

## Chapter 2: Anatomic Considerations

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### INTRODUCTION

#### FOCUS POINTS

1. Brain growth and development occurs most rapidly in the first 5 years of life. Notable changes of the face and skull are seen in this young period. During adolescence these features approach adulthood.
2. Neonate and young infants are considered preferential nasal breathers. Any obstruction to the nares (eg, secretions) often leads to increased work in breathing.
3. A proportionately larger tongue, young epiglottic shape (long, narrow, and omega), and a cephalad larynx can affect airway management (eg, intubation). Similar to adults, the vocal cord region is the narrowest point of the airway in children.
4. The airway dimensions (short trachea, less acutely angled right main bronchi) of neonate and young children predispose them to mainstem intubation. Meticulous placement of the endotracheal tube is needed because of limited size.
5. Young pediatric patients have immature skeletal muscle and cartilaginous thorax (ie, compliant chest). These features can promote respiratory fatigue during increased demands in breathing (eg, illness).
6. The caudal level of the dura and spinal cord encompasses about two to three interspaces lowered in a neonate compared to an adult. By the first year of life, they are in the adult position.

An understanding of the anatomic changes that occur from birth through late adolescent is essential in the care for the pediatric patient. Pediatric growth may seem continuous but oftentimes occurs in sporadic stages of development (eg, infancy, childhood, adolescence) where rapid changes may be divided by a period of relatively slower or uniform pace.

### SKULL

Infants and young children have a relatively large head to the size of their total body. This is attributed to the rapid growth and development of the brain. At birth, the head is one-fourth the total body length and is 25% of an adult size.<sup>1</sup> In the first year of life, the brain completes half its growth and by the age of 5, approximately 90% of cranial growth has occurred.<sup>2,3</sup> The occiput is also noted to be prominent in these early years. At birth, the anterior and posterior fontanelles are palpable. The posterior fontanel closes first, often in the first several months.<sup>4</sup> The anterior fontanel closes by the age of 2.<sup>5</sup> Except for the metopic suture, which closes completely during the first year of life, the remaining cranial sutures do not fully fuse until adulthood.<sup>6,7</sup> As the child matures, the body size increases relative to head size.

The face matures at a different rate compared to the head (cranium). At birth, the cranium-to-face ratio is 8:1. By the first year of life the ratio is 6:1. The facial characteristic presents as a prominent forehead and eyes in early childhood. During late adolescence, the ratio reaches adult level of 2:1. The facial complex of the nose, maxilla, and mandible increases in size with the role of development (ie, phonation and mastication). In infancy, the mandible is small with a notable oblique mandibular angle. As the child grows, the face becomes more prominent as it develops primarily downward and forward. Nasomaxillary and mandibular growths occur in parallel with dentition.<sup>8</sup> Skeletal maturity of the lower face is reached during mid to late

adolescence.<sup>2</sup> Primary (baby) teeth often occur around 6 months of age and continue in the first 2½ years of life.<sup>9</sup> After gradual shedding of primary teeth, permanent teeth erupt about 6 years of age and are mostly completed by age of 13.<sup>10</sup>

## UPPER AIRWAY

### Nasopharynx

Traditional teaching is that infants are obligate nasal breathers until 3 to 6 months of age.<sup>11</sup> It has been shown that this is not exclusively true.<sup>12</sup> Neonates and young infants are preferentially nasal breathers. Nonetheless, they rely upon patent nares for adequate breathing. Infant's nares are obviously small in size and rapidly increase during the first year of life. By 6 months of age the nares nearly double in dimension from birth. The nares can be quickly occluded (eg, secretions, edema) resulting in an increased work of breathing. There is linear growth of the nasopharynx during the first decade of life.<sup>13</sup> In early childhood, the adenoids develop and may obstruct breathing. Typically they shrink during adolescence.

### Oropharynx

In proportion to the adult, the tongue of the young child is larger resulting in a decreased space inside of the oral cavity. This can compromise breathing and obstruct the airway especially when pharyngeal tone is decreased or lost. Congenital syndromes such as Down syndrome or Beckwith-Wiedemann syndrome often impede visualization of vocal cords during intubation due to the decreased amount of space due to an enlarged tongue. Similar to the adenoids, the tonsils grow rapidly in the first decade of life and often decrease in size during the teenage years. They are a common cause of airway obstruction for school-aged children.

### Hypopharynx and Larynx

The epiglottis in infants differs from adults in several ways. It is proportionally longer, narrower, and omega-shaped. In addition, the epiglottis is oriented at a 45-degree angle into the lumen of the airway. In contrast, the axis of the epiglottis is parallel to the trachea in adults. These differences often impact airway management in infants especially when performing laryngoscopy (or suctioning). It is susceptible to trauma resulting in edema and obstruction around the glottic opening. In addition, in infants, the vocal cords are angled in a caudal orientation (ie, toward the feet) where the anterior location is inferior to the posterior end.<sup>14</sup> In adults, the vocal cord orientation is commonly at a 90-degree angle to the trachea.

During infancy, the larynx lies higher in the neck in relation to the cervical spine. In a neonate, it is at the third and fourth cervical vertebrae (C3-C4) and descends to the adult C5 position by early adolescence (some discrepancy cited at 4–5 years). This higher position of the larynx in children results in a sharper angle between the base of tongue and the glottic opening.<sup>15</sup> Clinically, this is often described as being more “anterior” to an adult but in fact it is more cephalad or “superior.” The cephalad location of the larynx in the young infant provides some protection against aspiration as protective reflexes (ie, swallow-breathe) develop.

Cadaveric studies from the 1950s provided the base for classical teachings of the pediatric airway.<sup>16</sup> For many years the pediatric larynx was taught and described as being funnel- or conical-shaped and the narrowest portion was at the level of the cricoid ring. It was believed that as the child matures between 8 and 10 years of age, the larynx transitioned into the cylindrical adult airway, in which the narrowest portion is at the rima glottis. Recent studies using advanced imaging (ie, computed tomography and magnetic resonance imaging) and direct bronchoscopic measurements have contested previous pediatric tenets.<sup>17–19</sup> These findings demonstrate that the pediatric larynx is similar to adult where the narrowest portion is at or immediately below (subglottic) the vocal cord. In addition, the pediatric airway is not funnel-shaped but is cylindrical in geometry. In young children, the cross-sectional area is elliptical with a greater anteroposterior (AP) to transverse dimension.<sup>20</sup> Despite these findings, the cricoid ring may functionally be the narrowest portion of the airway. It is completely cartilaginous and is relatively nondistensible compared to the upper glottic structures.

### Lower Airway

The developmental anatomy of the respiratory system of infants and young children is another factor to take into consideration. In addition to the extrathoracic portion of the airway is the intrathoracic portion of the airway, which includes the trachea, two mainstem bronchi, bronchi, and bronchioles that conduct air to the alveoli. The pediatric population differs from adults with respect to relative length of the trachea, size difference

between right and left main bronchi, the orientation of their rib cage, the compliance of their chest wall, the musculature of their diaphragm, and the incomplete alveolarization of the lung parenchyma (with regards to children less than 2 years of age).

Particularly in the neonate population, the trachea is short and measures approximately 5 cm. Therefore, precise placement and firm fixation of the endotracheal tube are essential. Table 2-1 demonstrates approximate airway dimensions (intrathoracic portion) in infants and children.

Table 2-1

**Approximate Airway Dimensions in Infants and Children**

Age (yr)	Tracheal Length (cm)	Trachea (AP)	Diameter (mm)	
			Right Bronchus	Left Bronchus
>0.5	5.9	5.0	4.5	3.5
0.5–1	7.2	5.5	4.8	3.7
1–2	7.5	6.3	5.1	3.9
2–4	8.0	7.5	6.4	4.9
4–6	8.6	8.0	6.7	5.3
6–8	9.5	9.2	7.9	6.1
8–10	10	9.75	8.5	6.5
10–12	11.5	10.5	9.2	6.8
12–14	13.5	11.5	9.8	7.5
14–16	14.5	13.2	11.5	8.8

AP, anteroposterior.

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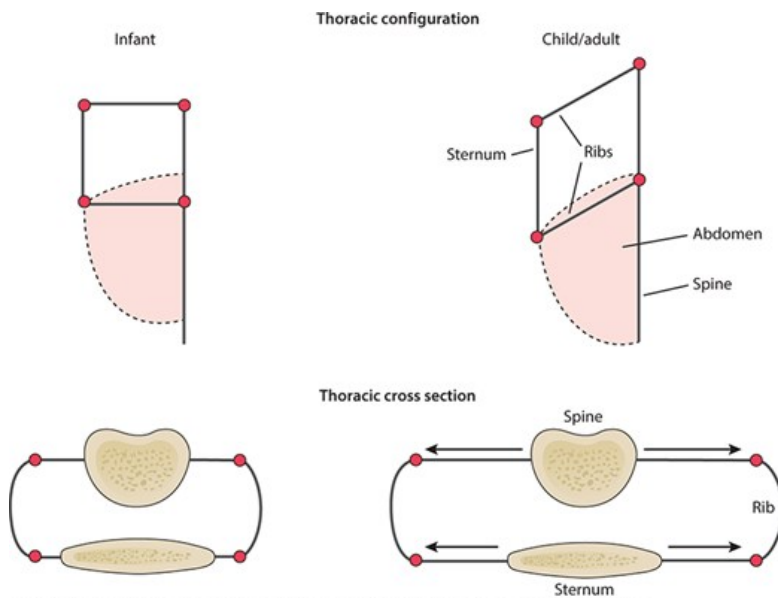
Also, regarding the main bronchi, the right main bronchus is larger than the left and is less acutely angled at its origin.<sup>21</sup> As discussed earlier, precision is crucial for placement of the endotracheal tube because if advancement is too far, it almost invariably will enter the right main bronchus. It is of the utmost importance to continuously reassess the equality of bilateral breath sounds after repositioning for surgeries that require changes in position.

The anatomy of the chest wall of infants and children differs from adults in the orientation of the rib cage. At birth, ribs project at right angles from the spine. The rib cage is also more circular than in adults, thus lacking mechanical efficiency. The angle of insertion of the diaphragm in children is not oblique as in adults but almost entirely horizontal, and ventilation is mainly diaphragmatic. This leads to a decrease in contraction efficiency with a tendency of the diaphragm to pull the lower rib cage inward instead of outward.<sup>22</sup> The abdominal viscera are bulky and further hinder diaphragmatic excursion, especially if the gastrointestinal tract is distended.<sup>21</sup>

Figure 2-1

Changes in configuration and cross-sectional shape of the thorax from infancy to early childhood. (Reproduced with permission, from Taussig L,

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Several anatomic differences make respiration less efficient in infants. In addition to the difference in orientation, the rib cage of infants and young children is rather cartilaginous, and the thorax is too compliant to resist inward recoil of the lungs. This can allow the chest wall to retract during episodes of respiratory distress and fatigue. With the chest wall of infants being highly compliant, the ribs provide very little support for the lungs. As a result, negative intrathoracic pressure is poorly maintained, and each breath is accompanied by functional airway closure.<sup>23</sup> As opposed to the awake state when the chest wall maintains a relative amount of rigidity with sustained inspiratory muscle tension, general anesthesia induces a state that affects the muscle tension by diminishing it, leading to FRC collapse and contributing to further airway closure and atelectasis.<sup>24</sup>

Relative to infants and smaller children, neonates' lungs are even stiffer due to greater alveolar recoil, which is offset by expression of surfactant, while the chest and abdominal walls are more compliant. As a result, the diaphragm muscle must create greater relative intrathoracic pressures to produce a given level of inspiratory airflow and tidal volume.<sup>25</sup>

Differences in chest wall configuration and compliance between young infants and older children can place them at a disadvantage, especially when trying to meet the increased ventilatory requirements, such as those inflicted by pulmonary disease or in the setting of increased metabolic demands. Immaturity of the respiratory muscles, combined with high chest wall compliance, can cause ventilation asynchrony and promote respiratory fatigue.<sup>22</sup> This is why differences in the anatomy of the microstructure of the respiratory muscular system of infants must also be taken into consideration. Skeletal muscle fibers, including those of the diaphragm, may be classified into two basic groups, type I and II, according to histochemical and electrophysiologic characteristics.<sup>26</sup> Type I fibers have a high capacity for oxidative phosphorylation, develop their maximal force generation slowly, also known as "slow-twitch," and are resistant to fatigue. Type II fibers rapidly develop their maximal force generation, also known as "fast-twitch," but this group is further subdivided according to additional properties. Type IIa fibers have intermediate oxidative phosphorylation and are fatigue resistant. Type IIb fibers have poor oxidative phosphorylation and fatigue easily. Type IIc fibers are found only in fetal and neonatal diaphragmatic muscle; they are highly oxidative and resistant to fatigue.

The musculature of the infant's diaphragm is characterized as immature, consisting mainly of type IIc undifferentiated fibers, which are gradually replaced by type I and type IIb fibers. Type IIc fibers co-express fetal and adult myosin heavy chains, whereas type I and type IIb fibers express only the adult myosin heavy chain isoform. The paucity of fatigue-resistant type I fibers, high proportion of fatigue-susceptible type IIc fibers, and low oxidative capacity of the neonatal diaphragm suggest that the muscle may be relatively prone to fatigue.<sup>22</sup>

The diaphragmatic and intercostal muscles do not achieve the adult configuration of type I muscle fibers until the child is approximately 2 years old.<sup>23</sup> Type I muscle fibers provide the ability to perform repeated exercise. With the lack of this type of muscle fiber in the child, any factor that increases the work of breathing will contribute to early fatigue of the respiratory muscles, thus explaining an infant's high respiratory rate, the rapidity with which

hemoglobin desaturation occurs, and their propensity for fatigue and apnea. One source suggests 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, and once a child reaches his/her first year of life, the percentage of type I fibers increases to 55%, the expected adult level.<sup>27</sup>

Alveolarization of lung parenchyma of the infant, more specifically children under the age of 2 years, begins in late gestation. According to some sources, the number of alveoli present at birth is estimated to be less than 20% of the number in adults. Also, alveolar size of an infant is smaller than alveolar size of an adult, thus demonstrating that postnatal growth and development of the lung parenchyma increases in number as well as size of alveoli.<sup>28</sup>

Regarding the respiratory system, the formation of adult-type alveoli begins at 36 weeks postconception but represents only a fraction of the terminal air sacs with thick septa at full-term birth. It takes more than several years for functional and morphologic development to be complete, with a tenfold increase in the number of terminal air sacs to 400 to 500 million by 18 months of age, along with the development of rich capillary networks surrounding the alveoli, a profound difference when comparing infants and young children against adults.<sup>24</sup>

While multiple sources state that alveolar multiplication continues well after birth, “earlier studies suggested that postnatal alveolar multiplication might end at 8 years of age; however, more recent studies showed that alveolar multiplication was complete by 2 years of age and possibly even earlier, between 1 and 2 years of age.”<sup>22</sup> For example, the study conducted by Thurlbeck in 1982 concludes: “The great bulk of alveoli are present by the age of 2 years and limited, or no, alveolar multiplication occurs subsequently.”<sup>29</sup>

By 1 to 2 years of age, most septa show the adult structure characterized by a single capillary network, interwoven with connective tissue strands, that serves to stabilize the interalveolar wall. After the septal restructuring, lung development is considered complete, and the lung enters a period of normal growth that lasts until adulthood. Conclusions of postnatal human lung development consist of (a) the alveolar stage, which starts in late fetal life and lasts at about 1 to 1½ years, and (b) the stage of microvascular maturation, thought to extend from the first months after birth to the age of 2 to 3 years.<sup>30</sup>

It is about 2 years of age that most former studies state the number of alveoli starts to vary substantially among individuals. After the end of alveolar multiplication, the individual alveoli continue to increase in size until thoracic growth is completed.<sup>22</sup>

From the limited number of morphometric studies of relatively few autopsied lungs from infants and toddlers, which have used different morphometric techniques to estimate alveolar number, it remains unclear whether the process of alveolarization is complete by 6 months, 2 to 3 years, or 8 years of age.<sup>28</sup> It is important to realize that it is near impossible to sharply delineate the absolute completion of alveolarization. The various studies that performed quantification measures through alveolar counting procedures have their methodological inaccuracies as well. Most studies base their morphological statements on general or average structural criteria. It is safe to state, however, that the main period of alveolar formation is over before the age of 1½ years, but one cannot exclude a slower addition of further alveoli beyond that age.<sup>30</sup>

The end result of alveolarization and parenchymal growth produces a lung with alveolar and capillary surface areas at least 20 times that of the neonate.<sup>31</sup> From an anatomical standpoint, this is a concept of importance to take into consideration for the anesthetic management of an infant or child versus an adult.

## BODY SIZE

Notable differences in body proportion are seen as a child ages. As noted earlier, the head changes in proportion to total length from birth (1:4) to adulthood (1:8). Similarly, a child's upper to lower segment ratio changes with maturity. This ratio is defined from the head to the symphysis pubis (SP) divided by SP to feet. At birth the ratio is approximately 1:7. At about 10 years of age, the ratio is 1. During late adolescence, the ratio is less than 1, in which the upper body is shorter than the lower body.<sup>32</sup>

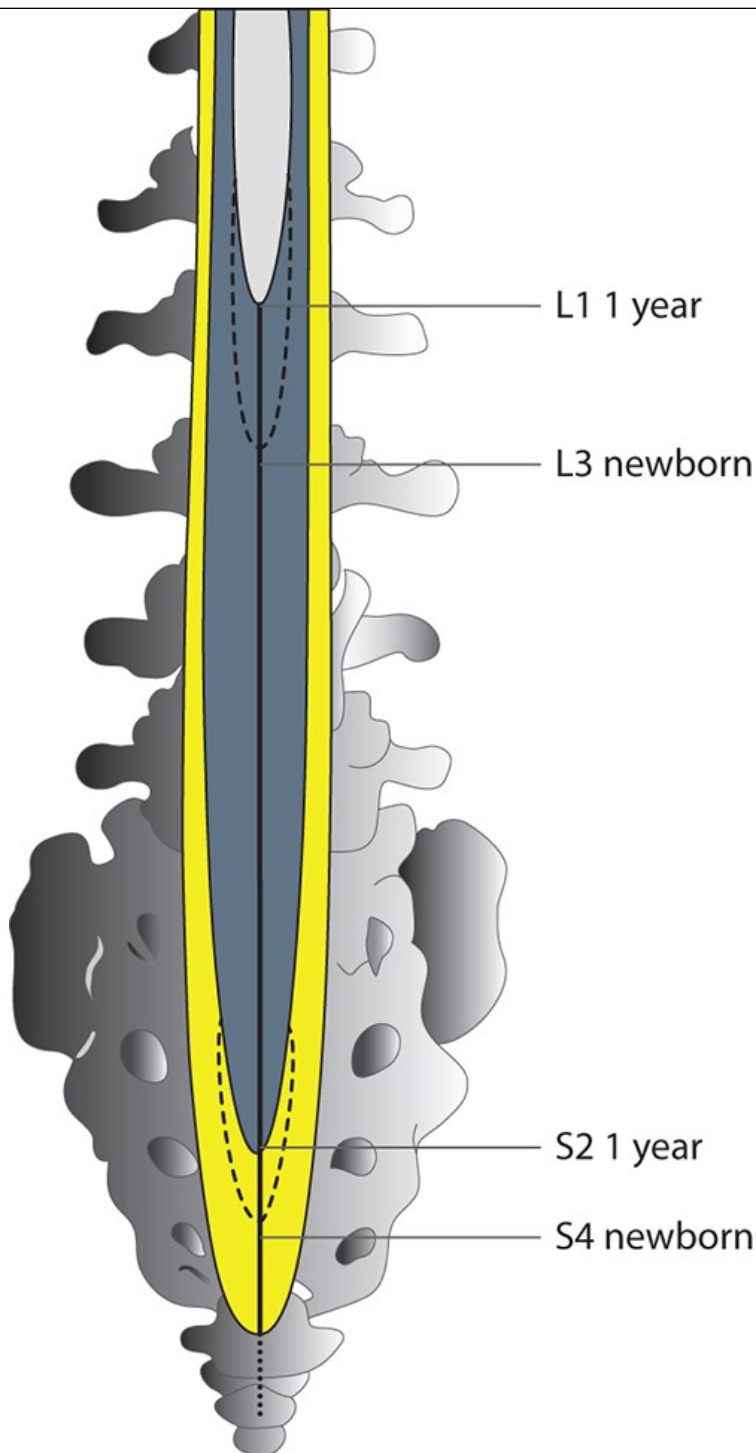
## SPINE

Age-related anatomic variations exist in the location of the caudal termination of the dural sac that can affect neuraxial anesthesia. Differential growth of the vertebrae compared to the nerves and cord accounts for a relative ascension of the cord within the canal over time.<sup>33</sup> By birth the dural sac ends

at S3 or S4, with the conus medullaris terminating at the L3 or L4 level. It is not until approximately 1 year of age that anatomic relationships resemble that of the adult, with the spinal cord and dura mater extending to L1 and S1 to S2, respectively. The lower-lying spinal cord in young infants places them at higher risk with more vulnerability to injury for providers that choose to insert their needle at mid- to upper-lumbar levels.<sup>34</sup> It is possible to enter the dural sac during caudal anesthesia. If spinal anesthesia is used in this population, a low approach to entering the dural sac is warranted to avoid the cord.

**Figure 2-2**

The spinal cord terminates at a much more caudad level in neonates and in infants compared to adults. The conus medullaris ends at approximately L1 in adults and at the L2 or L3 level in neonates and infants. (Reproduced with permission, from *Pediatric Epidural and Spinal Anesthesia and Analgesia*. 2017. <https://www.nysora.com>. Copyright © NYSORA: New York School of Regional Anesthesia. All rights reserved.)



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## REFERENCES

1. Dekaban AS. Changes in brain weights during the span of human life: relation of brain weights to body heights and body weights. *Ann Neurol*. 1978;4(4):345–356. [PubMed: 727739]

2. Tiwana PS, Vickers A. Pediatric cranio-maxillofacial trauma: mandibular fractures. In: Khan HA, Bell RB, Bagheri SC eds. *Current Therapy in Oral and Maxillofacial Surgery* [Internet]. Philadelphia, PA: Elsevier Saunders; 2012:841–850.
3. Marciniak B. Growth and development. In: Coté CJ, Lerman J, Anderson BJ eds. *Coté and Lerman's A Practice of Anesthesia for Infants and Children*. 5th ed. Philadelphia, PA: Elsevier/Saunders; 2013:7–20.
4. Kiesler J, Ricer R. The abnormal fontanel. *Am Fam Physician*. 2003;67(12):2547–2552. [PubMed: 12825844]
5. Duc G, Largo RH. Anterior fontanel: size and closure in term and preterm infants. *Pediatrics*. 1986;78(5):904–908. [PubMed: 3763303]
6. Vu HL, Panchal J, Parker EE, Levine NS, Francel P. The timing of physiologic closure of the metopic suture: a review of 159 patients using reconstructed 3D CT scans of the craniofacial region. *J Craniofac Surg*. 2001;12(6):527–532. [PubMed: 11711818]
7. Cohen MM Jr. Sutural biology and the correlates of craniosynostosis. *Am J Med Genet*. 1993;47(5):581–616. [PubMed: 8266985]
8. Smartt JM Jr, Low DW, Bartlett SP. The pediatric mandible: I. A primer on growth and development. *Plast Reconstr Surg*. 2005;116(1):14e–23e. [PubMed: 15988242]
9. Tooth eruption. *J Am Dent Assoc*. 2005;136(11):1619. [PubMed: 16329427]
10. Tooth eruption. *J Am Dent Assoc*. 2006;137(1):127. [PubMed: 16457009]
11. Miller MJ, Martin RJ, Carlo WA, Fanaroff AA. Oral breathing in response to nasal trauma in term infants. *J Pediatr*. 1987;111(6 pt 1):899–901. [PubMed: 3681558]
12. Miller MJ, Carlo WA, Strohl KP, Fanaroff AA, Martin RJ. Effect of maturation on oral breathing in sleeping premature infants. *J Pediatr*. 1986;109(3):515–519. [PubMed: 3746545]
13. Arens R, McDonough JM, Corbin AM et al. Linear dimensions of the upper airway structure during development: assessment by magnetic resonance imaging. *Am J Respir Crit Care Med*. 2002;165(1):117–122. [PubMed: 11779740]
14. Litman R, Fiadjoe J, Stricker P, Coté C. The pediatric airway. In: Coté CJ, Lerman J, Anderson BJ eds. *Coté and Lerman's A Practice of Anesthesia for Infants and Children*. 5th ed. Philadelphia, PA: Elsevier/Saunders; 2013:237–276.
15. Adewale L. Anatomy and assessment of the pediatric airway. *Paediatr Anaesth*. 2009;19(suppl 1):1–8. [PubMed: 19572839]
16. Eckenhoﬀ JE. Some anatomic considerations of the infant larynx influencing endotracheal anesthesia. *Anesthesiology*. 1951;12(4):401–410. [PubMed: 14847223]
17. Dalal PG, Murray D, Messner AH, Feng A, McAllister J, Molter D. Pediatric laryngeal dimensions: an age-based analysis. *Anesth Analg*. 2009;108(5):1475–1479. [PubMed: 19372324]
18. Litman RS, Weissend EE, Shibata D, Westesson PL. Developmental changes of laryngeal dimensions in unparalyzed, sedated children. *Anesthesiology*. 2003;98(1):41–45. [PubMed: 12502977]
19. Wani TM, Rafiq M, Talpur S, Soualmi L, Tobias JD. Pediatric upper airway dimensions using three-dimensional computed tomography imaging. *Paediatr Anaesth*. 2017;27(6):604–608. [PubMed: 28306197]
20. Tobias JD. Pediatric airway anatomy may not be what we thought: implications for clinical practice and the use of cuffed endotracheal tubes. *Paediatr Anaesth*. 2015;25(1):9–19. [PubMed: 25243638]



21. Lerman J, Coté CJ, Steward DJ. Anatomy and physiology. In: Lerman J, Coté CJ, Steward DJ eds. *Manual of Pediatric Anesthesia*. 7th ed. Switzerland: Springer; 2016.

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22. Gaultier C, Denjean A. Developmental anatomy and physiology of the respiratory system. In: Taussig L, Landau L eds. *Pediatric Respiratory Medicine*. 2nd ed. Elsevier Health Sciences; 2008:15–34.

---

23. Coté CJ. Pediatric anesthesia. In: Miller R, Eriksson L, Fleisher L, Wiener-Kronish, Cohen N, Young W eds. *Miller's Anesthesia*. Vol 2. 8th ed. Philadelphia, PA: Elsevier/Saunders; 2015:2757–2798.

---

24. Davis PJ, Motoyama EK, Cladis FP. Special characteristics of pediatric anesthesia. In: Davis PJ, Cladis FP eds. *Smith's Anesthesia for Infants and Children*. 9th ed. Elsevier Health Sciences; 2016:2–9.

---

25. Mantilla CB, Fahim MA, Bradenburg JE, Sieck GC. Functional development of respiratory muscles. In: Polin RA, Abman SH, Rowitch D, Benitz WE eds. *Fetal and Neonatal Physiology*. Vol 1. 5th ed. Elsevier Health Sciences; 2016:692–705.

---

26. Gutierrez JA, Duke T, Henning R, South M. Respiratory failure and acute respiratory distress syndrome. In: Taussig LM, Landau LI eds. *Pediatric Respiratory Medicine*. 2nd ed. Elsevier Health Sciences; 2008:253–274.

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27. McMurray JS. General considerations in pediatric otolaryngology. In: Lesperance MM, Flint PW eds. *Cummings Pediatric Otolaryngology*. Philadelphia, PA: Elsevier Saunders; 2015:1–10.

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28. Balinotti JE, Tiller CJ, Llapur CJ, Jones MH, Kimmel RN, Coates CE et al. Growth of the lung parenchyma early in life. *Am J Respir Crit Care Med*. 2009;179(2):134–137. [[PubMed: 18996997](#)]

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29. Thurlbeck WM. Postnatal human lung growth. *Thorax*. 1982;37(8):564–571. [[PubMed: 7179184](#)]

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30. Zeltner TB, Burri PH. The postnatal development and growth of the human lung. II. Morphology. *Respir Physiol*. 1987;67(3):269–282. [[PubMed: 3575906](#)]

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31. Joza S, Post M. Development of the respiratory system (including the preterm infant). In: Rimensberger PC ed. *Pediatric and Neonatal Mechanical Ventilation: From Basics to Clinical Practice*. Springer Berlin Heidelberg; 2015:3–25.

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32. Nwosu BU, Lee MM. Evaluation of short and tall stature in children. *Am Fam Physician*. 2008;78(5):597–604. [[PubMed: 18788236](#)]

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33. Brull R, Macfarlane AJ, Chan VW. Spinal, epidural, and caudal anesthesia. In: Miller RD, Eriksson LI, Fleisher L, Wiener-Kronish JP, Cohen NH, Young WL eds. *Miller's Anesthesia*. Vol 1. 8th ed. Philadelphia, PA: Elsevier/Saunders; 2015:1684–1720.

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34. Birmingham PK. Pediatric postoperative pain. In: Benzon HT, Raja SN, Liu SS, Fishman SM, Cohen SP eds. *Essentials of Pain Medicine*. 3rd ed. Elsevier/Saunders; 2011:238–242.

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