue with the anterior two thirds of the tongue formed from the lateral lingual swelling and medial lingual swelling or tuberculum impar from the first branchial arch; the posterior one third of the tongue develops from the copula or hypobranchial eminence, which arises from the second, third, and fourth branchial arches.
- A Sistrunk procedure with removal of the midportion of the hyoid along with the thyroglossal duct (within the base of the tongue to the foramen cecum) and removal of the cyst is recommended to minimize the risk of recurrence of a thyroglossal duct cyst.

Surgeons use a number of nomenclature systems to organize the anatomy of the head and neck in an attempt to manage surgical approaches and anticipate deviations from normal anatomy. In most textbooks, the head and neck anatomy is divided into fascial layers and triangles or levels. The use of layers and levels is an organizational approach to manage the volume of anatomic detail in the neck by parceling it into reasonable functional units. For example, when a retropharyngeal abscess is diagnosed, surgeons can anticipate the spread of infections within the retropharyngeal space down to the mediastinum or into adjacent layers of the deep cervical fascia, and when an incision is made over the posterior triangle or level V, surgeons can reliably predict the structures they will encounter in precise order. Similarly, understanding the normal embryologic development of the face and neck is important to diagnose anomalies or masses of the head and neck and to define surgical approaches to address them. This chapter begins with brief descriptions of the fascial layers and the anatomic levels of the neck. The chapter continues with a discussion of the embryology of the face that includes the palate and tongue, the neck, the branchial or pharyngeal apparatus, and the contribution of this developmental anatomy to various abnormal and pathologic conditions of the head and neck.

FASCIAL LAYERS OF THE NECK

An understanding of the fascial relationships in the neck is important, not only because of the boundary relationships but also because fasciae form planes that provide routes of surgical access or pathways for hemorrhage and infection.[1] For this reason, a brief discussion of the fascial planes is necessary before proceeding with the anatomy of the triangles or levels of the neck.

One of the earliest lessons in anatomy is that there are two types of fascia in the body, superficial and deep. In the region of the abdominal wall, *superficial fascia* consists of two layers—a fatty (Camper) layer and a deeper, membranous (Scarpa) layer.[2] The *deep fascia* of the abdominal wall is not subdivided but is a layer that simply envelops the abdominal muscles. In

the neck, the superficial layer of cervical fascia is a single layer of fascia that underlies the skin and contains the platysma muscle and cutaneous nerves and vessels. It is usually thin, except in the obese person, in whom it is thickened by adipose tissue. Its primary surgical significance is that it provides a fascial pad that protects underlying structures when a skin incision is made. In exceptionally lean people, however, the paucity of this layer may not protect underlying structures, such as the accessory nerve, so the surgeon should be wary when operating on such patients.

The deep cervical fascia is divided into three layers, illustrated best when the neck is viewed in cross section (Fig. 2-1). These are the superficial (investing), middle (pretracheal or visceral), and deep (prevertebral) layers of the deep cervical fascia. The *superficial layer* of deep fascia underlies the platysma muscle and completely invests or encircles all of the superficial neck structures. For these reasons, the superficial layer is also known as the *investing layer* of deep fascia. In the region of the sternocleidomastoid and trapezius muscles, it splits and envelops the individual muscles. The superficial layer of the deep cervical fascia also invests the strap muscles and parotid and submandibular glands. The *middle layer* of the deep cervical fascia encloses the visceral structures of the neck: the trachea, esophagus, and thyroid gland. Hence, the synonym for the middle layer is the *pretracheal* or *visceral fascia*. The *deep layer* of the deep cervical fascia surrounds the deep muscles of the neck and cervical vertebrae (see Fig. 2-1). This layer is also known by its descriptive term, the *prevertebral fascia*. The muscles enclosed by the prevertebral fascia include the deep muscles of the neck: the levator scapulae; scalenus anterior, middle, and posterior; and longus colli and capitis, which lie on the anterior aspect of the cervical vertebrae. In addition, within the prevertebral fascia are the phrenic nerve and brachial plexus, located near the anterior and middle scalene muscles and the sympathetic chain lying anterior to the longus colli muscle. The superfical layer of deep fascia, along with the middle and deep layers, envelops the carotid and jugular vessels and vagus nerve to form the carotid sheath.

An effective means of visualizing the spatial relationships of these layers of fascia is to examine a cross section of the neck (see Fig. 2-1). This view is not only informative in defining the three layers of deep fascia but it also serves to relate them to the triangles of the neck (i.e., submandibular, posterior). Although the term *triangle* connotes a flat or planar form, the neck triangles are three-dimensional structures that should be visualized as pyramidal spaces that not only have three sides but also have a roof (top) and a floor (bottom). Most of the triangles are three-dimensional spaces bounded by bone and muscles with distinct fascial layers that form the roof and floor of the space. In general terms, each triangle contains blood vessels, nerves, lymphatic vessels, and lymph nodes. Using the fascial layers and triangles, the anatomy of the neck can be organized and simplified; for example, if you place a finger over the middle of the posterior triangle or level V, between the trapezius and sternocleidomastoid muscles (SCMs), you see that the roof (lateral wall) of the triangle is formed by the superficial layer of deep fascia. Palpation deeper into the triangle brings the tip of the finger into contact with the prevertebral fascia that forms the floor of the posterior triangle. If the superficial layer of the deep fascia is incised and you insert a finger into the space to explore anteriorly between the sternocleidomastoid and prevertebral muscles, you encounter the carotid sheath. This is a surgical approach to the retropharyngeal area or to the carotid vessels.

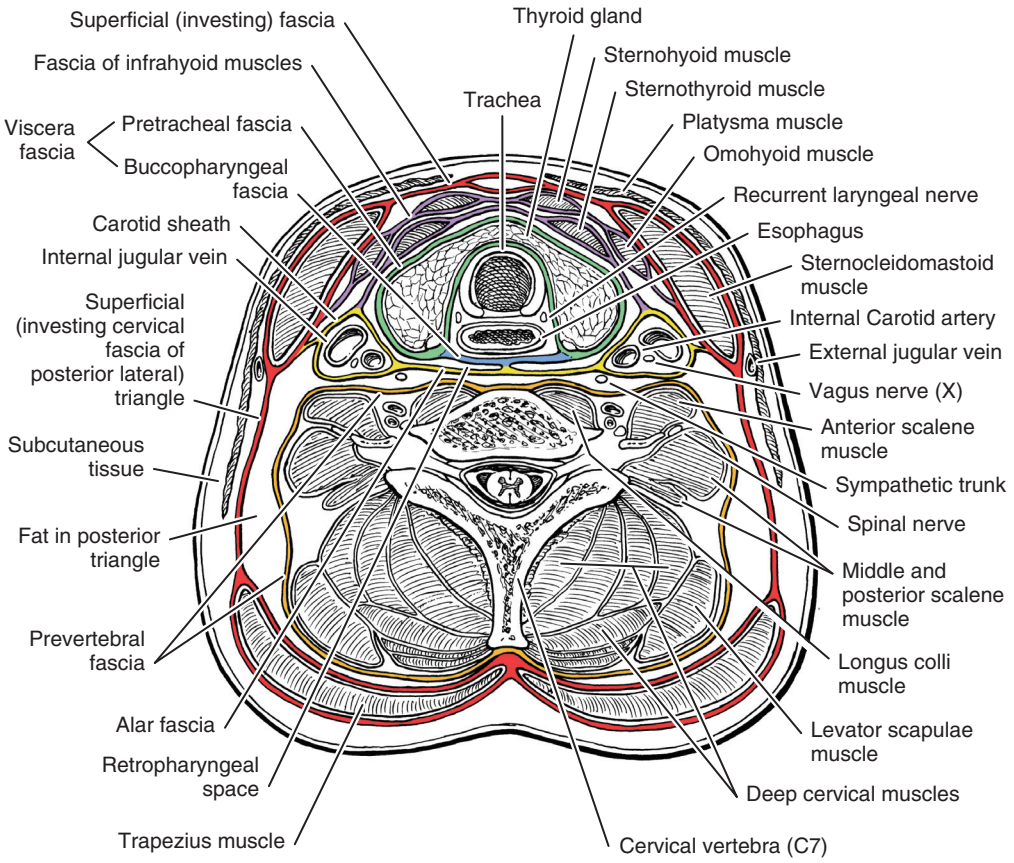

FIGURE 2-1. Divisions of and structures contained by the deep cervical fascia seen in transverse section at the level of cranial nerve VII. (Modified from http://www.projectlumi.org/blog/wp-content/uploads/2012/04/Picture1.jpg.)

TRIANGLES AND LEVELS OF THE NECK

Nomenclature of the cervical lymphatic tissues historically has been inconsistent with descriptive terms such as *deep, superficial, anterior,* and *lateral.* As the focus of oncologic surgery has shifted from radical extirpation to functional preservation, it has become more important to standardize the classification system for the cervical nodes. The American Academy of Otolaryngology–Head and Neck Surgery, along with the American Society of Head and Neck Surgery, has developed a standardized classification system for the cervical lymphatic system based on levels I through VI of the neck (Fig. 2-2).[3]

Lymph node levels I through VI can also be divided into subzones (see Fig. 2-2). Level I includes lymph nodes in the submental (Ia) and submandibular (Ib) triangles. Levels II through IV include the jugular nodes in fibroadipose tissue located between the lateral border of the sternohyoid muscle and the posterior border of the SCM. Level II encompasses the upper jugular lymph nodes and extends from the skull base superiorly to the carotid bifurcation or hyoid inferiorly. The spinal accessory nerve divides level II into anterior (IIa) and posterior (IIb) compartments. Level III includes the middle jugular nodes around the middle third of the jugular vein and extends from the carotid bifurcation superiorly to the omohyoid or cricothyroid notch inferiorly. Level IV includes the lower jugular group of lymph nodes that surround the jugular vein from the omohyoid to the clavicle. Level V is the posterior triangle group, which encompasses lymph nodes located between the posterior border of the SCM and the anterior border of the trapezius muscle. Level V includes the nodes that surround the lower half of the spinal accessory nerve (Va) and the transverse cervical artery (Vb or the supraclavicular nodes). Level VI includes the anterior compartment group that surrounds the visceral structures of the neck from the hyoid superiorly to the sternal notch inferiorly. The lateral boundary on either side of level VI is the medial aspect of the carotid sheath. This space crosses the midline and includes the perithyroidal, paratracheal, and precricoid nodes in addition to the nodes along the recurrent laryngeal nerve.[3]

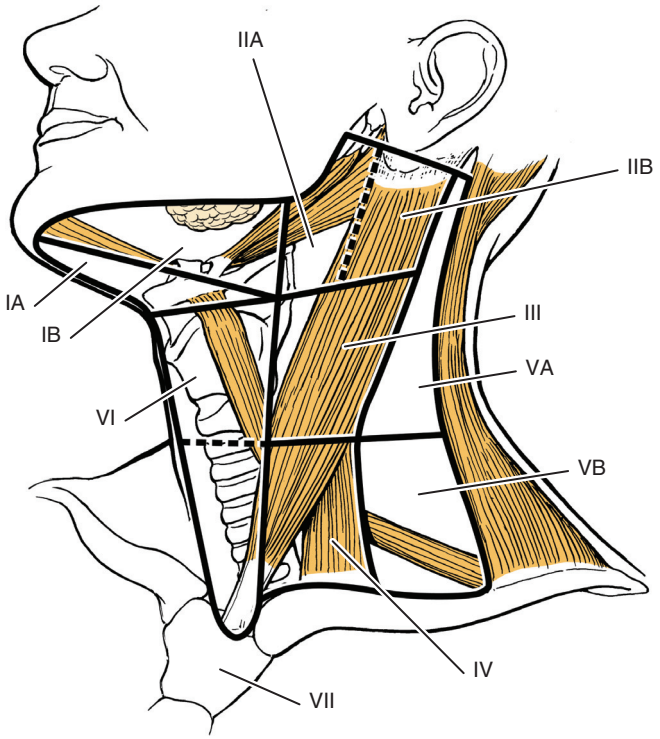

FIGURE 2-2. Lymph node levels and boundaries of the neck. (Modified from http://www.aboutcancer.com/Lymph_node_levels_of_neck_0509.jpg.)

DEVELOPMENT OF THE HEAD AND NECK

The development of many structures in the head and neck is intimately related to the branchial or pharyngeal apparatus. These are transient embryonic structures that undergo substantial remodeling so that their original embryonic form is essentially unrecognizable by the time a child is born. The derivatives of these structures, nevertheless, are important to adult morphology; hence, aberrations in branchial apparatus development may produce significant malformations.

EMBRYOLOGY OF BRANCHIAL ARCHES

At approximately 4 to 5 weeks of gestation, the area of the future face and neck of the embryo consists of five or six pairs of fingerlike masses of tissue named the *branchial arches* (Fig. 2-3). Prominent in lateral profile, these masses are aligned transversely to the plane of the neck and are separated externally by indentations, termed the *branchial clefts.* The outer surfaces of the arches, as well as the clefts, are lined by ectoderm, whereas the substance of the branchial arches contains mesoderm and neural crest cells. Only the first branchial cleft

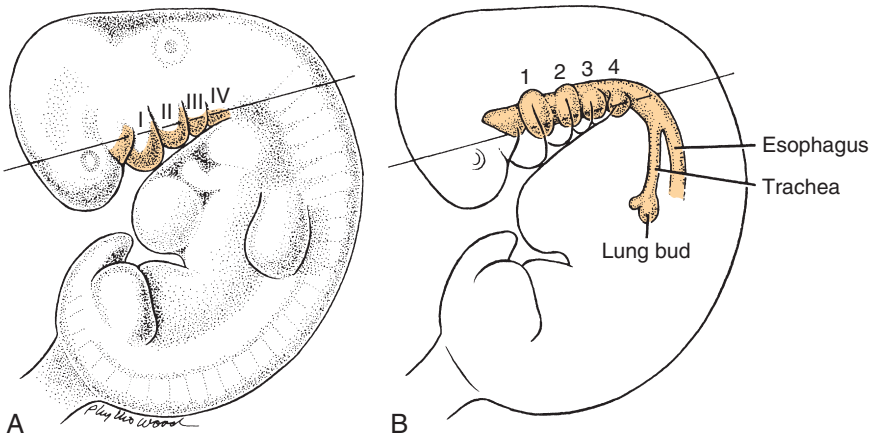

FIGURE 2-3. A, Branchial arches. **B,** Pharyngeal pouches.

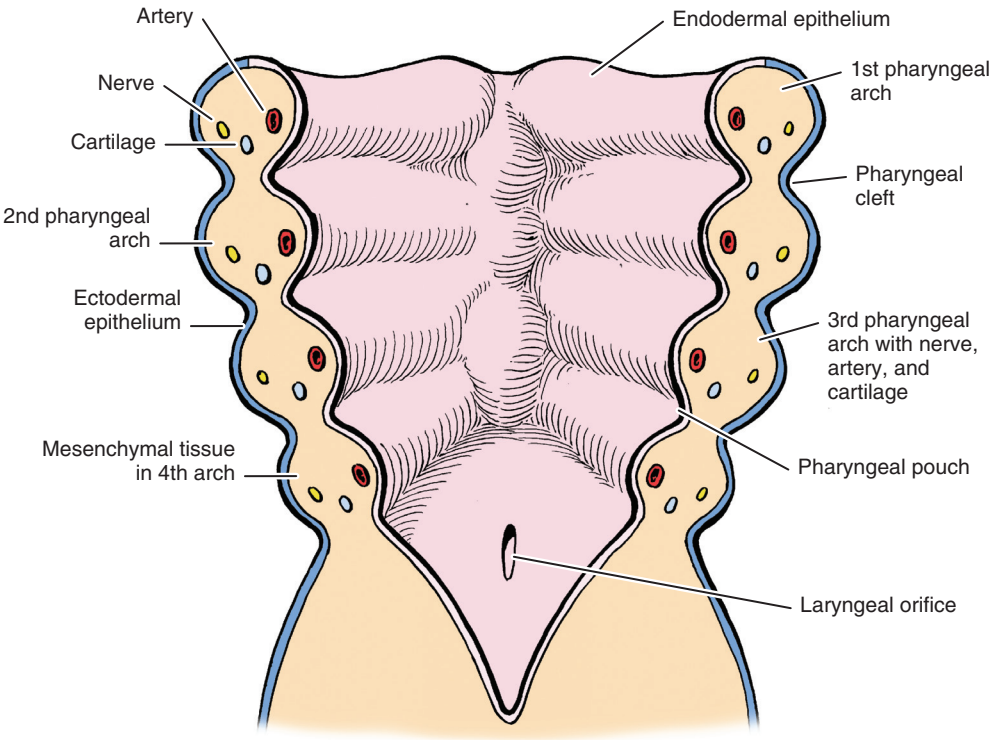

FIGURE 2-4. Relationships of the branchial arches, clefts, and pouches in the floor of the mouth. (From Robbins KT, et al: Standardizing neck dissection terminology: official report of the academy's committee for head and neck surgery and oncology. *Arch Otolaryngol Head Neck Surg* 1991;117:601.)

contributes to a definitive structure of the embryo: the external auditory canal and the outer layer of the tympanic membrane. The remaining second, third, and fourth branchial clefts fuse with the epicardial ridge in the lower part of the neck.[4]

The pharyngeal pouches are outpouchings from the foregut region (Fig. 2-4) and are composed of endoderm. The derivatives of the arches, clefts, and pouches are distinct, because they are derived from different embryonic germ layers: mesoderm, ectoderm, and endoderm, respectively. To generalize, it can be stated that in the adult, the derivatives of the branchial arch will be structures composed of muscle, bone, or similar mesodermal derivatives, and the derivatives of the endodermal pharyngeal pouch will be glandular or associated with the digestive tract.

Derivatives of Branchial Arches

In the early phase of branchial arch development, the mesodermal mass of the arch forms a bar of cartilage, which remodels into bone, cartilage, or other connective tissue elements in the adult. Similarly, the adult musculature of the face and neck develops from the mesoderm of the branchial arches. Each arch also has an associated cranial nerve and artery. Because of the proximity of the developing branchial arches to the brainstem, each branchial arch receives motor or sensory innervation from an adjacent cranial nerve. A comparable parallel to this pattern occurs in the trunk, where a muscle is derived from the myotome region of a somite and receives its innervation from the adjacent segmental spinal nerve. In both cases, regardless of where the primordial muscle cell migrates, it retains its primary embryonic innervation. After each arch receives its cranial nerve innervation, the adult pattern is established, regardless of its future migration onto the back of the head or base of the neck. The development of each branchial arch is considered separately.

First Branchial Arch. The trigeminal nerve supplies motor innervation to all the muscles derived from the first branchial arch. In addition, sensory innervation is provided not only over the region of the mandible via the third division of the trigeminal nerve but also over the maxillary process of the first arch and the frontal nasal process via the second maxillary and first ophthalmic division, respectively, of the trigeminal nerve. The artery of the first branchial arch is the internal maxillary artery.

For skeletal derivatives of the first arch, the proximal part of the Meckel cartilage is remodeled and contributes to the formation of the ramus of the mandible (Fig. 2-5). The distal part of the cartilage withers, and the body of the mandible is formed from intramembranous bony growth. Other structures

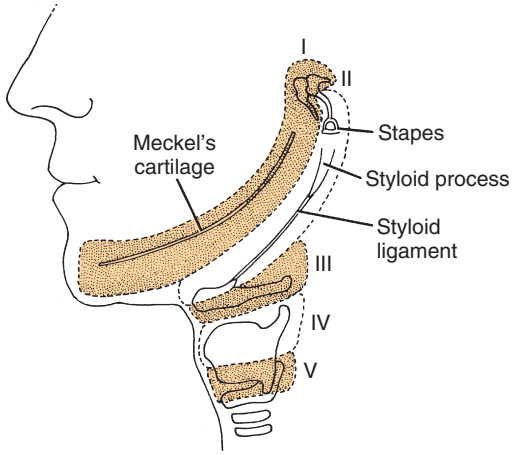

FIGURE 2-5. Skeletal derivatives of the branchial arches.

formed by the proximal part of the Meckel cartilage are the sphenomandibular ligament, anterior malleolar ligament, malleus (except the manubrium, which is from the second arch), and the incus (except for its long process, which is from the second arch). The maxillary process is derived from the dorsal portion of the first arch and contributes to the development of the premaxilla, maxilla, zygoma, and part of the temporal bone.

The muscles formed by mesodermal elements of the first branchial arch include the muscles of mastication: the temporalis, masseter, and medial and lateral pterygoid muscles. In addition, the tensor tympani, tensor veli palatini, anterior belly of the digastric, and mylohyoid muscle are also derived from first arch mesoderm.

Second Branchial Arch. The facial nerve, the nerve of the second branchial arch, supplies motor innervation to all of the muscles derived from this mesoderm, namely the muscles of facial expression. Other than a small sensory branch of cranial nerve VII, which may supply part of the external auditory meatus, there is no sensory distribution of cranial nerve VII to ectoderm. The second branchial arch vascular derivative is the stapedial artery, which only rarely persists.

The Reichert cartilage of the second branchial arch forms bony structures proximally and distally. Its central portion withers, leaving a fibrous band—the stylohyoid ligament. Proximally, it forms the styloid process, manubrium of the malleus, long process of the incus, and stapes suprastructure (see Fig. 2-5). The stapes foot plate is mostly derived from the otic capsule.[5,6] Distally (anteroinferiorly), the second arch cartilage forms the superior portion of the body and the lesser cornu of the hyoid bone. In the adult, the path of the embryonic second arch cartilage can be traced from the styloid process to the stylohyoid ligament, ending at the lesser cornu of the hyoid bone (see Fig. 2-5).

In the second branchial arch, the mesoderm forms all of the muscles of facial expression from the scalp (frontalis) to the platysma muscle in the neck and includes the orbicularis oculi, orbicularis oris, and buccinators. In addition to the muscles of facial expression, the second arch mesoderm gives rise to the posterior belly of the digastric, stylohyoid, auricularis, and stapedius muscles.

Third Branchial Arch. Cranial nerve IX (glossopharyngeal) supplies the only muscle derived from the third arch—the stylopharyngeus, a small muscle formed from the mesoderm that aids in elevating the pharynx during swallowing. However, as will be seen in the next section of this chapter, the glossopharyngeal nerve also supplies sensory innervation to parts of the pharynx associated with this region. The common carotid artery and proximal internal carotid artery are derived from the third branchial arch, and the cartilage elements derived from the third arch form the remaining portions of the hyoid bone (i.e., the inferior body and greater cornu; see Fig. 2-5).

Fourth and Sixth Branchial Arches. The vagus nerve and the cranial part of the accessory nerve supply the muscles derived from the fourth (superior laryngeal branch of the vagus) and sixth (recurrent laryngeal branch of the vagus) branchial arches; the fifth arch structures undergo resorption. Originating in the nucleus ambiguus of the medulla, the axonal processes of these nerves descend in the vagus nerve after they exit the skull via the jugular foramen. Specifically, the pharyngeal constrictors are supplied by the pharyngeal branch of the vagus and the transitional portion of the pharynx and esophagus is supplied via the recurrent laryngeal branch of the vagus. The superior laryngeal nerve supplies sensation to the larynx via its internal branch and provides motor innervation to the cricothyroid muscle via the external branch. The recurrent laryngeal nerve supplies the intrinsic muscles of the larynx. The arch of the aorta between the common carotid and subclavian arteries and the right proximal subclavian artery are derived fourth branchial arch. The sixth arch gives rise to the ductus arteriosus on the left and the proximal right pulmonary artery on the right.

The fourth and sixth branchial arches comprise the cartilaginous elements, which contribute to the formation of the thyroid and cuneiform laryngeal cartilages from the fourth arch and the cricoid, arytenoid, and corniculate cartilages from the sixth arch (see Fig. 2-5).

The mesoderm from the fourth through sixth branchial arches forms the muscles that comprise the pharynx and larynx. Pharyngeal muscles include the levator veli palatini and pharyngeal constrictor muscles. In addition, the mesodermal elements from these branchial arches form the striated muscle that composes the upper half of the esophagus. The inferior part of the esophagus is usually composed of smooth muscle derived from the splanchnic mesoderm of the primitive foregut. The laryngeal muscles are also formed from mesodermal elements in the fourth, fifth, and sixth arches. These include the *extrinsic* laryngeal muscles—the thyroepiglottic, aryepiglottic, and cricothyroid—and the *intrinsic* muscles associated with movement of the arytenoid cartilages and true and false vocal folds, the posterior cricoarytenoid, lateral cricoarytenoid, transverse arytenoid, and oblique arytenoid muscles.

EMBRYOLOGY OF THE PHARYNGEAL POUCHES

The pharyngeal pouches are lateral outpouchings of the foregut or the region of the primitive pharynx (see Fig. 2-3). At the extreme lateral wall of each pharyngeal pouch, the endodermal lining contacts the ectodermal epithelium of the corresponding branchial cleft (Fig. 2-6; see also Fig. 2-4). Thus the branchial clefts are named in relation to the pharyngeal pouch with which they are apposed. The endodermal epithelium lining of the pharyngeal pouch contributes to the formation of specific elements of the pharynx in the adult (see Fig. 2-6).[4] An animated illustration of the derivatives of the pharyngeal pouches is available online at the Indiana University Human Embryology Animations Web site.[7]

First Pharyngeal Pouch

The first pharyngeal pouch becomes elongated and is incorporated into the temporal bone and forms the tubotympanic recess, which eventually becomes the middle ear and eustachian tube (see Fig. 2-6). The most lateral portion of the pouch, along with the closing plate of the first branchial cleft, forms the inner layer of the tympanic membrane. From this relationship, it is clear that the external auditory canal is formed from remodeling of the first branchial cleft.

Second Pharyngeal Pouch

The endodermal layer of the second pharyngeal pouch forms the epithelial lining of the tonsillar fossa and palatine tonsil, whereas the underlying mesenchymal elements contribute to the incorporation of the lymphoid tissue of the tonsil proper (see Fig. 2-6).

Third Pharyngeal Pouch

The region of the third pharyngeal pouch is subdivided into superior and inferior portions. The *superior portion* forms cells that eventually differentiate into the paired inferior parathyroid glands; the *inferior portion* of the third pouch forms thymic tissue, which eventually migrates into the neck and

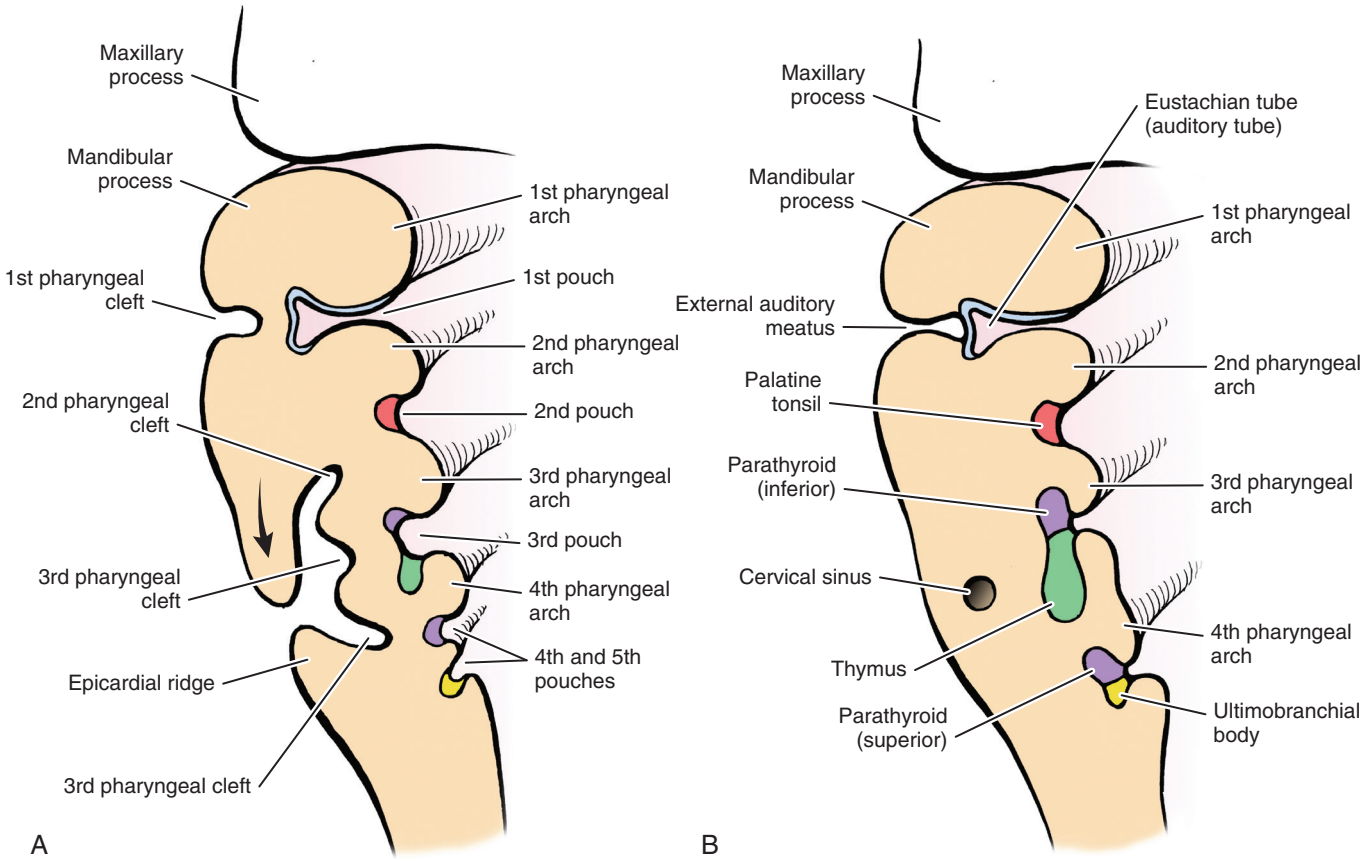

FIGURE 2-6. A, Derivatives of the pharyngeal pouches and formation of the cervical sinus. **B,** Maturation of the pharyngeal pouches.

retrosternal mediastinum to form the thymus (Fig. 2-6). Aberrant migration of the third pharyngeal pouch may result in ectopic inferior parathyroid or thymus tissue.

Fourth Through Sixth Pharyngeal Pouches

The endoderm of the fourth pouch forms the parathyroid glands in the superior poles of the thyroid gland. The adjacent area is variably named as the *fifth pouch, sixth pouch,* or the *ultimobranchial body* (see Fig. 2-6). Subsequently, the ultimobranchial body is infiltrated by cells that migrate from the neural crest region. These cells are eventually incorporated into the thyroid gland and become the parafollicular cells (C cells) that are responsible for the secretion of calcitonin.[8,9]

CRANIOFACIAL SYNDROMES RELATED TO BRANCHIAL ARCH STRUCTURES

Treacher Collins syndrome, or mandibulofacial dysostosis, is related to abnormalities of the first branchial arch. This syndrome is characterized by lid colobomata, down-slanting palpebral fissures, malar hypoplasia, micrognathia, and malformations of the external ear and ossicles.[10] Goldenhar syndrome (oculoauricular vertebral dysplasia), in addition to ocular and vertebral anomalies, includes abnormalities of the first and second branchial arches with unilateral facial hypoplasia, micrognathia, microtia, external auditory canal atresia, malformation of the ossicles, and facial nerve hypoplasia. Craniofacial or hemifacial microsomia is thought to be related to a vascular injury in the region of the stapedial artery (second arch artery) that results in microtia, aural atresia, and mandibular hypoplasia.[11]

Other syndromes related to dysmorphogenesis of the first and second branchial arches include branchio-oto-renal syndrome, characterized by branchial cleft cysts, preauricular pits, abnormal pinnae, malformed ossicles, and hearing loss, as well as renal dysplasia, and Pierre Robin sequence with micrognathia, glossoptosis, and cleft palate. DiGeorge sequence, or third and fourth pharyngeal pouch syndrome, is characterized by hypoplasia or absence of the thymus and/or parathyroid glands, cardiovascular defects, immunologic problems, and hypocalcemia.

ANOMALIES OF THE BRANCHIAL APPARATUS

The first branchial cleft becomes part of the external auditory canal, but the remaining branchial clefts are remodeled and normally do not form derivatives identifiable in the adult. However, the complicated morphodynamics of the branchial arch region seem to predispose this area to abnormalities that range from minor cysts to major orofacial malformations. The clinical terminology of the various branchial abnormalities is confusing. Part of the confusion results from the clinical spectrum of these anomalies; that is, they may present as a cyst, sinus, or fistula. A *cyst* has no communication with the body surface; a *sinus* communicates with a single body surface, either the skin or pharynx; and a *fistula* communicates with two body surfaces. A cystic dilation of the tract may be associated with either a sinus or a fistula. The terms *branchial cleft* and *branchial pouch* are often used interchangeably, although they refer to distinct structures. Occasionally, the term *groove* is used instead of *pouch* or *cleft*. Isolated branchial arch remnants may present

as subcutaneous cartilaginous tags along the anterior border of the SCM.

ABNORMALITIES OF THE FIRST BRANCHIAL CLEFT

Aberrant development of the first branchial cleft may lead to the formation of a cervical cyst or sinus in the region of the ear. Work[12] and Aronsohn and others[13] emphasized the difference in the embryogenesis of preauricular cysts and cysts of the first branchial cleft. Preauricular cysts or sinuses occur anterior to the external auditory canal, usually superior to the tragus. In essence, they are inclusion cysts related to incomplete fusion of the ectodermal hillocks of His from the first and second branchial arches during formation of the auricle.[14]

In contrast, true first branchial cleft abnormalities include stenosis or atresia of the external auditory canal or duplications of the membranous part of the external auditory canal. First branchial cleft anomalies are rare and can present clinically as cysts, sinuses, or fistulae. Work[12] has classified these anomalies into two types: *type I* represents duplication of the ectodermal external auditory canal; *type II* is duplication that involves both ectodermal and mesodermal elements of the canal, including the cartilaginous portion of the canal. As cystic masses or sinus tracts, they may involve the parotid gland and cranial nerve VII (this is particularly true of type II) and lie inferior to the ear or in the superior neck, and they may present as a recurrent inflammatory lesion in the external auditory canal or periauricular region. If a first branchial cyst or sinus is infected, treating with antibiotics and waiting for resolution of the acute infection prior to excision via a parotidectomy incision are recommended to optimize identification and preservation of the facial nerve.

ABNORMALITIES OF THE SECOND BRANCHIAL CLEFT

During closure of the cervical sinus that lies between the second branchial arch and the epicardial ridge, ectoderm may become trapped and can result in an inclusion cyst with or without a sinus or fistula tract (see Fig. 2-6). These are the most common branchial cleft anomalies. Second branchial cleft cysts lie in the lateral neck anterior to the SCM. The fistulous forms of these abnormalities are surgically challenging, because they may extend from the superficial area of the neck near the clavicle to the bed of the palatine tonsil superiorly. The course of the fistula tract starts with the external opening in the mid-lower neck at the anterior border of the SCM, penetrates the platysma muscle, heads superiorly between the internal and external carotid arteries, and passes over cranial nerves IX and XII and below the stylohyoid ligament to end in the palatine tonsil bed. The pathway of this tract is easily explained on an embryologic basis.

ABNORMALITIES OF THIRD AND FOURTH BRANCHIAL CLEFTS AND POUCHES

Third branchial cleft anomalies also travel from the medial edge of the SCM, lateral to the common carotid artery, medial and posterior to the internal carotid artery, over cranial nerve XII, under cranial nerve IX, and through the thyrohyoid membrane into the piriform sinus. Thymopharyngeal cysts represent third pharyngeal pouch remnants. Piriform apex sinus tracts are debated to be thymopharyngeal duct remnants versus anomalies of the fourth branchial or pharyngeal pouch. These sinus tracts clinically present as recurrent suppurative thyroiditis and are usually located on the left side.[15-17] These lesions may be associated with parathyroid, thymic, or thyroid tissue. Fourth branchial cleft fistulae theoretically start medial to the SCM, travel inferiorly around the subclavian artery on the right or around the arch of the aorta on the left, lateral to cranial nerve XII, inferior to superior laryngeal nerve, and then end in the apex of the piriform sinus or cervical esophagus.

DEVELOPMENT OF THE FACE, PALATE, AND LIP

The paired maxillary and mandibular prominences from the first branchial arch, along with the unpaired frontonasal prominence, grow and come together to form the structures of the face during weeks 4 to 10 of embryonic development (Fig. 2-7). First, at the fifth week, the nasal placode develops as an ectodermal thickening in the inferior lateral portion of the frontonasal process. The mandibular prominences migrate medially to merge to form the mandible, lower lip, and lower cheeks. During the sixth week of development, each nasal placode develops a central pit and is divided into lateral and medial nasal processes, which will eventually form the nostril. At the seventh week, the paired nasal processes and maxillary prominences migrate medially. The medial nasal prominences fuse with each other and the maxillary prominences to form the philtrum and upper lip, nasal tip and septum, and intermaxillary segment (eventual primary palate). The lateral nasal prominences merge with the maxillary prominences to form the lateral nasal ala and nasolacrimal groove. The maxillary prominences give rise to the lateral palatine processes, which contribute to the maxillary bone and secondary palate. By 10 weeks of gestation, the features of the neonatal face have all developed (see Fig. 2-7).

The palate develops starting around week 6, when the palatine shelves from the maxillary prominences migrate medially toward the developing tongue, and the medial nasal processes merge to form the primary palate. At weeks 7 to 8, as the tongue drops, the palatine shelves begin to migrate medially to fuse together posteriorly, with the primary palate anteriorly and with the nasal septum superiorly, to form the complete palate (Fig. 2-8). The incisive foramen marks the boundary between the primary and secondary palate.

Abnormal fusion of the medial nasal processes may result in median cleft lip. When the maxillary prominence on one side fails to fuse properly with the intermaxillary segment from merged medial nasal processes, a unilateral cleft lip will result. If this occurs on both sides, a bilateral cleft lip will occur. When the maxillary prominence does not fuse properly with the lateral nasal process, an oblique facial cleft will develop. Cleft palates can occur anteriorly if the fusion of the primary and secondary palates fails and posteriorly if the lateral palatal processes that form the secondary palate fail to fuse.

DEVELOPMENT OF THE TONGUE

Branchial arches 1 through 4 all contribute to the development of the tongue (Fig. 2-9). At 4 weeks of gestation, the lateral lingual swelling and medial lingual swelling or tuberculum impar from the first branchial arch form the anterior two thirds of the tongue. Sensory innervation comes from the lingual nerve of the mandibular branch of the trigeminal nerve, and taste sensation is carried on the chorda tympani, a branch of the facial nerve. The posterior one third of the tongue develops from the copula or hypobranchial eminence, which arises from the second, third, and fourth branchial arches. Sensory and taste innervation is carried primarily on the glossopharyngeal nerve from the third arch, although the posterior tongue (vallecula) and epiglottis are innervated by the superior laryngeal branch of the vagus nerve from the fourth arch. The junction

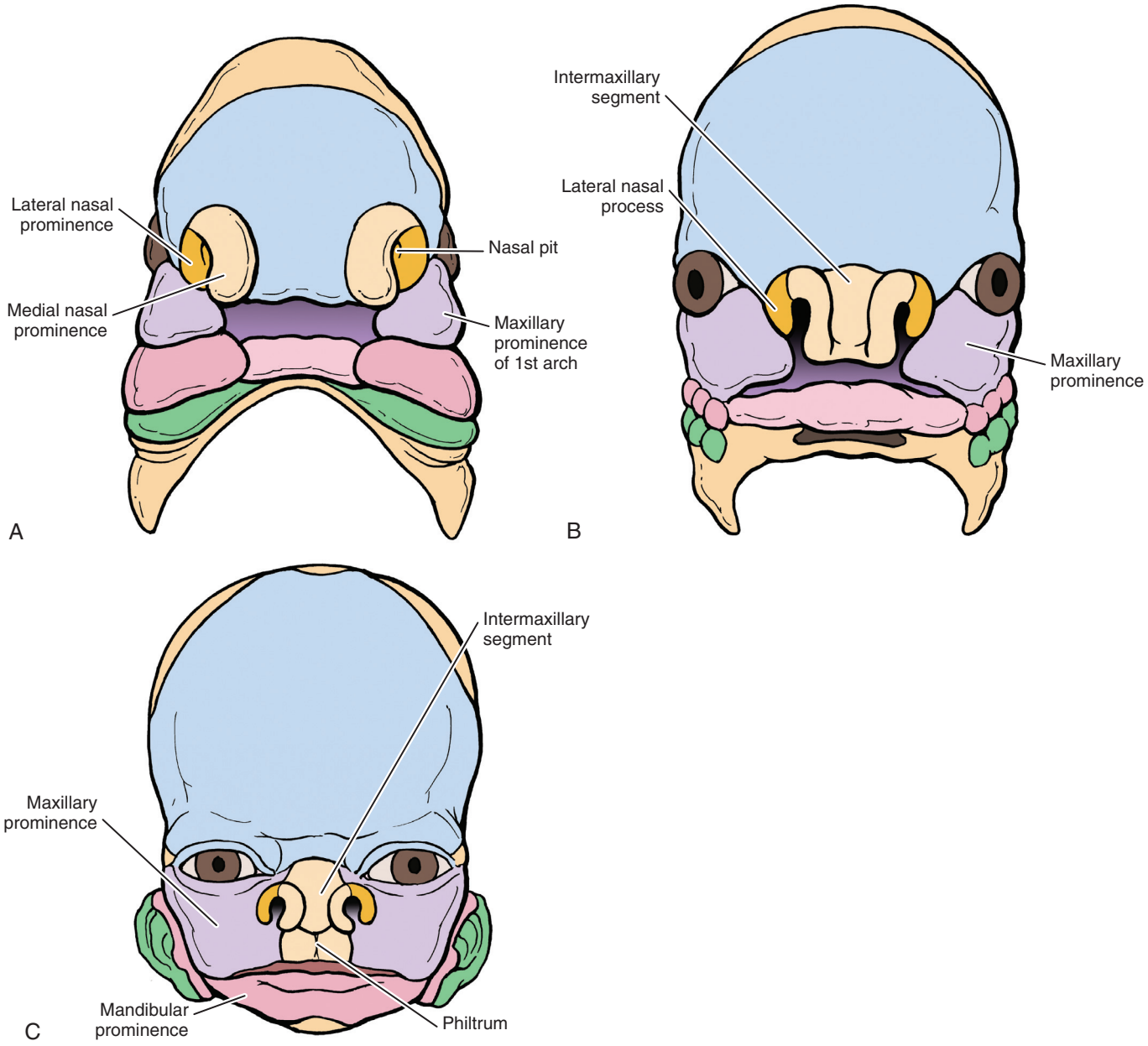

FIGURE 2-7. Developmental anatomy of the face. (Modified from Stoffer J: Development of the Head and Neck. Available at www.indiana.edu/~anat550/hnanim/face/JAS_facetween.html.)

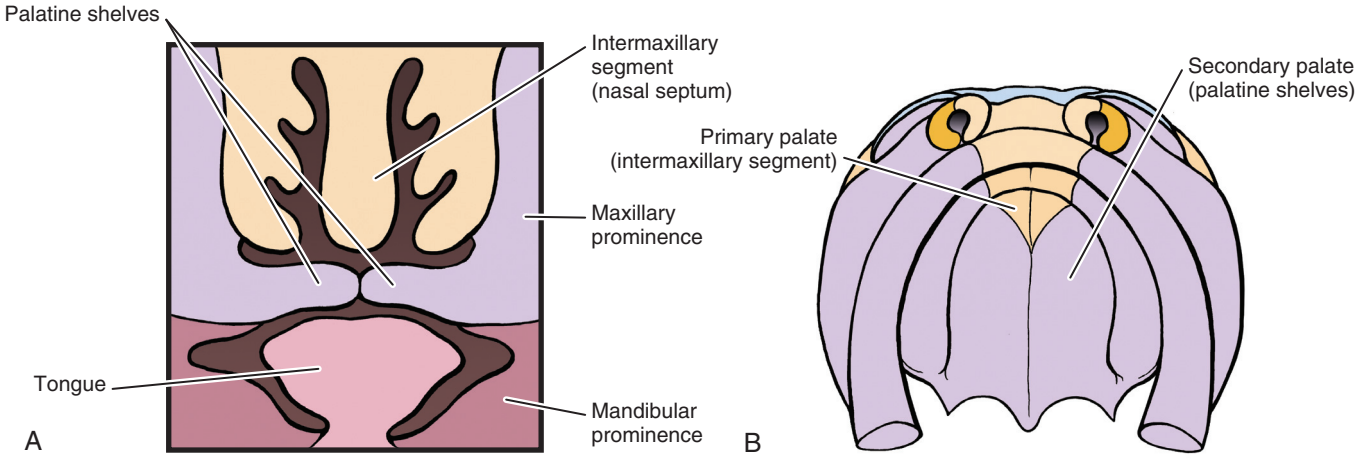

FIGURE 2-8. Development of the palate. (Modified from Stoffer J: Development of the Head and Neck. Available at www.indiana.edu~anat550/hnanim/face/face.html.)

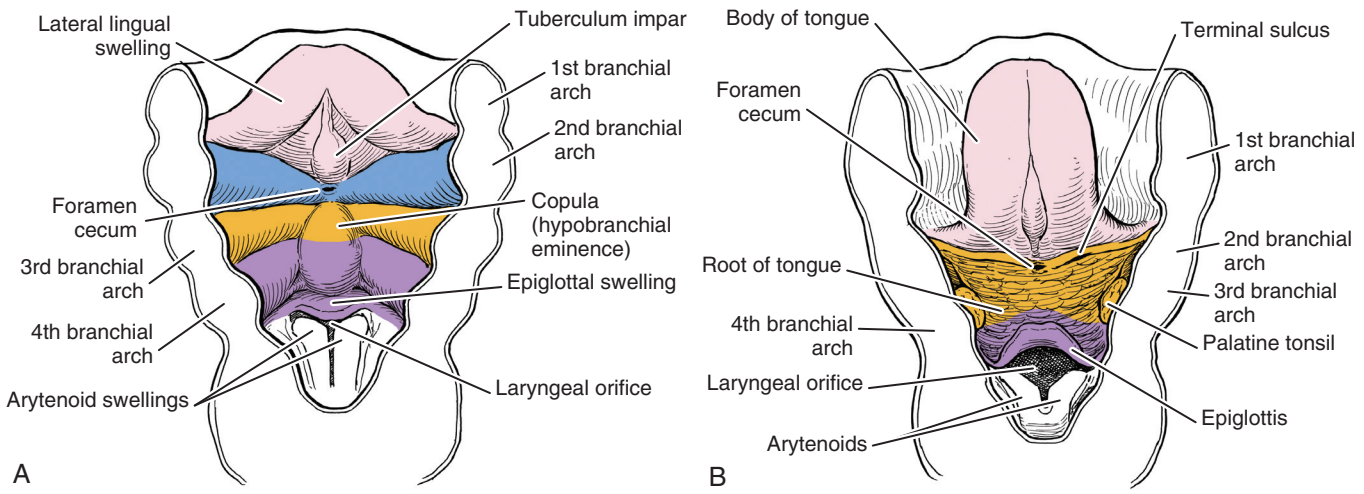

FIGURE 2-9. Development of the tongue. **A,** At weeks 4 to 5, the first arch forms the medial tuberculum impar and the lateral lingual swellings. The second, third, and fourth arches form the copula, or hypobranchial eminence. **B,** The first arch forms the anterior tongue, whereas the third arch primarily contributes to the posterior tongue. The fourth arch forms the root of the tongue and epiglottis. (Modified from Sadler TW: *Langman's medical embryology*, ed 12, Philadelphia, 2011, Lippincott Williams & Wilkins.)

between the anterior and posterior tongue is V shaped and is called the *terminal sulcus*. The intrinsic tongue musculature differentiates from migration of myoblasts from occipital somites and is innervated by the hypoglossal nerve, except for the palatoglossus, which is innervated by the pharyngeal plexus of the vagus (Fig. 2-10).

DEVELOPMENT OF THE THYROID GLAND

The thyroid develops as a small mass in the tongue, which invaginates around the fifth week of embryonic development. This point of origin and invagination is called the *foramen cecum.* The thyroid gland migrates inferiorly through the developing neck and is intimately associated with the hyoid bone. By the seventh week, the thyroid gland reaches its final destination in the neck just inferior to the cricoid cartilage anterior to the trachea. The thyroid gland is functional by the tenth to twelfth week of development. Abnormal migration of the thyroid gland may result in ectopic thyroid tissue. The tract of the migration of the thyroid gland, termed the *thyroglossal duct,* normally collapses and atrophies. If the duct fails to undergo atrophy, it may result in a thyroglossal duct cyst.

The thyroglossal duct cyst derives from persistence of the embryonic thyroglossal duct anywhere between the foramen cecum and the thyroid gland (Fig. 2-11). Most commonly, the thyroglossal duct cyst is found just above the thyroid lamina and

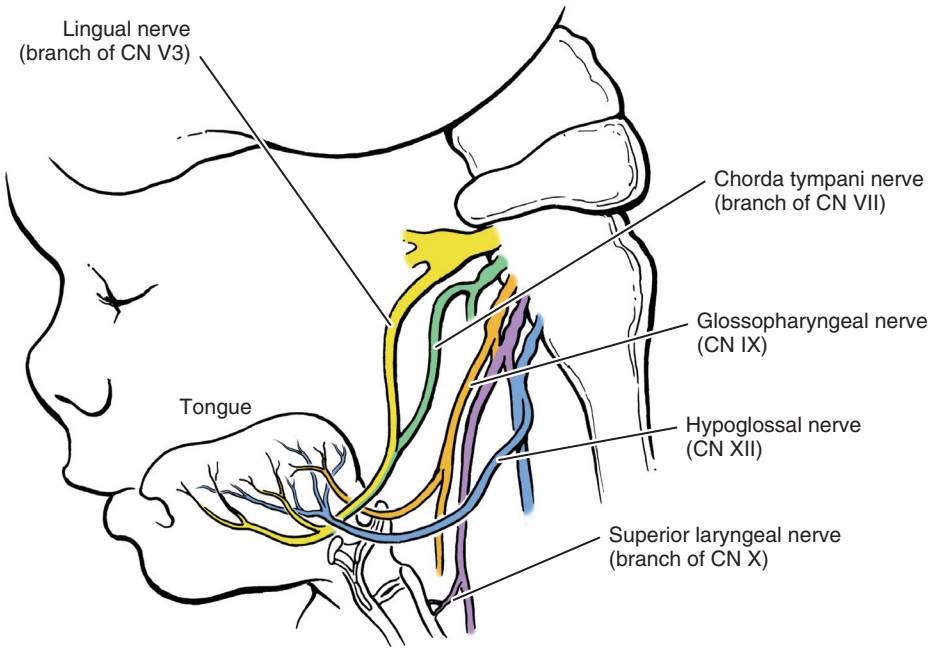

FIGURE 2-10. Sensory and taste innervations of the tongue. CN, cranial nerve. (Modified from Stoffer J: Development of the Head and Neck. Available at www.indiana.edu~anat550/hnanim/tongue/tongue.html.)

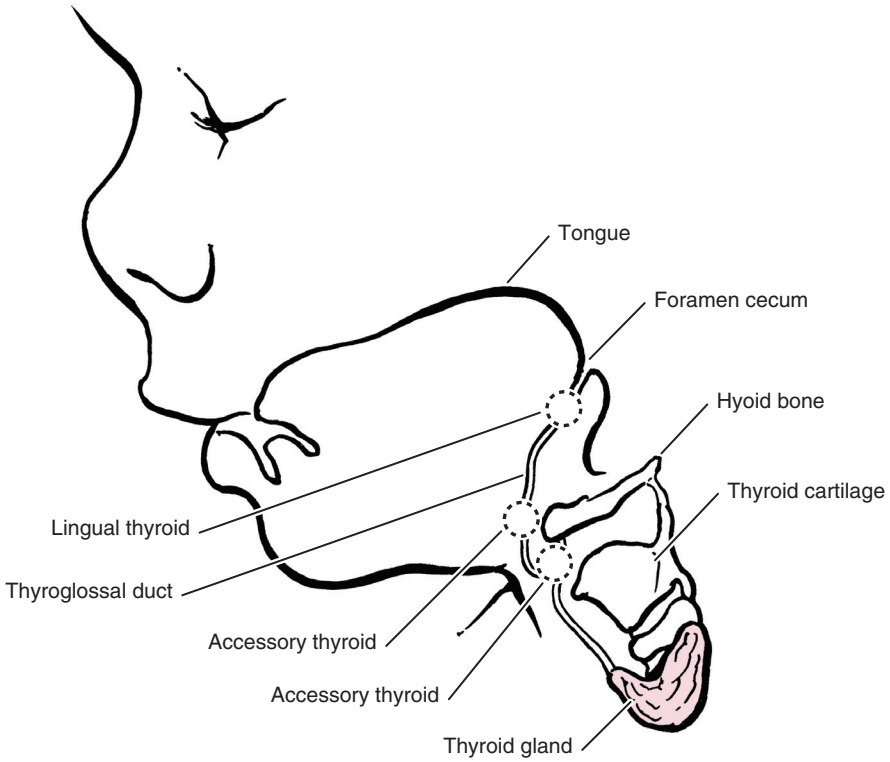

FIGURE 2-11. Migration of thyroid tissue. (Modified from Sadler TW: *Langman's medical embryology*, ed 12, Philadelphia, 2011, Lippincott Williams & Wilkins.)

below the hyoid bone. Because of the attachments to the base of the tongue, thyroglossal duct remnants will move superiorly in the neck when the tongue is protruded; the thyroglossal duct remnant may contain ectopic thyroid tissue, and occasionally, the cyst contains all of the functioning thyroid tissue. Because of the potential for permanent hypothyroidism after surgical excision, many authors advocate routine preoperative assessment of the thyroid.[18,19] Ultrasound of the neck provides information regarding the consistency of the cystic lesion and can also be used to assess for the presence of thyroid tissue in its normal position lower in the neck. Ultrasound examination is easily tolerated by children but does not give functional information; radionuclide thyroid scans provide functional information regarding thyroid tissue. It is important to note that the possibility of malignancy, usually arising from thyroid tissue, is present within these lesions. Thyroglossal duct cysts often present as a midline neck mass that can become infected with upper respiratory tract infections that can cause rapid enlargement, erythema, and drainage. Surgical excision is the treatment of choice. Because the thyroglossal duct remnants are intimately associated with the central part of the hyoid, removal of the midportion of the hyoid, along with the thyroglossal duct (within the base of the tongue to the foramen cecum) and the cyst—a procedure first described by Sistrunk[20]—is recommended to minimize the risk of recurrence.

SUMMARY

The otolaryngologist–head and neck surgeon must be familiar with the developmental anatomy of the head and neck, because a working knowledge of head and neck anatomy and embryology is critical in diagnosing and treating patients effectively.

For a complete list of references, see expertconsult.com.

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3

Anesthesia in Pediatric Otolaryngology

Veronica C. Swanson | Pravin A. Taneja | Heike Gries | Jeffrey Koh

Key Points

- When anesthetic risk is correlated with age, children younger than 1 month of age are at the highest risk for adverse events during any procedure. The greatest improvements in patient safety have been in reducing the frequency of respiratory events. Elective procedures should be deferred until after the age of 6 months, when appropriate.
- The anesthetic plan is tailored to the patient's age—for physiologic differences as well as differences in psychological development.
- Preoperative preparation that includes patient education leads to greater satisfaction with the overall hospital experience for both child and parent.
- Routes of premedication and induction of anesthesia include oral, intranasal, intramuscular, intravenous, and inhalational anesthetics.
- Decisions regarding deep versus awake extubation require careful consideration of clinical factors; awake extubation is always the appropriate choice in patients who have difficult airway anatomy or full-stomach physiology.
- Postoperative nausea and vomiting are best treated with multimodal therapy; a second dose of antiemetic agent in the same class of drug already given is rarely effective.
- Effective pain assessment and management are mandated by the Joint Commission for institutions to receive accreditation.
- Multimodal therapy for pain relief, with or without opioids, is now accepted as a more balanced approach to pain management in surgical patients. Such therapy aims to decrease the dose of any single agent and thereby improve antinociception and reduce side effects.
- Anesthetizing patients in locations outside of the operating room minimizes interference with surgical needs and contributes to efficient delivery of care.
- Implications of anesthetic neurotoxicity on the developing nervous system in humans is still unclear and is being researched. The most conservative approach would be to delay all elective procedures until after two years of age. The Food and Drug Administration does not support this position but suggests full disclosure when discussing surgical options with patients and their families.
- Medical conditions with particular impact on anesthetic considerations include upper respiratory infections, reactive airway disease, obstructive sleep apnea, cystic fibrosis, diabetes mellitus, and congenital heart disease.
- The 2007 American Heart Association guidelines for the prevention of infective endocarditis in patients with congenital heart disease seek to use evidence-based practices to minimize unnecessary exposure to antibiotics.
- Management of patients for bronchoscopy, adenotonsillectomy, and laryngeal surgery requires careful planning preoperatively and clear communication between the surgeon and the anesthesiologist throughout the procedure.

Because of the inherent complexity of the structure-function relationship in otolaryngology, pediatric otolaryngologists and anesthesiologists must work together closely in the operating room (OR) to provide optimal care to the patient. This chapter focuses on anesthesia topics that are relevant for the pediatric otolaryngologist, beginning with an overview of anesthesia for children and the preoperative assessment of pediatric patients. A discussion of the perioperative management, including choice of drug and technique to match the situational demands, is described next, along with emergence from anesthesia and postoperative care. A brief overview of anesthesia for children outside the operating room and the current review of safety of anesthesia in children are also discussed. Finally, the anesthetic implications of specific diseases and the anesthetic considerations in commonly performed pediatric otolaryngology procedures are reviewed.

ANESTHESIA FOR CHILDREN

Anesthesia carries increased risk in children compared with adults, as reported more than 50 years ago.[1] Work from the 1960s correlated this increased risk with age less than 1 year,[2]

which is not surprising given the physiologic changes that occur during the first year of life. Nowadays, children younger than 1 month of age are recognized as being at the highest risk,[3] which suggests that the risk of adverse events with anesthesia induction in infants is inversely proportional to age. Respiratory control is immature in infants, the carbon dioxide (CO_2) response curve is flatter and is shifted to the right compared with the curve in older children and adults, and hypoxia may induce apnea rather than hyperventilation. Cardiovascular responses also are immature; the myocardium is poorly compliant, so cardiac output is rate dependent. Similarly, responses to anesthetic agents differ in accordance with patient age. Preterm and full-term newborns require lower concentrations of inhaled anesthetics than those needed in older children or adults. Inhalational agents may cause more depression of cardiac output in infants and may have a more profound effect on baroreflexes. Pharmacokinetic parameters of volume of distribution, hepatic clearance, and renal clearance differ profoundly from those in older children, which affects the dose of nearly every drug.

These differences in physiology and pharmacology are reflected in the types of anesthetic mishaps that tend to occur in newborns and infants. Inadequate ventilation and anesthetic overdose were the most frequent sources of anesthetic morbidity and mortality in the 1980s.[4] Changes in anesthetic practice, such as improvement in monitoring—that is, using pulse oximeters and CO_2 monitors—and use of different drugs, such as sevoflurane instead of halothane, have decreased respiratory events and shifted the number of anesthesia-related cardiac arrests in infants toward cardiovascular causes of cardiac arrest.[5,6]

Adherence to the American Society of Anesthesiologists monitoring standards[7] is basic to all anesthesia care performed by an anesthetist and has reduced the incidence of intraoperative complications.[8] The components of these standards are presented in Box 3-1. Most anesthesiologists consider pulse oximetry to be an indispensable part of the intraoperative monitoring armamentarium, although it has been difficult to document improvement in outcome as a result of this technique, even in very large series.[9,10] The use of pulse oximetry alone or in combination with capnography has reduced the relative frequency of adverse respiratory events as compared with adverse cardiovascular events, perhaps because pulse oximetry and capnography are more effective for preventing respiratory events than cardiovascular events.[11]

Box 3-1. INTRAOPERATIVE MONITORING STANDARDS

Continuous presence of qualified anesthesia personnel in the operating room
Continuous evaluation of the following:
- Oxygenation
- Inspired oxygen concentration with an oxygen analyzer
- Blood oxygenation with a pulse oximeter
- Ventilation

Qualitative assessment that include chest excursion, observation of the reservoir bag, and auscultation of breath sounds
Quantitative assessment with end-tidal CO_2 analysis
A low-pressure disconnect alarm when mechanical ventilation is used
Circulation
Continuous electrocardiogram and heart rate
Arterial blood pressure at least every 5 minutes
At least one of the following:
- Palpation of pulse
- Auscultation of heart sounds
- Intraarterial blood pressure monitoring
- Ultrasound peripheral pulse monitoring
- Pulse plethysmography or oximetry

Elective procedures should be deferred until the infant is at least 6 months to 1 year old, when anesthetic risk is likely to be lower. This is particularly true in infants who were born prematurely because they have an increased risk of postoperative apnea until approximately 55 weeks of postconceptual age. The use of trained subspecialty pediatric anesthesiologists for the care of infants and children has been suggested to be associated with improved outcomes,[12] although this is difficult to prove.[13]

Current estimates of anesthetic mortality vary widely and depend on the definition of "anesthetic death." However, mortality in healthy pediatric outpatients as the result of an anesthetic-related cause is very rare, with rates perhaps as low as 0.36 in 10,000 cases.[6,14] Accordingly, parents of otherwise healthy children can be reassured that outcomes in modern-day pediatric anesthesia are excellent.

PREOPERATIVE ASSESSMENT

Today's emphasis on cost containment has forced many changes in the practice of pediatric anesthesia. More and more patients with complex medical problems are having procedures as outpatients, which forces the preoperative contact between the anesthesiologist and the patients and family into a very few minutes. In addition, efficiency pressures have forced institutions to search for ways to maximize patient flow in surgery areas and to minimize unexpected delays or cancellations. Preoperative anesthesia clinics have developed in response to these changes.

A visit to the preanesthesia clinic before the day of surgery affords the opportunity for taking an anesthetic history, performing a physical examination, obtaining necessary laboratory tests, and informing the family about the anticipated procedures. Information conveyed includes times for restriction of oral intake to nothing by mouth (NPO status), premedication, anesthetic induction and maintenance, and postoperative pain management techniques. It also allows confirmation that all necessary paperwork, such as consent and surgical history and physical, is present in the medical record. An attending anesthesiologist is available for consultation for more complicated cases or to address specific clinical questions. Anesthetic risk can be discussed with patients and their families, with ample time allowed for them to ask questions. In addition, the clinic nurse can take this opportunity to educate the patient and family about the impending surgery and to identify fears and concerns of the parents and child.

A preoperative medical assessment includes a review of systems and the patient's response to previous anesthetics; current medications; allergies; recent illnesses, including upper respiratory infections; and family history of problems with anesthesia. The physical examination concentrates on airway anatomy and includes the mouth, jaw, teeth, and neck along with chest and heart auscultation to confirm the absence of significant cardiorespiratory disease. Laboratory tests are obtained as indicated by the child's condition and type of surgery planned, rather than as a routine component of the assessment.[15] Identification of clinical problems at a preanesthesia visit also allows appropriate evaluation or referral before surgery, thereby avoiding delay or cancellation on the day of surgery.

PREPARATION FOR ANESTHESIA

BEHAVIORAL PREPARATION

The days and hours leading up to surgery can be an anxious time for both children and parents. Fears about hospitalization are common among children, including fear of separation, pain, loss of control, and even death. Many of these fears depend on the child's age (Table 3-1).[16] Awareness of these

TABLE 3-1. Age-Specific Concerns of Children

Age	Specific Concerns
Infant (6-18 months)	Separation anxiety
Toddler (1-2 years)	Fear of strangers, loss of mastery of the environment
Preschool (2-5 years)	Separation anxiety; difficulty distinguishing reality from fantasy; fear of pain, mutilation
School age (6-12 years)	Fear of the unknown, pain, mutilation, loss of control or autonomy
Adolescent (13-18 years)	Precarious sense of self; fear of pain, mutilation, loss of control or autonomy

From Orr RJ, Lynn AM. *Curr Rev Clin Anesth* 1991;12:29.

developmental stages allows the anesthesiologist to better anticipate the needs of most children. In addition, understanding the impact of parental anxiety on a child's anxiety can help in the selection of interventions that will be helpful to both the child and the parents.

Details of the anesthetic and surgical experience should be presented in age-specific language for children of appropriate age.[17] Children who are given specific information before surgery have been shown to be less anxious than children who are given only general information.[18] Movies or videotapes can help children and parents cope with the mysteries of hospital routines.[19] Comprehensive preoperative preparation programs have been shown to be helpful for reducing preoperative anxiety and enhancing coping behavior in children.[20] Learning coping strategies and developing a better understanding of the environment in which they will receive their care may be especially important for children who will require repeated procedures. Parents also should be involved in these programs because decreasing their anxiety is likely to provide the added benefit of further decreasing the child's anxiety.[21] These efforts are important even if pharmacologic agents will be used to decrease anxiety because the combination of psychologic and pharmacologic interventions can produce synergistic effects.[22]

PREMEDICATION

The choice of premedication is based on the age and specific needs of the patient. The usual goals of premedication are to reduce anxiety and to ease separation from parents. Because most children fear needles, the oral administration of sedatives has become the standard of care at many institutions. Children who are younger than 8 months of age generally do not need premedication and separate easily from their parents. Children between 8 months and 8 years of age often experience enough preoperative anxiety that preoperative medication may be helpful. Midazolam (0.5 to 1.0 mg/kg up to a maximum of 20 mg), mixed in flavored syrup and administered orally 10 to 20 minutes beforehand has become the most popular agent for preoperative sedation for children to smooth separation from the parents and ease induction of anesthesia.[23] Midazolam usually is well tolerated, although a rare decrease in oxygen saturation or blood pressure may be observed. In addition, intravenous (IV) flumazenil (10 µg/kg, administered over 1 minute, repeated as needed for a total of 5 doses; max. 0.2 mg/dose, 1 mg total dose) can be administered to reverse adverse effects such as oversedation, paradoxic agitation, or emergence delirium.

Older children may be willing to have an IV line started preoperatively, especially if a topical anesthetic cream such as EMLA (***e***utectic ***m***ixture of ***l***ocal ***a***nesthetics) or ELA-Max is used. EMLA cream is a mixture of lidocaine and prilocaine that penetrates intact skin, and when applied for 60 minutes or more, it significantly decreases the pain associated with an IV insertion.[24,25] EMLA should be used with caution in children younger than 3 months of age or in children who are receiving other medications that may induce methemoglobin because of the risk of methemoglobinemia.[26] Plasma concentrations of lidocaine and prilocaine achieved with topical application are well below the toxic level,[26] but they may be higher in children with traumatized or inflamed skin. Another effect of EMLA that may be problematic is vasoconstrictive blanching, which makes the identification of potential IV insertion sites difficult for some practitioners. ELA-Max is an alternative to EMLA, and it contains only lidocaine in a liposomal matrix that allows for effective absorption across intact skin.[27] ELA-Max has been shown to provide equal topical anesthesia to EMLA, and it has a quicker onset of 30 minutes.[8,28] ELA-Max also produces less blanching than that seen with EMLA. If an IV line is placed, midazolam or another sedative can be titrated intravenously until the desired effect is achieved. Although the anesthetic creams are most frequently used, alternative methods for providing topical anesthesia may be standard in some institutions.

Younger children may not accept oral premedication well, which makes this method of administration as anxiety provoking as separation from the parents without any premedication. Intranasal or intramuscular administration of premedications may also be useful alternatives. Intranasal midazolam (0.2 to 0.3 mg/kg) can be easily administered using either an atomizer or simply a needleless tuberculin syringe in a patient small enough to be held or restrained.[29] Intranasal midazolam begins acting immediately after dosing and reaches peak concentrations by 14 minutes, with the advantage of increased bioavailability with minimal first-pass metabolism in the liver.[30] Intramuscular (IM) administration of premedication becomes necessary in larger children with behavioral issues that limit patient cooperation—autism, for instance. Because IM injection is technically easier than IV line placement, this technique can be achieved quickly, and the child then be allowed some time with the parent to minimize the trauma from the encounter. IM ketamine at a dose of 2 to 5 mg/kg or a combination of IM ketamine and midazolam causes sedation and dissociation from the environment, which makes the patient more approachable for procedures, anesthesia induction, and other interventions. Larger doses of ketamine administered intramuscularly (up to 10 mg/kg) can induce general anesthesia (see the following discussion).

Anticholinergic agents such as atropine (0.02 mg/kg) or glycopyrrolate (0.01 mg/kg) given intravenously at the time of induction may be useful as premedications. Anticholinergics act to ameliorate the increase in vagal tone that may occur in cases that involve airway manipulation, when halothane is used as an inhalational agent, or in patients with copious airway secretions. Although halothane is no longer available for clinical use in the United States, it is still used in developing countries. Halothane may cause bradycardia and a significant reduction in cardiac output, particularly in children younger than 6 months of age; in younger patients, the routine use of anticholinergic agents with halothane can prevent these changes.[31] Anticholinergics are also a useful adjunctive premedication to ketamine, which increases salivary secretions.

NOTHING-BY-MOUTH ORDERS

To minimize the risk of the aspiration of gastric contents during the induction of anesthesia, an adequate period of fasting must be maintained. Traditionally, both food and fluids have been withheld after the midnight before surgery. However, a

TABLE 3-2. Recommended Preoperative Fasting Guidelines for Children

Ingested Material	Minimum Fasting Period (hr)
Clear liquids	2
Breast milk	4
Infant formula and nonhuman milk	6
Light meal	6
Fatty food and meat	8

Modified from Practice guidelines for preoperative fasting and the use of pharmacologic agents to reduce the risk of pulmonary aspiration: application to healthy patients undergoing elective procedures: An updated report by the American Society of Anesthesiologists Committee on Standards and Practice Parameters. *Anesthesiology* 2011; 114:495-511.

prolonged period of fasting is uncomfortable for children, unpleasant for the family, and risks hypovolemia. It has recently been recognized that the administration of clear liquids to children up to 2 to 3 hours before induction does not increase gastric residual volume or acidity compared with a more prolonged fast.[32,33] In addition, morbidity and mortality rates for aspiration of gastric contents in children are exceedingly low.[34] Thus, fasting guidelines have been liberalized in recent years (Table 3-2). Some debate surrounds whether breast milk should be considered a solid or clear liquid, with many institutions allowing breastfeeding within the same guidelines as clear liquids. Nevertheless, in giving these instructions to parents, it is important to emphasize the safety issues involved to maximize compliance and avoid unnecessary delays in the start of the procedure.

PARENTAL PRESENCE

With increasing frequency, parents are expressing a desire to be present at the time of their child's anesthetic induction.[35] Although parents' motives vary, most hope that the child's anxiety will be lessened by the emotional comfort of their presence. Some hospitals have induction areas outside of the sterile envelope of the OR. If not, parents can be dressed in cover gowns and escorted into and out of the OR for the induction.

Studies to examine the impact of parental presence have had variable results: some show no significant impact,[36] and some show a decrease in patient anxiety,[37,38] and still others show an increase in patient anxiety.[39] Parents with a high anxiety level seem to have a negative impact on their child during induction, whereas calm parents seem to have a positive impact.[40] One survey reported that a majority of anesthesiologists are in favor of the practice of having parents present during induction,[41] and many hospitals tend to support this request as a mechanism to improve patient and parent satisfaction. Thus it is likely that the trend will continue. Parents should be thoroughly educated about what to expect and how they can best help their child; they must agree to leave the induction area immediately when instructed to do so, and one member of the care team must be identified to help them during the induction. Highly anxious parents should be encouraged not to participate.

ANESTHETIC INDUCTION

Anesthesia can be induced by mask inhalation of a potent anesthetic agent in oxygen with or without nitrous oxide (inhalation induction), by injection of a sedative-hypnotic agent through an IV catheter (IV induction), or by intramuscular injection. As much as possible, children are given a choice of technique; younger children tend to fear needles and prefer an inhalation induction, whereas older children and teenagers usually accept IV induction. Inhalation induction will take longer in teen-aged and older patients than in younger patients because of age-related physiologic differences. The prolonged time to effect must be taken into consideration if an inhalation induction is selected in older children, but it does not preclude such an induction. Certain clinical factors (e.g., obesity, severe gastroesophageal reflux, full stomach) also may incline the choice of induction technique toward IV.

INHALATION INDUCTION

Sevoflurane is the inhalational induction agent of choice because of its minimal pungency, fewer cardiovascular side effects, and rapid uptake. A small amount of pleasantly scented liquid (e.g., bubble gum flavoring) applied to the inside of the mask disguises the odor of the anesthetic agent and improves the patient's acceptance of the technique. Many anesthesiologists will begin the induction with the patient breathing 50% to 70% nitrous oxide, which is odorless, before introducing sevoflurane, thus making the addition of the sevoflurane less distressing. Noise in the room should be kept to a minimum during anesthesia induction, and the anesthesiologist often tells the child an entertaining story to direct the child's thoughts away from the reality of the situation. As the concentration of the anesthetic agent is increased, most children lose consciousness in less than 1 minute. The parents, if present, can be ushered out at this point, and an IV catheter can then be placed.

INTRAVENOUS INDUCTION

Intravenous induction requires the presence of an IV catheter and thus is reserved for older children or teenagers, children with a preexisting IV catheter, children who need a rapid-sequence induction because of a full stomach, or children who object to an inhalation induction. Owing to slower uptake of inhaled anesthetic agents, inhalational induction takes much longer in older, heavier children, and it poses a consequently higher risk for laryngospasm and bronchospasm. An IV induction is therefore also the preferable method in this patient group.

The most common agent for IV induction is propofol, an alkylphenol formulated as a 1% solution in a white soybean oil, egg lecithin, and glycerol emulsion. Propofol is highly lipophilic and rapidly redistributes throughout body compartments, with a distribution half-life of approximately 2 minutes and an elimination half-life of approximately 30 minutes. The dose for the induction of anesthesia varies with age[42]; infants 1 to 6 months of age require approximately 3 mg/kg, whereas older children require approximately 2.5 mg/kg. Propofol is the IV induction agent of choice for outpatient procedures because recovery is rapid, and the patient is clear-headed with minimal associated nausea and vomiting. The major disadvantage is pain on injection, particularly into small veins.[43] Lidocaine (0.5 to 1.0 mg/kg) administered intravenously before or during propofol injection may decrease the incidence of pain. Propofol may cause hypotension, apnea, and desaturation; it can also be used for anesthesia maintenance as an IV infusion of 100 to 200 μg/kg/min. This technique can be very useful for patients who have a history of severe postoperative nausea or who are undergoing procedures in which postoperative nausea is especially common (e.g., tympanoplasty). Many pediatric anesthesiologists use a propofol infusion to provide anesthesia during bronchoscopy because it provides a reliable,

stable method of administering anesthesia with spontaneous ventilation when the airway is shared with the surgeon, and it decreases the exposure of the surgeon to unscavenged anesthetic gases. An alternative IV agent for induction, thiopental, is an ultrashort-acting barbiturate that is no longer available for use in the United States

Etomidate, a short-acting nonbarbiturate, is also used as an IV induction drug. It is an imidazole-derived, steroid-based, potent sedative-hypnotic agent without analgesic properties. Etomidate has a rapid onset of action (5 to 15 seconds) with a peak effect at 60 seconds, and a short duration of action (3 to 5 minutes) that is terminated by redistribution.[44] Its primary advantage is that it has minimal cardiac depressant effects or hemodynamic impact.[45-48] Etomidate also reduces the intracranial pressure, the cerebral blood flow, and cerebral metabolic rate but has little effect on ventilation and smooth muscle tone.[49,50] Bolus injection of etomidate does not release histamine. These features make it an extremely useful agent in critically ill children who have an unstable cardiovascular status and those with a head injury, hypovolemia, and trauma.[46]

The recommended induction dose of etomidate in healthy children is 0.2 to 0.3 mg/kg, depending on cardiovascular status.[51] Metabolism is via the liver. Etomidate has a number of side effects that preclude regular use. Like propofol, it burns with injection, an effect that is ameliorated with the newer formulation that dissolves the drug in a fat emulsion.[52] Myoclonic movements are also common with its use[53] but may be attenuated by prior opioid administration. Adrenal suppression occurs with multiple doses or infusion, and steroid supplementation may be required.[54-58]

RAPID-SEQUENCE INDUCTION

Patients with a full stomach are at increased risk for the aspiration of stomach contents during the induction of anesthesia. Patients who require full-stomach precautions include those who have not fasted for an adequate period, those with gastrointestinal obstruction, trauma patients whose gastric motility is depressed, or those who are at a mechanical predisposition for aspiration (e.g., patients with ascites). It is mandatory that these patients have an IV induction. Patients are preoxygenated with 100% oxygen by mask and are then given an induction dose of a hypnotic and a rapid-acting muscle relaxant—either succinylcholine (2 mg/kg) or rocuronium (1.2 mg/kg). Application of manual pressure over the cricoid, known as the *Sellick maneuver,* can be performed in an attempt to seal the esophagus and to prevent passive regurgitation.[59] Pressure is not released until the endotracheal tube is in place, proper position is confirmed, and the cuff, if necessary, is inflated. At the end of the procedure, these patients should be allowed to emerge completely before extubation to ensure adequate airway protection.

INTRAMUSCULAR INDUCTION

Ketamine is a derivative of phencyclidine with potent analgesic and amnestic properties. Anesthetic induction can be achieved with the recommended dose of 1 to 3 mg/kg intravenously or 5 to 10 mg/kg intramuscularly. As noted previously, ketamine is very useful as an intramuscular induction agent in children who are combative or unable to cooperate with a standard inhalation or IV induction, often because of intellectual disabilities and behavioral disorders. Ketamine is an excellent choice for anesthetic induction in hypovolemic patients because of its sympathomimetic properties; it rarely causes hypotension in these patients.

Ketamine tends to produce copious secretions and should be accompanied by the administration of atropine or glycopyrrolate. Nystagmus and diplopia are common side effects and will resolve as the drug is cleared from the patient's circulation. Intraoperative and postoperative dreams and hallucinations have been reported,[60] more often in older than in younger children and less often when other sedative-hypnotic agents are used in conjunction with ketamine. If this agent is given preoperatively with the parents still present, they should be informed to expect these side effects.

ANESTHESIA MAINTENANCE

A variety of agents may be used to maintain anesthesia in the pediatric population. The otolaryngologist should be familiar with the effects of each of these.

INHALATIONAL AGENTS

Nitrous Oxide

Nitrous oxide is a widely used inhalational induction agent in children. Its popularity is a result of its low solubility, which results in rapid uptake and distribution. Nitrous oxide also is odorless and does not cause cardiovascular depression. Although nitrous oxide allows the more rapid uptake of lower concentrations of other anesthetic agents, nitrous oxide itself is not a very potent anesthetic agent. Consequently, it must be delivered in high concentrations to have an analgesic and hypnotic effect, thereby limiting the concentration of oxygen that can be delivered. It is not useful for patients who require high concentrations of oxygen and should be avoided in patients with pulmonary hypertension because it may aggravate it. Because nitrous oxide is less potent and is implicated in an increased incidence of postoperative nausea and vomiting (PONV), its use for anesthetic maintenance has decreased. A 34-fold difference exists in the blood-gas coefficients of nitrogen (0.013) and nitrous oxide (0.46); thus, nitrous oxide will enter air-filled cavities faster than nitrogen can leave. In a fixed cavity such as the middle ear, the result is an increase in pressure. During tympanoplasty, the middle ear pressure generated by nitrous oxide can lift off the tympanic membrane graft, and it is therefore usually avoided entirely during those procedures.[61] Nitrous oxide use also may present a hazard for patients with previous reconstructive middle ear surgery.[62] Finally, nitrous oxide is flammable and should be avoided when fire is a risk, such as during tonsillectomy or with use of a laser in the airway.

Sevoflurane

Sevoflurane has completely taken the place of halothane in pediatric institutions in the United States. It is a valuable addition to the anesthesiologist's armamentarium for several reasons. First, it is less pungent than halothane, and it is even better tolerated during inhalation induction. Second, it has a blood-gas partition coefficient similar to that of nitrous oxide, so induction and emergence times are shorter than with halothane. Third, it has fewer cardiovascular effects than halothane.

Sevoflurane has several disadvantages. Its offset is so rapid that without adjunctive administration of narcotics, the perception of and response to pain may be accentuated.[63] There are reports in the literature of a significant incidence of emergence excitement associated with cases in which sevoflurane was used.[64] For these reasons, sevoflurane has assumed a role as an induction agent and a maintenance agent for very short procedures, but it is often replaced by isoflurane or desflurane after induction, depending on case type and anesthesiologist preference. Like all halogenated inhalational agents, it is a potential trigger agent for malignant hyperthermia and should be avoided in susceptible patients.

Isoflurane

Isoflurane has been used for many years as a standard maintenance inhalational agent for children and adults. In practice, it offers no clear advantage for the pediatric otolaryngology patient. It is more pungent and not nearly as well accepted by children during inhalation induction as sevoflurane. Isoflurane does increase, rather than decrease, heart rate. It also causes decreases in blood pressure via peripheral vasodilation. [65]

Desflurane

Desflurane is the newest inhalational agent to be introduced into clinical practice. Its principal advantage is its quicker time to recovery compared with sevoflurane.[63] It appears to be well tolerated as a maintenance anesthetic because it provides stable hemodynamics and respiratory parameters during its use. Similar to sevoflurane, desflurane has been associated with increased agitation and emergence delirium.[43] However, this effect does not seem to occur frequently enough to justify avoiding its use in children.

Desflurane does have an irritant effect on the airway that makes it contraindicated for inhalational induction or for use during airway procedures such as bronchoscopy. However, many centers have adopted a practice of inducing anesthesia with sevoflurane and then switching to desflurane to take advantage of its quick recovery time while avoiding its airway effects during induction. It also has been suggested that this agent may be particularly useful in neonates and formerly premature babies, in whom residual anesthesia effects may increase the risk of apnea postoperatively.[66]

INTRAVENOUS AGENTS

Intravenous agents include the various opioids and other agents. The otolaryngologists should be familiar with their effects in the pediatric population.

Opioids

Fentanyl. Fentanyl is a potent synthetic opioid and is the most commonly used supplement to anesthesia in children. It has a relatively short duration of action (1 to 2 hr), and is about 100 times as potent as morphine. Fentanyl is ideal for patients who need short-term anesthesia and analgesia. It is useful for patients who require rapid recovery to baseline ventilatory function and respiratory drive, such as patients having same-day surgery, neurosurgical procedures, and airway instrumentation. IV infusion doses that range from 0.5 to 4 mμg/kg/hr infusions provide adequate analgesia and sedation and can be used as adjuncts for maintenance of anesthesia. Fentanyl doses in excess of 100 μg/kg have been given to cardiac surgery patients with minimal cardiovascular depression.[67] An initial peripheral vasodilation occurs, but tachyphylaxis to this side effect occurs with additional dosing. Preterm and full-term newborns have variable and prolonged clearance, probably related to reduced hepatic blood flow.[68] In addition, they are extremely sensitive to the effects of fentanyl on chest wall rigidity. In neonatal patients who are not intubated, fentanyl should be given only in small increments.

The effect of low-dose fentanyl is terminated largely by redistribution and thereby results in a rapid reduction in clinical effect. However, higher or repeated doses will result in drug accumulation, because fentanyl is highly lipophilic. Clearance then becomes dependent on metabolism,[69] and the clinical effect, including respiratory depression, may last for hours. Chest wall rigidity has been reported after rapid fentanyl administration, although the etiology of this problem is unclear. Bradycardia may occur as a result of increased vagal tone, especially when given with other agents that may have a similar effect.

Morphine Sulfate. Overall, morphine is the most frequently used opioid in children. It can be used in the OR to supplement inhalational agents and to provide postoperative analgesia. The usual IV dose is 0.05 to 0.1 mg/kg as part of a balanced anesthetic technique, although higher doses may be used for a narcotic-based technique. The dose may need to be reduced in critically ill children or young infants. The half-life after IV administration is approximately 3 hours in older children but is significantly longer in infants as a result of diminished clearance.[70] The major side effect is respiratory depression, which results in diminished minute ventilation with a greater effect on respiratory rate than tidal volume. Many believe that newborns are at higher risk than older children for respiratory depression. Infusions of morphine at rates of 10 to 30 μg/kg/hr have been described in small infants without significant respiratory depression.[71] Appropriate monitoring for apnea should occur in premature infants and those neonates with a history of apnea and bradycardia. Histamine release is also common with the administration of morphine, and it most commonly results in a localized or generalized rash. Bronchospasm and hypotension have been reported, but they are much less common.

Hydromorphone. Hydromorphone has become a commonly used alternative to morphine. It is approximately five to seven times more potent than morphine, and the usual IV dose ranges from 0.015 to 0.02 mg/kg. Its half-life is similar to that of morphine, and it has a similar duration of action. Hydromorphone may be useful in patients with renal failure because its metabolic by-products are less active than those of morphine. In addition, hydromorphone may be a good alternative for those patients who suffer side effects (e.g., itching, nausea, hallucinations) with morphine. As with other opioids, respiratory depression is the most concerning side effect, but it is rare with appropriate dosing.

Meperidine. The use of meperidine has dramatically decreased because of its unique side effect profile and the availability of equianalgesic alternatives. The principal metabolite of meperidine is normeperidine, a compound that can cause neurologic excitation manifested as tremors, irritability, or seizures. This side effect is especially pronounced with prolonged use or in patients with hepatic or renal dysfunction. Meperidine's superiority over other opioid agonists in the treatment of postoperative shivering probably is related to its κ-receptor activity, and it is the one reason that it has not become entirely obsolete.

Alfentanil. As a short-acting analog of fentanyl, alfentanil is commonly used for short outpatient procedures. It is approximately one fourth as potent as fentanyl and has one third the duration of action. It has a very fast onset of action (1 min) and a short elimination half-time (1.5 hr). It is less lipophilic than fentanyl, and the dose range is 10 to 20 μg/kg given intravenously. Renal failure does not alter the clearance of alfentanil. Beside the opioid-related side effects, thorax rigidity and bradycardia may be seen.

Remifentanil. Remifentanil has the same analgesic potency as that of fentanyl. The key feature of remifentanil is the ester side chain hydrolysis by blood and tissue esterases, which results in rapid metabolism (elimination half-time is 10 to 20 min). This results in zero order kinetics, meaning that its offset of action is 10 to 20 minutes after discontinuation of the infusion regardless of the duration of the infusion; in other words, it does not accumulate. Remifentanil does have a rapid onset, and because of its short duration of action, it usually needs to be administered as a continuous IV infusion at the rate of 0.05 to 1 μg/kg/min for neonates and up to 2 μg/kg/min for older

TABLE 3-3. Comparative Opioid Potencies, Onset, and Duration of Action

Drug	Order of Potency	Onset of Action (min)	Duration of Action	Routes of Administration	Drug Doses	
					Adjunct to GA (IV)	Postop. Pain (IV)
Fentanyl	150×	6.8	Shorter than morphine if single dose, (but due to redistribution is most prolonged if continuous IV)	IV, IM, intranasal, OTFC, transdermal patch	2 to 10 μg/kg	1 μg/kg
Sufentanil	1000 to 1500×	6.2	Similar to fentanyl	IV, intranasal	0.5 to 1.5 μg/kg/min	—
Remifentanil	300×	1-2	Very short (6 to 8 min offset)	IV	0.025 to 2 μg/kg/min	—
Alfentanil	20 to 40×	1.5	One third of fentanyl	IV	0.5 to 3 μg/kg/min	—
Morphine	1	15-30	3 to 4 h	IV, IM, SC, PO	0.05 to 0.1 mg/kg	0.05 to 0.1 mg/kg
Hydromorphone	5 to 10×	10-20	2 to 4 h	IV, PO	0.015 to .020 mg/kg	0.005 to 0.015 mg/kg
Methadone	1	15-30	12 to 50 h	IV, PO	0.1 mg/kg	0.05 to 0.1 mg/kg

These are general guidelines for drug therapies to control pain in children. Doses are for opioid-naive patients. For infants and children younger than 6 months, start at a lower dose than suggested and titrate to effect to achieve the desired result. Caution should be used in dosing.
GA, general anesthesia; IM, intramuscular; IV, intravenous; OTFC, oral transmucosal fentanyl citrate (lollipop); PO, by mouth; SC, subcutaneous.

children. Pharmacokinetic parameters of remifentanil are unchanged by hepatic or renal disease. Disadvantages are the high cost and the lack of postoperative analgesia, which can be overcome with application of longer-acting opioids at the end of surgery (Table 3-3).

Ketamine

Ketamine is a versatile nonbarbiturate drug that dissociates the thalamus from the limbic cortex, thus producing a cataleptic state of dissociative anesthesia. The patient is noncommunicative, and the eyes remain open with a slow nystagmic gaze. Varying degrees of hypertonic and purposeful movements occur independent of stimuli.

Ketamine rapidly induces general anesthesia within 0.5 to 2.0 minutes when given intravenously in doses of 0.5 to 2 mg/kg. Further anesthesia can be maintained with infusions of 0.01 to 0.1 mg/kg/min. It is also well absorbed following oral (3 to 6 mg/kg),[72,73] rectal (4 to 6 mg/kg),[74] nasal (4 to 6 mg/kg),[75] and intramuscular administration. The onset of action may be delayed with these routes from 10 to 35 minutes. Administration of subanesthetic doses produces intense analgesia, amnesia, and altered consciousness. These properties make ketamine an ideal agent for painful procedures, particularly in children with congenital heart disease, asthma, trauma, hemodynamic instability, burns, and poor IV access.

Ketamine increases arterial blood pressure, heart rate, and cardiac output. It also increases cerebral oxygen consumption, cerebral blood flow, and intracranial pressure. It does not produce significant respiratory depression in low doses, and the upper airway and laryngeal airway reflexes remain intact, and the ventilatory response to hypoxia and hypercarbia are maintained. Ketamine further increases circulating catecholamines and causes the relaxation of tracheal, bronchial, and alveolar smooth muscle, making it the IV induction agent of choice in patients with reactive airways.[76] Additionally, ketamine also raises the intraocular pressure and is associated with increased incidence of emergence delirium and nausea and vomiting. It is metabolized extensively by hepatic microsomal enzymes and its active metabolite, norketamine, may contribute to the prolonged effects of ketamine.

Dexmedetomidine

Dexmedetomidine is the most recently approved agent by the Food and Drug Administration (FDA) for anesthesia and sedation. It is a selective α-2 agonist with sedative, anxiolytic, and analgesic properties.[77,78] It has a short distribution half-life of 6 minutes[79] and an elimination half-life of 2 hours.[80] It is metabolized in the liver, eliminated by the kidneys, and is 93% protein bound.

Dexmedetomidine is a centrally acting agent that produces sedation and anxiolysis via activation of inhibitory γ-aminobutyric acid neurons. Specifically, it decreases central sympathetic activity, and increases central parasympathetic outflow from the locus ceruleus.[80] Being highly lipid-soluble, it quickly crosses the blood-brain barrier. In a dose-dependent manner, it decreases heart rate and mean arterial pressure. The effects of sympatholysis cause peripheral vasodilation and hypotension at lower doses and vasoconstriction and hypertension at higher doses.[81]

Dexmedetomidine provides respiratory stability and minimal ventilatory depression, which distinguishes it from opioids, benzodiazepines, and other hypnotic agents.[78,82-87] In spontaneously ventilating patients, the respiratory rate, carbon dioxide tension, and oxygen saturation are generally maintained[88-90] despite some blunting of the carbon dioxide response curve.[91,92] Analgesia is another useful property of the drug, which exerts its effect on the dorsal horn of the spinal cord and the supraspinal and peripheral nerves,[77] thereby inhibiting the release of substance P. When used with other opioids, it potentiates their analgesic effects.[93] Finally, in decreasing sympathetic tone, dexmedetomidine attenuates the stress response. IV dosing is by loading 1 μg/kg infused over 10 minutes to minimize hypertensive effects,[94,95] followed by infusing 0.5 to 1 μg/kg/hr.[78,89] Additional possible routes of administration include intramuscular, intranasal, buccal, and oral.

For a relatively new drug, dexmedetomidine has found a plethora of uses. Given its synergy with hypnotics and opioids, it reduces the overall doses of sedation drugs and decreases long-term exposure in mechanically ventilated patients. In neonates and children, dexmedetomidine is frequently used for invasive and noninvasive procedural sedation and as an anesthetic adjunct during surgery. Its analgesic properties make it useful during airway stimulating procedures such as debridement of burns, bronchoscopy, laryngoscopy, and tonsillectomy among others. Dexmedetomidine provides an interesting quality of sedation that permits arousal with gentle stimulation.[90] This subtle wake-up allows for an agitation-free emergence from anesthesia.[96] In the treatment of anesthetic

emergence delirium, a dose of 0.3 μg/kg has been shown to be very effective.[97] In patients with opioid tolerance, it facilitates in the weaning of opioids relatively quickly with minimal hemodynamic consequences.[98] Other indications such as treatment of shivering, chronic regional pain syndrome, and withdrawal continue to emerge.[99-102] The side effect of bradycardia makes it contraindicated for patients on digoxin[103] and unsuitable as a sedative for electrophysiologic studies. It should be used with caution in children with preexisting bradycardia, atrioventricular conduction defects, hypertension, and decreased cardiac output.[104,105]

Muscle Relaxants

Succinylcholine. Succinylcholine is the only depolarizing muscle relaxant. With the advent of intermediate-acting nondepolarizing relaxants, the use of succinylcholine for routine surgical procedures is declining, primarily as a result of side effects that relate to its depolarizing mode of action (see below). However, it remains the most rapid acting of all muscle relaxants, and it is still indicated for use in rapid-sequence inductions and in the treatment of laryngospasm. IV administration of 1.5 to 2.0 mg/kg achieves 95% twitch depression in 40 seconds[106] and results in excellent intubating conditions. Succinylcholine also can be given intramuscularly (4 to 5 mg/kg) if the IV route is unavailable, although the clinical effect is delayed in onset, depending on perfusion to the area of deposition.

The side effects of succinylcholine are numerous. Some degree of increase in masseter muscle tone is common and, in some cases, is extreme enough to mimic true trismus.[107] This is an important distinction to make because as many as 50% of patients with trismus after succinylcholine are biopsy positive for susceptibility to malignant hyperthermia.[108] Succinylcholine can also cause bradycardia as a result of an increase in vagal tone, an effect that is especially prominent in younger children and infants. The decline in heart rate usually is transient; if persistent, it is responsive to IV atropine, and occasionally, asystole is seen. Hyperkalemia can be seen as a result of depolarization of the myoneural junction, and increases of serum potassium of 0.5 mEq/L occur even in normal children.[109] Life-threatening hyperkalemia after succinylcholine administration can occur in children with burns, tetanus, paraplegia, encephalitis, crush injuries, and neuromuscular disease.[109] Myoglobinemia occurs in approximately 40% of children anesthetized with halothane who are given succinylcholine. This effect can be partially attenuated with a previous dose of a nondepolarizing agent to prevent fasciculations. A series of cardiac arrests were reported in boys who were given succinylcholine, with subsequent development of massive muscle breakdown and potassium release. The presumed etiology was previously undiagnosed muscular dystrophies.[110] In 1994, in response to these reports, the manufacturer placed a warning on the package insert cautioning the practitioner about the routine use of succinylcholine. As a result, many pediatric anesthesiologists have abandoned the routine use of succinylcholine except for rapid-sequence inductions and to treat laryngospasm.

Of note, 90% of an IV dose of succinylcholine is rapidly hydrolyzed in the plasma by pseudocholinesterase. Patients with deficient or reduced levels of pseudocholinesterase exhibit a prolonged effect from succinylcholine.

Nondepolarizing Muscle Relaxants. A variety of nondepolarizing muscle relaxants are available. These agents vary in dose, speed of onset, duration of action, and side effects. As shown in Table 3-4, the pharmacologic properties of these drugs may be different in infants from those in older children. The choice generally is based on the duration of the surgical procedure. As noted, rocuronium has been shown to produce good intubating conditions but with a slightly longer onset of action (less than 1 min) than with succinylcholine (less than 30 sec).

TABLE 3-4. Intubation Doses, Onset Times, and Recovery Times for Commonly Used Muscle Relaxants

	Intubating Dose (mg/kg)		Onset Times* (min)	Recovery Times in Infants (min)
	Adults	Children		
Atracurium	0.5	0.5	2	40-60
Cisatracurium	0.1	0.1-0.2	2.5	53
Vecuronium	0.07-0.1	0.1	2.4	35
Rocuronium	0.5-1.0	0.6-1.2	1.3	42
Pancuronium	0.1	0.1	2.5	50

*Time to maximal blockade.

REVERSAL AGENTS OF NEUROMUSCULAR RELAXANTS

SUGAMMADEX

Sugammadex is a biologically inactive neuromuscular reversal drug with a radically new mechanism of action. It is a modified γ-cyclodextrin and exerts its effect by forming very tight water-soluble complexes at a 1:1 ratio with steroidal neuromuscular blocking drugs like rocuronium, pancuronium, and vecuronium.[111,112] It then directly removes these complexes from the neuromuscular junction.[113,114]

Prior to sugammadex, only two options—succinylcholine and rocuronium—were available to facilitate tracheal intubation during a rapid-sequence induction. Relaxation with succinylcholine carries concomitant risks, including that of hyperkalemic arrest.[115] However, a high dose of rocuronium is needed for rapid-sequence inductions, and it cannot be immediately reversed. In the case of a difficult airway, in which the patient cannot be ventilated after administration of rocuronium, there would be no way to back out of the anesthetic and return the patient to spontaneous ventilation in a life-sustaining amount of time. At least 30 minutes is required before neostigmine can be administered, and anticholinergics must also be administered because neostigmine is an anticholinesterase inhibitor and indirectly increases the activity of the cholinergic system.[113,114] Thus, the patient must be subjected to the hemodynamic side effects of both neostigmine and anticholinergics.

During rocuronium-induced neuromuscular blockade, the IV administration of sugammadex creates a concentration gradient that favors the movement of rocuronium molecules from the neuromuscular junction back into the plasma. This results in a fast recovery of neuromuscular function and rapid termination of the block.[116,117] Sugammadex administered 3 to 5 minutes after rapid-sequence induction with rocuronium produces markedly faster recovery than placebo or spontaneous recovery from succinylcholine-induced block.[118,119]

The recommended dose of sugammadex depends on the level of neuromuscular blockade to be reversed. Different doses (2, 4, and 16 mg/kg) are available to reverse different levels of neuromuscular blocks.[120] A dose of 2 mg/kg is indicated for the reversal of shallow neuromuscular blockade, whereas 4 mg/kg is indicated for the reversal of profound neuromuscular blockade by rocuronium. Reversal and return of train of four of 0.9 is likely within 1.5 minutes of the shallow block and within 3.3 minutes of the profound block. Immediate reversal of neuromuscular blockade by high-dose rocuronium requires sugammadex at a dose of 16 mg/kg, and return of train of four of 0.9 is likely within 5.7 to 6.7 minutes.[121] The speed of recovery is

dose dependent, and the reversal is sustained without any signs of return of paralysis.[122] Sugammadex 4 mg/kg IV reverses rocuronium neuromuscular blockade more rapidly than neostigmine or edrophonium.[123]

Sugammadex is safe and well tolerated, and it does not bind to plasma proteins.[124,125] It is excreted through the kidneys and enhances the renal excretion of rocuronium. Its efficacy as an antagonist does not appear to rely on renal excretion of the cyclodextrin-relaxant complex.

The most frequently reported and anesthetically concerning side effects have been hypersensitivity allergic reaction,[125] anaphylaxis,[126] hypotension, and prolongation of the corrected QT interval.[124,127,128] In view of the potential of sugammadex to reverse even a profound neuromuscular block, as well as its favorable safety profile, this agent may fulfill the criteria of an ideal reversal agent for rocuronium.[129]

TOTAL INTRAVENOUS ANESTHESIA

Total intravenous anesthesia (TIVA) is a continuous titration of IV drugs for maintenance of anesthesia. Drugs are used in some combination of propofol, remifentanyl, alfentanil, fentanyl, ketamine, and midazolam. This technique provides safe anesthesia in the malignant hyperthermia–susceptible patient. It is often used in patients with severe PONV and is also the technique of choice in some airway cases.

EMERGENCE FROM ANESTHESIA

The decision of whether to extubate a patient while he or she is awake or under surgical planes of anesthesia (deep extubation) depends on a number of factors. Deep extubations are smoother and facilitate rapid turnover. Considerations in making this decision include the patient's age, airway anatomy, underlying disease, the presence of blood or secretions in the airway at the time of extubation, the presence of a full stomach preoperatively, the ability of the postanesthesia care unit staff to manage an obstructed airway, and the immediate availability of an individual skilled in reintubation should it be required. Deep extubation is preferred in patients with asthma or in those for whom coughing would be contraindicated. Factors that favor awake extubation include a full stomach at the start of the procedure, age younger than 6 months, difficult airway anatomy for either a mask or intubation, copious blood or secretions in the airway, and a predisposition to apnea. Systematic comparisons of awake versus deep extubation have revealed that patients extubated while awake have lower oxygen saturations, probably because of coughing during emergence, but there is no difference in overall morbidity or mortality.[130,131] Either technique can be performed safely; the anesthesiologist should have both approaches in his or her armamentarium and, using clinical judgment, should choose the one that best fits the situation.

Emergence delirium is an abnormal condition of a dissociated state of consciousness and increased psychomotor activity that occurs after administration of inhalational anesthetics in children.[132,133] Presentation ranges from involuntary jerking movements with muscular hypertonia of the trunk and extremities[134,135] to thrashing, disorientation, crying, screaming, inconsolability, and inability to recognize the surroundings.[136,137] The incidence is approximately 13%, although there is quite a range in different studies. It is more frequent in younger children.[132,138]

The exact etiology of the restlessness and agitation is still uncertain; thus no preventative treatment has been found. Although emergence delirium is an unpleasant condition, it is generally self-limiting within 5 to 15 minutes.[139,140] Treatment with sedatives or analgesics may potentially prolong recovery and time until discharge of the patient.[141] Variable success has been reported with analgesics,[142] opioids,[143] benzodiazepines,[138,144] clonidine,[135,145] ketorolac,[142] midazolam,[146] propofol, thiopental, and dexmedetomidine.[133] Although no clinical evidence suggests that emergence delirium has long-term behavioral effects, it is a cause of dissatisfaction for most parents and caregivers.

POSTOPERATIVE MANAGEMENT

NAUSEA AND VOMITING

Nausea and vomiting can result in an unpleasant recovery from surgery and anesthesia, a delayed discharge from the postanesthesia recovery unit, and an increase in overall cost. In the pediatric population, the incidence is variable and depends on the type of surgery, the anesthetic used, gender, age, history of motion sickness, and adequacy of hydration.[147] Although the overall incidence is lower in children younger than 2 years of age, children older than 3 years have an average vomiting incidence of more than 40%. Procedures such as tonsillectomy and middle ear surgery may be associated with an incidence of vomiting as high as 70%. The use of narcotics, halogenated inhalational agents, and perhaps nitrous oxide increases the risk of nausea and vomiting. By contrast, propofol reduces the incidence of vomiting compared with halothane in children undergoing adenotonsillectomy.[16]

Consequently, antiemetics often are given prophylactically for children at risk for PONV, and a multimodal approach has been shown to be the most effective one.[148] The use of IV ondansetron (50 to 100 μg/kg up to 4 mg), IV dexamethasone (150 to 500 μg/kg up to 8 mg), and IV droperidol (50 to 75 μg/kg up to 1.25 mg) is evidence-based as a prophylaxis for patients at risk for PONV and for treatment of patients with PONV.

Serotonin (5-hydroxytryptamine$_3$) receptor antagonists such as ondansetron have shown efficacy and safety in prophylaxis and treatment of PONV, with a greater antiemetic than antinausea effect.[149] When prophylaxis with a serotonin receptor antagonists fails, rescue therapy with serotonin receptor antagonists is not recommended within the first 6 hours of surgery because it confers no additional benefit.[150]

Another drug that has been shown to help control PONV during the 24 hours after surgery is dexamethasone. Children who receive a single intraoperative dose of dexamethasone were two times less likely to vomit during the first 24 hours than children who received placebo. In other words, routine use in four children would be expected to result in posttonsillectomy emesis experienced by one fewer patient. Additionally, children who received dexamethasone were more likely to advance to a soft to solid diet on posttonsillectomy day 1 than were those children who received placebo.[151]

Droperidol is an antidopaminergic antiemetic. Although effective in the treatment of PONV, its sedating and extrapyramidal side effects should be considered.[152] In 2001, the FDA issued a black box warning against the use of this drug because of concerns of serious cardiac arrhythmias secondary to QT prolongation. An overwhelming majority of anesthesiologists surveyed (92%) did not consider the ban warranted because torsades de pointes was seen mostly when the drug was administered in very high doses or when there were confounding factors. Still, prudence would limit its use to cases in which patients do not have underlying cardiac disease, and when other classes of antiemetics have been exhausted; the maximum total pediatric dose is less than 100 μg/kg or 2.5 mg total.[153]

Finally, an inexpensive but effective method of reducing the incidence of nausea and vomiting is to withhold oral fluids

during the postoperative period until the child asks for them because forced oral fluids increase the incidence of vomiting and delay discharge.[147,154,155] This can be safely done by generously hydrating the patient with IV fluids intraoperatively. Nontraditional treatment methods such as acupressure are being studied and thus far confirm their effectiveness in the prevention of PONV; they are useful adjuncts, especially in view of the total absence of side effects.[156]

POSTINTUBATION CROUP

In children up to approximately 8 years of age, the subglottis is the narrowest portion of the airway, and it may be the source of postoperative airway obstruction secondary to mucosal swelling. A reduction in the reported incidence of postintubation croup from 1%[157] to 0.1%[158] may be obtained with use of proper-sized, non–tissue-reactive tubes that allow an air leak around the tube at 25 cm H_2O or less.

Racemic epinephrine (0.5 mL of 2.25% in 2.5 to 3.0 mL of normal saline for children weighing 15 kg or more) nebulized in oxygen is the management of choice for postintubation croup; the effect is thought to result from the vasoconstriction of the subglottic mucosa. (The dose varies and ranges from 0.2 mL for infants weighing less than 5 kg, 0.3 mL for 5- to 10-kg infants, and 0.4 mL for 10- to 15-kg infants.) Because symptoms can recur within 2 to 4 hours, the child should be observed for 4 hours after treatment before being sent home. If frequent and repeated treatments are necessary, the patient should be admitted. Dexamethasone (0.3 to 0.5 mg/kg) also may be used,[159] although its effect is slow in onset, if it occurs at all.

PAIN MANAGEMENT

The theory that children, particularly neonates, perceive pain less acutely and therefore do not need pain medication is no longer accepted.[160] As a result, since the early 1990s, there has been a significant change in how pediatric pain is approached. Perhaps the most powerful sign of this change was that the Joint Commission (formerly the Joint Commission on Accreditation of Healthcare Organizations) added pain management to its accreditation review in 2000. Areas that now must be addressed by accredited institutions include pain assessment, pain treatment, and education about pain management.[161]

Pain assessment can be challenging in children. Assessing the degree of pain in preverbal children is problematic, although several observational scales have been developed to assist with this population.[162] Children 4 to 8 years of age have been shown to be capable of giving a self-report with simplified tools, and older children can use the same scales as those for adults.[163] Patients in the intensive care unit setting (immediately postoperatively) and cognitively impaired children remain populations to be studied in the area of pain assessment.

IV opioids—whether given by bolus, infusion, or patient-controlled analgesia—are the mainstay of therapy for severe pain.[164] Children 8 years of age and older and some mature 6- to 7-year-old children do well with patient-controlled analgesia, tend to use less total drug, and have a better feeling of control than with traditional methods of administration. Younger children who require IV opioids may benefit from administration accomplished using parent- or nurse-controlled analgesia. Appropriate parent education is a necessary prerequisite to parent-controlled analgesia.

Milder degrees of pain can be handled with nonopioid analgesics. Acetaminophen (10 to 15 mg/kg q4-6h to a maximum 4 g q24h), alone or with oxycodone (0.1 to 0.15 mg/kg), is useful for children who can take oral medications. Rectal acetaminophen can also be given, although doses of 20 to 40 mg/kg are necessary to achieve serum levels equivalent to those obtained with 10 to 15 mg/kg given orally.[165] IV acetaminophen (15 mg/kg q6h to a maximum daily dose of 75 mg/kg) has been newly approved for children older than 2 years and has been shown to be effective and safe. Ketorolac (0.5 mg/kg IM or IV q6h up to 48 hr) is a nonsteroidal antiinflammatory drug that has potent analgesic properties; these may be equivalent to those of morphine[166] but without the respiratory depression or nausea associated with opioids. However, ketorolac requires 30 to 45 minutes for onset, has a 12% incidence of nausea, and may be associated with a higher incidence of bleeding because of effects on platelet function.[167] Nonopioid analgesics also are a good adjunct to opioid and other pain medications, although multimodal therapy has been shown to be most effective in the treatment of pain, especially in difficult-to-treat cases.

Codeine, often used as a first-line oral opioid, seems to be a weak analgesic. Essentially a prodrug of morphine, it needs to be converted to its active form in the liver. Approximately 10% of the population lack the enzyme CYP2D6 and will not achieve pain relief from codeine. Accordingly, a more rational approach is to use a stronger oral analgesic as a first-line agent, especially after significantly painful procedures. Some children appear to be ultrarapid metabolizers of codeine, which is an inherited (genetic) ability that causes the liver to convert codeine into life-threatening or fatal amounts of morphine in the body. Consequently, a new *Boxed Warning*, FDA's strongest warning, has been added to the drug label of codeine-containing products about the risk of codeine in postoperative pain management in children following tonsillectomy and/or adenoidectomy. As with all opioids, nausea and vomiting are potential side effects and may be more prevalent with use of codeine.

SEDATION AND ANESTHESIA OUTSIDE OF THE OPERATING ROOM

Over the past 10 years, an increasing number of procedures have been performed outside of the OR. Physician time constraints, reimbursement changes, new technology for procedures that cannot be done in the OR, and the development of new anesthetic agents have all contributed to this development. However, the risk of adverse events is potentially greater than that for OR procedures as a result of monitoring variation, poor presedation evaluation, inadequate training, and lack of adequate recovery procedures.[168] More recently, a greater degree of scrutiny has been given to the practice of sedation for children outside of the OR. In an effort to improve safety, the Joint Commission has been instrumental in forcing institutions to develop policies to improve the care of children and adults who are receiving sedation as part of overall accreditation efforts. In addition, specialty organizations have developed policy statements about this topic.[169-171] As a result, in many institutions, pediatric anesthesiologists are more directly involved with sedation outside of the OR than in the past.

A large number of procedures that are performed in hospitals may require use of sedation or analgesia, especially in children. Painful procedures such as lumbar puncture, bone marrow biopsy, and chest tube placement are obvious examples. Other procedures, such as magnetic resonance imaging or gastrointestinal endoscopy, also may require sedation or anesthesia because of the distressing nature of one or more aspects of the examination or the need for immobility. In the context of otolaryngology, the most common candidates for out-of-the-OR or clinic procedures may be the removal of pressure equalization tubes, nasal ciliary biopsy, fine-needle aspiration, or the removal of sutures. One report suggests that these procedures can be performed in the clinic safely and with less