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Review Article

REVIEW ARTICLE

Effects of anaesthesia on paediatric lung function

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Abstract

Respiratory adverse events are one of the major causes of morbidity and mortality in paediatric anaesthesia. Aside from predisposing conditions associated with an increased risk of respiratory incidents in children such as concurrent infections and chronic airway irritation, there are adverse respiratory events directly attributable to the impact of anaesthesia on the respiratory system. Anaesthesia can negatively affect respiratory drive, ventilation/perfusion (V/Q) matching and tidal breathing, all resulting in potentially devastating hypoxaemia. Understanding paediatric respiratory physiology and its changes during anaesthesia will enable anaesthetists to anticipate, recognize and prevent deterioration that can lead to respiratory failure. This review aims to give a comprehensive overview of the effects of anaesthesia on respiration in children. It focuses on the impact of the different components of anaesthesia, patient positioning and procedure-related changes on respiratory physiology.

Key words: anaesthesia; anaesthetic agents; child; lung fuction; lung volumes; paediatric; respiratory system; ventilation

Respiratory adverse events are a major cause of morbidity and mortality in paediatric anaesthesia, occurring in up to 20% of all anaesthetics.[1 2] More than three quarters of critical incidents are caused by respiratory problems,[3] and a quarter of all anaesthesia related cardiac arrests in children are directly linked to respiratory complications, most commonly laryngospasm.[4] Predisposing conditions associated with an increased risk of respiratory adverse events in children include concurrent infections and chronic airway irritation, evidenced by chronic nocturnal cough or recurrent wheezing.[2] There are also inherent risks of respiratory adverse events attributable to the impact of anaesthesia on the respiratory system. Anaesthesia can negatively affect respiratory drive, ventilation/perfusion (V/Q) matching and tidal breathing. Understanding the respiratory physiology and its changes during anaesthesia in children of varying ages will thus help to anticipate, recognize and prevent deteriorations that can precipitate respiratory failure. In recent yr there has been a renewed interest in the effects of specific components of anaesthesia on lung function. These components include the different anaesthetic agents, different ventilation strategies, patient positioning and the effects of cardiopulmonary bypass during cardiothoracic surgery. The present review aims to summarize current knowledge in order to reduce respiratory adverse events in paediatric anaesthesia.

Patients at risk: essentials of children's respiratory physiology

A number of age-specific characteristics of respiratory physiology put infants and young children at a higher risk of respiratory failure during anaesthesia. These characteristics include the immaturity of respiratory control and certain protective reflexes (in the youngest children), the size and collapsibility of the upper and lower airways, the lower efficiency of the respiratory musculature, the reduced surface area available for gas exchange, and the altered balance of chest wall *vs* lung compliance. The characteristics of respiratory physiology in the neonatal period are prototypical for these differences compared with adults, and it is these

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differences that underlie the vulnerability of infants and young children to respiratory adverse events. Many of these physiologic characteristics, though, become less clinically apparent within the first 2 yr of life. However, as the maturation of respiratory function is a gradual process from birth to adulthood, distinct age limits for any of these characteristics are difficult to define.

Control of breathing and protective reflexes

Autonomous ventilation is mainly driven by the partial pressure of carbon dioxide (CO_2) and the pH of the arterial blood and cerebrospinal fluid, and, to a lesser extent, by the arterial partial pressure of oxygen (Pa_{O_2}). Immature infants exhibit an attenuated CO_2-response with a blunted acceleration of the respiratory rate,[5 6] which is in contrast to healthy infants who show an even stronger CO_2-response than adults.[7] A dampened ventilatory response to hypercapnia is also seen in sick children such as infants of substance misusing mothers.[8] The ventilatory response to hypoxia is dependent on the CO_2-level and is typically biphasic: after an initial increase in ventilation, sustained hypoxia reduces ventilatory drive. This hypoxic ventilatory depression is observed through all ages, but is most pronounced in immature infants in whom respiratory rates decrease to below baseline after 1–2 min of hypoxia in experimental settings.[6 9 10] This is in contrast to adults where sustained hypoxia dampens the hypoxia-response only partially and only after a longer interval of 10–15 min (Fig. 1).[11] The response to hypoxia is stimulated as Pa_{CO_2} increases.[12] Hypocapnia in awake healthy individuals typically causes little changes in respiratory rate apart from a slight reduction of the respiratory effort (i.e. the tidal volumes).[13] This is in contrast to sleep and sedation, where hypocapnia causes periodic breathing and apneas as a result of the loss of suprapontine ventilatory control.[14]

The laryngeal chemoreceptor reflex inducing swallowing and laryngeal closure to prevent aspiration is operative immediately after birth,[15] whilst the cough reflex gradually evolves during the first months of life.[16]

In summary, blunted response to inadequate gas exchange and weaker protective reflexes need to be anticipated during anaesthesia of preterm and young infants.

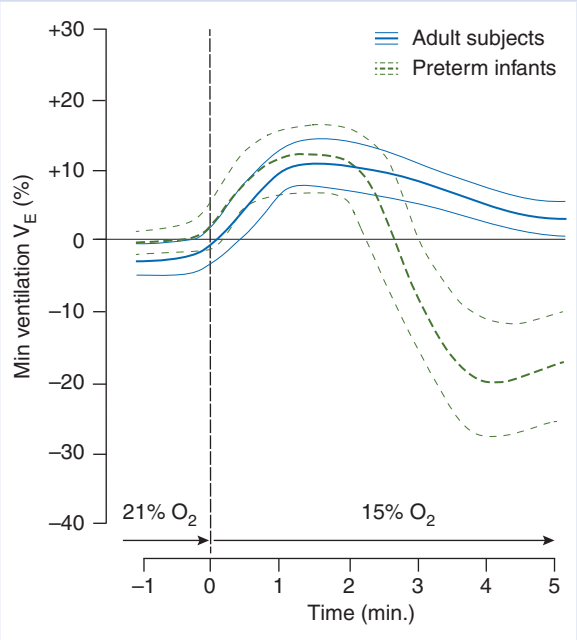

Fig 1 Ventilatory response to hypoxia in preterm infants [gestational age 33.5 (05) weeks] at one week of age compared with adult subjects. Lines indicate means (thick lines) SD (thin lines). Adapted from Sankaran and colleagues[10]

Upper and lower airway resistance

Up to 50% of airway resistance in infants occurs in the nasal passages,[17] and given infants' preference for nasal breathing, any obstruction at this level impacts overall airway resistance. Compared with adults, children have smaller upper airways which, when children are sedated, are more prone to collapse. Because of efficacious protective reflexes however, airway collapse is not observed in healthy sleeping children. In fact, a rapid and vigorous protective neuromotor response to both negative pressures (such as during inspiration) and hypercapnia, render the upper airways even less collapsible during sleep compared with adults.[7 18] These protective reflexes,[19] however, may be compromised in various conditions. Muscular hypotonia and neuronal immaturity in newborns, for instance, predispose to dynamic upper airway obstruction, which usually resolves by two months of age.[20] A number of studies indicate that upper airway patency is controlled by the central nervous system,[21] so that any process affecting neuronal function, such as general and topical anaesthesia increases the vulnerability to upper airway collapse.[22 23] In addition, children with obstructive sleep apnea (OSA) have diminished neuromotor responses to negative pressures compared with healthy children, increasing upper airway collapsibility both in sleep and under anaesthesia.[24 25]

The airway resistance of the lower airways is substantially higher in infants compared with older children and adults.[26] In addition, owing to reduced elastic pulmonary recoil forces, the lower airways collapse more easily, favouring early flow limitation.[27] The airway smooth muscle tone is thought to be important in infants for maintaining airway wall stability,[28 29] as smooth muscle relaxation may worsen airway obstruction in wheezy infants.[30] Beyond infancy, however, inhalation with a β2-agonist does not negatively affect expiratory flows in healthy children.[31] Although anaesthesia alters thoracic recoil forces, potential effects on airflow during anaesthesia are primarily dependent on bronchoconstrictive properties of specific agents.

Developmental pulmonary physiology and lung mechanics

A number of physiologic characteristics negatively affect pulmonary reserves of infants; mainly a shifted balance between the lung's inward and the thoracic outward recoil, the disynaptic growth of airways and lung parenchyma, and the lack of functional collateral ventilation.

The highly compliant thoracic cage of infants offers reduced counterbalance to the inward recoil of the lungs, resulting in a physiologically lower resting functional residual capacity (FRC). In fact, FRC may be as low as 15% of vital capacity when compared with more than 30% in adults.[32] This places the FRC in infants close to the lower inflection point of the pressure/volume curve of the lungs and thus at a significantly higher risk of derecruitment and atelectasis (Fig. 2).[33] This is, however, counteracted by an active elevation of the FRC level in infants both during the wake state and the different stages of sleep. The mechanisms involved in actively elevating FRC are:

- A high respiratory rate with shortened expiratory phase, and initiation of inspiration before completion of passive

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