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 function; 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 Pre-disposing 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.5 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
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 (CO₂) and the pH of the arterial blood and cerebrospinal fluid, and, to a lesser extent, by the arterial partial pressure of oxygen (PaO₂). Immature infants exhibit an attenuated CO₂-response with a blunted acceleration of the respiratory rate, and which is in contrast to healthy infants who show an even stronger CO₂-response than adults. A dampened ventilatory response to hypocapnia is also seen in sick children such as infants of substance misusing mothers. The ventilatory response to hypoxia is dependent on the CO₂-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. This is in contrast to adults where sustained hypoxia dampens the hypoxia-response only partially and only after a longer interval of 10 min (Fig. 1). The response to hypoxia is stimulated as PaCO₂ increases. 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). 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.

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

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

Upper and lower airway resistance

Up to 50% of airway resistance in infants occurs in the nasal passages, 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 hypocapnia, render the upper airways even less collapsible during sleep compared with adults. However, these protective reflexes, however, may be compromised in various conditions. Muscular hypotonnia and neuronal immaturity in newborns, for instance, predispose to dynamic upper airway obstruction, which usually resolves by two months of age. A number of studies indicate that upper airway patency is controlled by the central nervous system, so that any process affecting neuronal function, such as general and topical anaesthesia increases the vulnerability to upper airway collapse. 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.

The airway resistance of the lower airways is substantially higher in infants compared with older children and adults. In addition, owing to reduced elastic pulmonary recoil forces, the lower airways collapse more easily, favouring early flow limitation. The airway smooth muscle tone is thought to be important in infants for maintaining airway wall stability, as smooth muscle relaxation may worsen airway obstruction in wheezy infants. Beyond infancy, however, inhalation with a β2-agonist does not negatively affect expiratory flows in healthy children. 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 dysnaptic 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. 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). 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
As the lungs mature, pulmonary elastic recoil decreases and compliance increases.26 The ribcage is contemporaneously getting stiffer, offering stronger opposition to derecruitment and thus stabilizing FRC. In addition, ribcage geometry changes, lowering the ribs from a horizontal to a more oblique antero-posterior orientation and thereby improving efficacy of the auxiliary respiratory musculature.37

Disynaptic growth refers to the unsynchronized growth of airways and lung parenchyma. By the end of the 16th gestational week, intrapulmonary pre-acinar airway formation and arterial branching are complete and the number of airway divisions varies from 10 to 25 generations, depending on their location within the lungs.38 No new conducting airways will develop after this point in time, but lung maturation continues by formation of new respiratory units, which differentiate by alveolarization into fully functional gas exchanging units.39

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Dead space and ventilation/perfusion (V/Q) matching

The anatomical dead space per kilogram body weight decreases during childhood, mostly during the first four yr of life, as a result of a proportionate decrease of the extrathoracic dead space (i.e. the oro- and nasopharyngeal cavities).40 This implies a higher dead space-to-tidal volume ratio in young children. This however is highly variable because of the dynamic changes of upper airway dimensions and of the proportion of nasal and oropharyngeal dead space participating in ventilation.41 In adults, both ventilation and perfusion of the lungs are preferentially directed towards the dependent part of the lungs.42 43 Traditional teaching is that, in contrast to adults, ventilation in children is predominantly in the upward part of the lung, leading to V/Q mismatch.42 43 Recent work, however, has shown that in spontaneously breathing children, distribution of ventilation is in fact variable and poorly predictable, with one third of children aged six months to nine yr consistently showing greater ventilation to the upward lung, 15% revealing preferential ventilation of the dependent part, and half of children showing a varied pattern.44

Overview of the effects of anaesthesia on lung function

Anaesthesia may negatively affect control of breathing, bulbar function and upper airway patency. It can lead to de-recruitment and consecutively to relevant changes of lung compliance.

Lung de-recruitment, atelectasis and V/Q mismatch

Immediately after the induction of general anaesthesia with volatile anaesthetics or propofol, there is a reduction in FRC by 15–20% in adults,55 which may persist for several h after cessation of anaesthesia and which is beyond what occurs because of supine positioning alone.56 57 This reduction of FRC may cause small airway closure, atelectasis and V/Q mismatch, ultimately resulting in hypoxaemia.58 In infants, airway closure at low FRC occurs more readily, as the surrounding lung parenchyma offers less elastic recoil to support the small airways.59 Deeper states of sedation and anaesthesia inhibit the infants’ FRC defending reflexes. As a result, atelectasis occurs in dependent lung regions in more than 80% of children below three yr of age immediately after induction of anaesthesia, particularly in conjunction with neuromuscular block.59–65 In an early study by Dobinson and colleagues64, children and adolescents under general anaesthesia with paralysis showed a 35% reduction in FRC compared with the awake supine state, which was even more pronounced in children under 11 yr (FRC – 44%). These findings were

Respiratory system resistance and compliance

Respiratory system resistance (to volume change) comprises of 1) airway resistance to gas flow 2) tissue (or viscoelastic) resistance, a measure of the frictional resistance of the lung and thorax to changing shape and 3) mass inertia of the gas and the tissue (which is generally considered negligible).43 As a result of these resistances, dynamic compliance is always lower than the static compliance, which reflects the pure elastic properties of the tissue. Flow resistance within the airways becomes relevant at high breathing frequencies in healthy lungs,44 whereas visco-elastic properties are the predominant system resistance at lower respiratory rates.45 These viscoelastic properties are more relevant in infants and children compared with adults.46 In any viscoelastic organ, the relationship between elastic and viscous properties differs during inspiration and expiration, forming the typical pressure volume (P/V) loop showing hysteresis (Fig. 2). Lung parenchyma, chest wall and surfactant all show hysteresis,47 48 and it is common notion that optimal breathing should cycle on the steep part of the loop.
reproduced in a later study revealing a mean FRC reduction by 44% in muscle-relaxed infants when compared with a 10% reduction in FRC in older children, which was beyond that caused by general anaesthesia (with propofol) alone.62 Respiratory muscle relaxation is thus the most important factor of FRC decline, resulting in movement of the dependent part of the diaphragm to a more cephalad position, an increased lordosis of the thoracic spine and a chest volume reduction.34 66 67 Another mechanism by which FRC may be reduced is the loss of alveolar volume from nitrogen (N₂) wash-out by using high inspired oxygen concentrations.61

Impact on compliance and resistance

De-recruitment of the lungs, as discussed above, shifts tidal ventilation to a flatter part of the P/V curve.62 In addition, Westbrook and colleagues68 showed that anaesthesia is associated with an overall right shift and a decreased slope of the P/V curve (i.e. for any given transpulmonary pressure the lung volume decreases, and the pressure gradient to achieve any given tidal volume increases). Possible factors contributing to this include airway closure and microatelectases, and changes in secretion and distribution of surfactant under anaesthesia affecting alveolar surface tension.69 70 Neonates and infants tend to show the greatest changes in respiratory compliance under anaesthesia. There are a number of potential implications of an altered respiratory compliance. In spontaneously breathing patients, a reduced compliance results in an increased work of breathing and risk of fatigue that may lead to alveolar hypoventilation. Clinically, a loss of compliance is accompanied by a tidal volume reduction and an increase in spontaneous respiratory rate in infants and toddlers.71

The effects of anaesthesia on the airway resistance are dependent on a number of factors, which in the sum may result in either an overall increase or a decrease of airway resistance. A reduction in FRC decreases airway diameter, which in turn increases resistance. Contrary to common beliefs, tracheal tubes do not significantly increase airway resistance at normal airflow velocities, even in small children.72 Anaesthetic agents may have both bronchodilatory and bronchoconstrictory properties.73 These effects on airway resistance may be mediated by direct effects on airway smooth muscle, by indirect effects via the parasympathetic and sympathetic nervous systems, and by a release of broncho-active inflammatory substances.74 75

Agent specific impacts on respiration during anaesthesia

Anaesthetic agents have various specific effects on respiration which are mostly dose-dependent but may also reflect idiosyncratic reactions. An overview is given in Table 1.

Propofol

Propofol induces skeletal muscle relaxation by central inhibition of motor pathways and by sodium channel blockade in the muscular sarcolemma.76 77 In a study of spontaneously breathing children aged two to six yr, both FRC and ventilation homogeneity decreased significantly with increasing depth of propofol anaesthesia.78 Propofol reduces respiratory resistance in both asthmatic and non-asthmatic adult patients.79 In animal models, this is mediated via vagolytic and direct effects upon the bronchial and the central airway smooth muscles.74 80 81 In the paediatric age group, respiratory resistance and dynamic compliance remain similar in both children, with and without a history of asthma, suggesting that propofol is safe to use in children with reactive airway disease.82 Rarely, however, it may provoke broncho-constriction, most likely through histamine release induced by sensitization to propofol or to one of its preservatives.83 84

Benzodiazepines

In healthy adult subjects midazolam potentially affects ventilation by reducing FRC and tidal volumes, while min ventilation is maintained by a concomitant increase of the respiratory rate.85 Premedication of 3–8 yr-old children with 0.3 mg kg⁻¹ oral midazolam had a small effect on respiration, including a reduction in FRC and respiratory compliance, and an increase in respiratory resistance and ventilation inhomogeneity.86 These effects are likely attributable to the neuromuscular blocking agent properties of benzodiazepines impacting on respiratory and upper airway muscle tone.87 88 Children with a vulnerability to upper airway obstruction are conceivably at risk for adverse events when premedicated with midazolam and thus require closer monitoring. Serious adverse events, however, seem to be rare.89 90

Opioids

Opioids have – even in low dosages – a negative effect on control of breathing and also negatively affect upper airway patency1 and swallowing competence.91 Opioids exert a particular influence on respiration by activating abdominal and chest wall musculature. Since the first descriptions of opioid-induced expiratory muscle activation,91 92 the phenomenon has been better studied in adults, where a reduction of chest wall volume accompanied the decrease in FRC and respiratory compliance, after opioid administration.93 94 In children, chest wall rigidity after the administration of opioids is mainly known from case reports with fentanyl and remifentanil. It is noteworthy that active upper airway obstruction has been well described after sufentanil and may contribute to difficult mask ventilation,95 which can be clinically indistinguishable from chest wall rigidity. The true burden of this complication is therefore unknown. In neonates and preterm babies, chest wall rigidity with fentanyl has been estimated to occur in almost one in 10 children at dosages of 2–7 μg kg⁻¹.96 Empirical data suggest that in children less than three yr of age, higher doses of remifentanil of >0.1 μg kg⁻¹ min⁻¹ may be given before respiratory rate depression ensues.97

Volatile anaesthetics

Volatile anaesthetics have long been known to reduce FRC in both spontaneously breathing and ventilated adult subjects, albeit to a variable degree.95 100 101 Recent animal data raise the possibility that drug-induced alterations of surfactant composition may contribute to lung de-recruitment.102 In addition, volatile anaesthetics have been implicated in affecting the inflammatory responses to mechanical ventilation or experimental endotoxin-induced lung injury, both by either attenuating103–106 or enhancing local inflammation.107 108 After halothane induction the compliance of the respiratory system may drop by one-third on average in toddlers, resulting in decreased tidal volumes which are counteracted by an increased respiratory rate.71

Much research has concentrated on the effects of volatile anaesthetics on bronchial smooth muscle tone. The general notion is that in healthy subjects volatile anaesthetics are neutral to the airway muscle tone, or exert a mild bronchodilator effect,
<table>
<thead>
<tr>
<th>Anaesthetic Agents</th>
<th>Control of breathing (respiratory drive)</th>
<th>Bulbar function (pharyngeal and respiratory protective reflexes)</th>
<th>FRC defending mechanisms</th>
<th>Bronchoconstriction</th>
<th>Bronchodilation</th>
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<tbody>
<tr>
<td>Propofol</td>
<td>Dose dependent decrease, may occur before sufficient dampening of pharyngeal reflex</td>
<td>Dampened (especially pharyngeal reflexes)</td>
<td>Atelectasis formation common, dose-dependent</td>
<td>Rare case reports of bronchospasm in the context of allergic reaction to propofol</td>
<td>Some bronchodilatory properties. Weak protection against bronchoconstriction, mostly animal data</td>
</tr>
<tr>
<td>Benzodiazepines</td>
<td>Moderate, dose dependent decrease; accentuated in interaction with other hypnotic drugs and opioids</td>
<td>Dampened, dose dependent</td>
<td>Mild FRC decrease, depends on neuromuscular blocking agent properties</td>
<td>No evidence</td>
<td>No evidence</td>
</tr>
<tr>
<td>Opioids</td>
<td>Dose dependent decrease</td>
<td>Dampening, dose dependent</td>
<td>FRC decrease may be caused by respiratory muscle activation</td>
<td>Anaphylactoid histamine liberating process</td>
<td>Scarce data suggest no effect</td>
</tr>
<tr>
<td>Ketamine</td>
<td>Usually Preserved (in clinically relevant doses)</td>
<td>Usually Preserved (in clinically relevant doses)</td>
<td>Not affected</td>
<td>No evidence</td>
<td>Protection against bronchoconstriction, weak bronchodilation</td>
</tr>
<tr>
<td>Volatile anaesthetics</td>
<td>Decreased, dose dependent</td>
<td>Usually decreased, on some occasions exaggerated (laryngospasm with sevoflurane or desflurane)</td>
<td>Atelectasis formation common</td>
<td>Desflurane may cause bronchoconstriction particularly in children</td>
<td>Protection against bronchoconstriction, mild bronchodilation</td>
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<tr>
<td>Neuromuscular block</td>
<td>No direct effect</td>
<td>Abolished</td>
<td>Markedly impaired</td>
<td>Potential because of anaphylactoid reactions</td>
<td>No evidence</td>
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with the exception of higher dosages of desflurane (>1.5 minimum alveolar concentration, MAC) that may provoke bronchoconstriction. Desflurane can act as an airway irritant and may stimulate airway smooth muscles via a tachykinin pathway. Particular interest has focused on the ability of volatile anaesthetics to protect against or to relieve active bronchoconstriction. Animal models suggest that these drugs indeed counteract methacholine or histamine-induced bronchoconstriction. Quicker recovery was observed in allergy-induced bronchoconstriction with volatile anaesthetics in animals, albeit with a more doubtful efficacy as a preventative measure. In a study investigating the effects of sevoflurane and desflurane on airway resistance in children with and without asthma, sevoflurane slightly decreased airway resistance in children both with and without airway hyper reactivity when compared with propofol anaesthesia, whereas desflurane caused an increase in airway resistance in all children, most pronounced in those with susceptible airways. It may thus be prudent to avoid desflurane in children with a history of recent upper airway infection or reactive airway disease.

The sensitivity of different respiratory muscles to volatile anaesthetics varies; the glossopharyngeal muscles are generally the first to be weakened, followed by the intercostal muscles and, lastly the diaphragm. In spontaneously breathing patients, these changes are associated with an increased upper airway resistance that can result in thoraco-abdominal asynchrony (i.e. an inspiratory inward movement of the ribcage and simultaneous expansion of the abdomen).

**Ketamine**

At dosages of 1–2 mg kg\(^{-1}\), tidal volumes slightly increase with ketamine, while respiratory rate is affected inconsistently, ranging from an increased respiratory drive to bradypnoea and even apnoea. Consistent with the presumption that ketamine has little impact on intercostal and airway muscle function, it has little or no effect on FRC, as the breaking of expiration by which young children defend their FRC is preserved. Ketamine does not appear to affect airway resistance in healthy patients, but may reduce airway resistance in patients with bronchoconstriction. Proposed mechanisms for ketamine’s effects on airway tone include increased catecholamine release, inhibition of catecholamine uptake, blockade of voltage gated Ca\(^{2+}\) channels and inhibition of postsynaptic parasympathetic receptors. A number of studies have revealed a protective effect of ketamine against bronchoconspasm in patients with asthma undergoing anaesthesia, and after an initial case report describing a salvaging effect of ketamine as bronchodiator in a child with severe asthma, ketamine has even come into play as rescue medication for severe paediatric status asthmaticus. To this day, however, the efficacy of ketamine as anti-asthmatic treatment is controversial and not established in children.

**Neuraxial anaesthesia**

Neuraxial anaesthesia may be expected to impact on lung mechanics if it affects abdominal and respiratory muscle tone and their respective interplay. Local anaesthetics administered via the caudal route, the most commonly used neuraxial technique in the paediatric population, generally decrease abdominal muscle tone while sparing the respiratory muscles (diaphragm and intercostal muscles). The limited paediatric data available on the effect of caudal epidural anaesthesia on FRC, suggests an increase in FRC after caudal block. In addition, in an earlier study in young infants, spinal anaesthesia with a sensory block at the T2–T4 level, caused a reduction or loss of intercostal muscle activity while preserving diaphragmatic breathing, resulting in thoraco-abdominal asynchrony in some individuals.

**Impact of ventilation strategies during anaesthesia**

**Inspired fraction of oxygen (\(F_{IO2}\))**

A high (\(F_{IO2}\)) is commonly used in anaesthesia, particularly at induction and in emergency situations in order to increase the patient’s oxygen reserve and thus to maintain arterial oxygenation during hypoventilation. A high (\(F_{IO2}\)) however, results in derecruitment, ventilation inhomogeneity, and V/Q mismatch, as alveolar nitrogen (\(N_2\)) is washed out and alveolar \(O_2\) is absorbed into the pulmonary circulation. These changes may occur within 5 min of administration of 100% oxygen in adults. Lowering oxygen concentrations slows atelectasis formation in a time- and concentration-dependent manner. While an in-depth elaboration of the detrimental effects of high (\(F_{IO2}\)) on the immature lung is beyond the scope of this review, it is emphasized that high oxygen concentrations tend to mask \(V/Q\) mismatch from increasing alveolar collapse and ventilation inhomogeneity. Titrating (\(F_{IO2}\)) is a dilemma: while increasing \(F_{IO2}\) increases oxygen reserve and tolerance to hypoventilation, it also accelerates pulmonary derecruitment and masks worsening \(V/Q\) mismatch. A proposed approach is to pre-oxygenate with an (\(F_{IO2}\)) of 0.8 for a short period and then to reduce the (\(F_{IO2}\)) to 0.30–0.35 as soon as the airway is secured and ventilation is stabilized. A retrospective analysis of a large cohort of paediatric patients having undergone rapid sequence induction has shown that hypoxemia rarely occurred, even in infants, when pre-oxygenation with 100% oxygen was added by gentle mask ventilation.

**Recruitment manoeuvres**

A recruitment manoeuvre is a ventilation technique used to re-expand atelectatic lung areas and thereby improving oxygen transfer and compliance. In order to be effective, a recruitment manoeuvre must apply sufficient inspiratory pressure to open collapsed areas of the lung, followed by a sufficient level of PEEP to keep the alveoli open. Studies have suggested that in healthy adult lungs, airway pressures must be raised to 40 cm H\(_2\)O to overcome alveolar opening pressure and re-aerate collapsed areas during general anaesthesia. In a paediatric study involving children aged six months to six yr, a significant reduction in atelectasis was similarly found by MRI, after a recruitment manoeuvre with 10 breaths of 37–40 cm H\(_2\)O inspiratory pressure and a PEEP of 15 cm H\(_2\)O compared with the control groups (PEEP of 5 cm H\(_2\)O or zero PEEP). This was corroborated in another paediatric study, in which a recruitment manoeuvre was required to return FRC and ventilation homogeneity to baseline values, after repositioning the patients from a Trendelenburg to a supine position. A recruitment manoeuvre should therefore be considered after any event during anaesthesia that may have caused significant \(V/Q\) loss, or where avoidance of atelectasis formation is pivotal.

In the critical care setting suctioning of pulmonary secretions through the tracheal tube may occasionally cause transient...
hypoaxia and a reduction in lung compliance, however most children seem to tolerate the procedure without significant respiratory deterioration. A short period of pre-oxygenation and limiting the duration of suctioning may reduce the incidence of desaturation; however, recruitment manoeuvres after suctioning as a routine measure do not seem to be universally beneficial in children.

### Positive end-expiratory pressure

The use of PEEP during anaesthesia has significant effects on lung function. Studies in children and adults have shown that the addition of PEEP is important in minimizing or preventing FRC reduction, airway closure, and subsequent atelectasis formation. Without PEEP, FRC may drift towards the lower and flatter portion of the P/V curve where compliance is reduced and lung collapse occurs. The optimal level of PEEP maintains tidal ventilation on the steep slope of the P/V curve and thus preserves FRC and respiratory system compliance without compromising the haemodynamic function, which can accompany significant derecruitment or overexpansion of the lungs. PEEP may increase compliance by 50% in anaesthetized neonates and infants and by ~25% in young children under general anaesthesia. Thorsteinsson and colleagues determined that the mean PEEP level required to optimize respiratory compliance was 6 cm H2O in infants and 12 cm H2O in older children. This has been corroborated in a paediatric study showing that 6 cm H2O, but not 3 cm H2O PEEP was required to maintain FRC and ventilation homogeneity under high inspired oxygen concentrations (FI02) during anaesthesia. Recent work however has suggested that despite successful antagonism of FRC loss with PEEP, there is a redirection of ventilation from the dependent to the upward part of the lung, which is not corrected by the application of PEEP.

### The impact of different breathing systems for manual ventilation

The modified Jackson Rees T-piece system is often advocated as the best bag and mask ventilation system for use in paediatric anaesthesia. Paediatric anaesthetists suggest that this system allows a good ‘feel’ of the lung compliance and is a simple circuit through which PEEP or continuous positive airway pressure (CPAP) can be applied. Compared with paediatric circle systems, however, the Jackson Rees systems impose a higher resistance to flow which negatively affects tidal volumes and minute ventilation, and some lack an adjustable pressure limiting (APL) valve, allowing excessively high ventilation pressures that may cause gastric insufflation. Not surprisingly, the anaesthetist’s level of expertise, significantly affects the degree of gastric insufflation caused by non-invasive positive pressure ventilation. In a study involving 1-to-six-year-old children, the investigators reported that gastric insufflation from ventilation with the use of a Jackson Rees system was most pronounced in inexperienced staff, and that subsequent nasogastric suctioning was required to improve FRC and ventilation homogeneity.

### Impact of extubation

Mechanisms maintaining respiratory stability such as protective reflexes and FRC defending activity in infants are compromised immediately upon extubation. As a result of inherent difficulties in obtaining ventilatory measures immediately after extubation, little objective data is available on the physiologic events accompanying extubation. Early work in critically ill adults suggested that extubation causes transient increases of min ventilation and inspiratory airflow. Observations of rapid oxygen desaturation after extubation under tracheal suction as opposed to extubation after bag inflation, and observational reports of improved extubation success in patients at high risk of extubation failure, when immediately placed on non-invasive ventilation, indirectly suggest that FRC loss is an imminent risk after extubation, and that this may be counteracted by immediate application of positive airway pressure by mask. There are however, no randomized studies yet to confirm these observations.

### Impact of patient positioning

Various patient positions may be required during anaesthesia and surgery. All have differing effects on lung volumes and mechanics. In awake patients, supine positioning reduces FRC by 25% compared with the sitting position. Putting children into the lateral position may increase FRC (but not compliance) both in anaesthetised spontaneously breathing and in ventilated children compared with supine position, possibly by a decreased abdominal pressure on the diaphragm. In the Trendelenburg position with 20–30° head down tilt, there is a reduction in FRC by up to 12% and a concomitant decrease of ventilation homogeneity and respiratory compliance in children, as a result of gravity-driven cephalad displacement of the diaphragm and an increase in thoracic blood volume. Prone positioning frequently improves oxygenation and lung volumes in lung disease, but has varying effects in healthy patients. The variation in findings between studies may in part be explained by the different techniques of prone positioning – primarily the presence or absence of ‘augmentation’, a technique that allows free movement of the abdomen during respiration, which leads to an ~20% increase in FRC compared with the flat prone and to the supine position in paralysed healthy children.

### Impact of cardiopulmonary bypass (CPB) and cardiothoracic surgery

CPB affects lung function through a number of mechanisms including the induction of an inflammatory lung response, pulmonary interstitial oedema and the effects of the interruption in pulmonary blood flow. Lung volumes and mechanics are highly dependent on pulmonary blood flow. Repair of septal defects, which are associated with a pulmonary hyperperfusion and pulmonary vascular congestion may result in improved airway resistance. In contrast, in congenital heart defects associated with pulmonary hypoperfusion, airway resistance may increase after surgical correction. In the absence of pulmonary circulation, the structure of the alveolar wall changes, resulting in decreased lung volumes and increased tissue resistance and parenchymal elastance. Subsequent re-introduction of pulmonary capillary blood flow after bypass induces reorganisation of the elastin fibres and correction of the alveolar geometry.

As a consequence, CPB is associated with a reduction in FRC by up to 18% in children undergoing open heart surgery resulting in impaired ventilation homogeneity. During the operation, lung function is additionally affected by the changes in chest wall mechanics. Opening the chest increases FRC by up to 20%, with subsequent removal of the retractor and closure of the chest abolishing this effect on FRC and worsening ventilation homogeneity. The application of PEEP is considered to counteract these adverse sequences. Upon completion of

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cardiac surgery involving CPB, lung volumes and homogeneity do not return to baseline for at least 90 min.\textsuperscript{182}

Conclusion
Perioperative disturbance in lung function may occur as a result of a number of anaesthetic and surgical factors. These changes in combination with a high resting oxygen requirement render children, particularly neonates and infants, vulnerable to hypoxaemia and respiratory complications. Anticipation, recognition and effective management of these changes are essential components to minimize the risks associated with anaesthesia in children. Furthermore, recognition of the different effects of various anaesthetic techniques will allow paediatric anaesthetists to tailor management for specific patients and specific surgical procedures.

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Revising paper: all authors

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