Chapter 9

Paediatric Respiratory Diseases

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Introduction

Respiratory failure, the second most common cause of death in infants, accounts for around 50% of intensive care unit admission of infants and children [1]. This is due to the wide array and high incidence of respiratory diseases in the paediatric age group. It is also due to the fact that respiratory function is particularly at risk. The limited ability of the developing respiratory system to compensate for disease-induced mechanical abnormalities makes the child susceptible to respiratory failure, which is very often the cause or the main effect of most paediatric emergencies. Respiratory function is critical for all organ systems of infants and young children. Early recognition of respiratory insufficiency is critical to allowing quick treatment, before progression of the vicious cycle of asphyxia and haemodynamic deterioration, which may lead to cardiorespiratory arrest (Fig. 1).

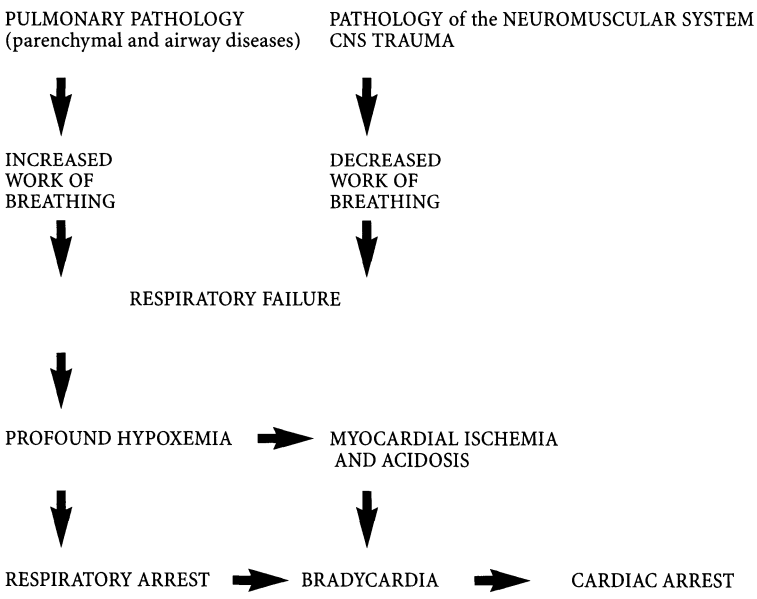

Fig. 1. Escalation of pulmonary disease emergencies

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However, it must be appreciated that since many lung development changes occur in children up to at least 8 years of age, there is a great chance for spontaneous healing and improvement in respiratory function in the paediatric age, even after critical conditions. The understanding of the respiratory dysfunction depends on the specific disease and also on the knowledge of the specific respiratory anatomy and physiology of the development stage of the single paediatric patient.

This paper reviewes some aspects of anatomy and physiology and the most frequent respiratory diseases the anaesthesiologist and the intensivist must face in the clinical practice beyond the neonatal age.

Anatomy and Physiology

After birth, the development of the lung can be divided into two phases. During the first phase, from birth to the first 18 months of life, there is a brisk increase in the surface and volume of the bronchioalveolar spaces while the capillary volume increases even more; the solid tissue grows more slowly. This process is very active in early infancy. Stimuli, such as hypoxemia and increased shear stress or tension in the vascular wall, can produce smooth-muscle hypertrophy and deposition of elastin and collagen, leading eventually to vasoconstriction and even obliteration of small pulmonary vessels. A failure to curtail this proliferative response is probably the origin of many cases of pulmonary hypertension complicating parenchymal lung diseases and right to left shunting in childhood. In the second phase, all compartments of the lung grow proportionately to each other and the configuration of the air spaces becomes more complex because of the development of new septa, and because of lengthening and folding of the alveolar structures [2]. Thus, the configuration of the air spaces become progressively more complex. At birth, the alveolar surface area is 2.8 m^2; it becomes 32 m^2 by 8 years of age and reaches 75 m^2 in adulthood [3].

The paediatric lung is less compliant than the adult lung because of the differences in the air space architecture and the amounts of elastin and surfactant. The amount of elastin increases over the first 18 years of life.

The airway enlarges in diameter and length with age. During the first 5 years of life, the growth of the distal airways lags behind that of the proximal airways; the narrow distal airway accounts for the high peripheral flow resistance in this age group. In infants and children, small distal airway resistance accounts for most of the work of breathing, whereas in adults the nasal passages provide the major proportion of flow resistance. The increased resistance to flow of the bronchi and bronchioles in the young results in increased work of breathing and increased vulnerability to diseases affecting the small airways.

Infants are described as obligate nasal breathers; thus, the obstruction caused by a naso-gastric probe may easily worsen the ventilation of a distressed child. The tongue is closer to the roof of the mouth and thus very easily obstructs the airway. Moreover, the infant's tongue is more likely to obstruct

the view of the larynx during laryngoscopy. The infant's larynx is higher in the neck (C3-4) than is an adult's (C5) and it is "rostral" in the neck compared with that of the adult subject. The narrowest portion of the infant and small-child larynx is the cricoid cartilage. Thus, an endotracheal tube which easily goes through the glottis can stop at the cricoid level.

Resistance to flow is inversely related to the fourth power of the radius; therefore, a small decrement in the diameter of the airways in infants and small children, such as an inflammatory oedema at subglottic level, may lead to a dramatic increase in airways resistance. Cartilagenous support is essential for the stability of airway conduct. After birth, cartilaginous tissue increases and spreads up to the segmental bronchi. The weakness of this support in infants and small children accounts for dynamic compression of the trachea and bronchi in conditions associated with high air-flow rates, such as with bronchiolitis and asthma. A very common cause of upper airway obstruction in children is adenoidal and tonsillar hypertrophy due to frequent upper airway infections. Extrathoracic airway obstruction caused by croup, epiglottitis or a foreign body alters the airway dynamics. A more negative intrathoracic pressure leads to an increased tendency toward dynamic collapse below the level of the obstruction. Particularly at the extremes of transluminal pressure that occur when a child is crying, the tendency to collapse is increased [4]. Avoiding dynamic collapse is thus very important. It is common for children with respiratory diseases to breathe transiently at frequencies that depart from optimum. Agitation can in this manner reduce the efficiency of the respiratory system and precipitate respiratory failure. For this reason, it is imperative to keep the child with an airway obstruction as calm as possible, avoiding any unnecessary, frightening procedure. A brisk increase in the work of breathing may also occur with long endotracheal tubes of small diameter, or with a partially obstructed tube, because of thick, viscous secretions. An obstructed endotracheal tube may cause a respiratory distress very similar to that in a bronchospastic attack.

The chest wall of the infant is cartilagenous, soft and pliable. The infant's chest can behave much like a "flail" chest [5]. Since the chest wall is so compliant, the elastic recoil may lead to excessive reduction of the lung volume, increasing the risk of lung collapse. In fact, most tidal breathing in the infant and small child takes place in the range of closing capacity. The relationship between functional residual capacity (FRC) and closing capacity determines the matching of ventilation and perfusion and thus the PaO_2. This is crucial because most paediatric respiratory diseases show an alteration of closing capacity or functional residual capacity or both. FRC is also acutely decreased during anaesthesia, which can be reversed by the administration of PEEP [1]. Poorly ventilated areas of the lung have a parallel reduction in blood flow because of hypoxic pulmonary vasoconstriction. Thus, the oxygen-desaturated blood from areas with low ventilation-perfusion ratio mixes with oxygenated blood from well-ventilated areas. A potential disadvantage of all halogenated agents is their tendency to blunt hypoxic pulmonary vasoconstriction. Thus, any poorly ventilated area of the lung, e.g. an atelectasis, is well perfused and

leads to desaturated and oxygenated blood mixing and hypoxemia. All injectable anaesthetics have no effect on hypoxic pulmonary vasoconstriction.

Units with high ventilation-perfusion ratios cause an increase of dead space ventilation, which can lead to a rise in arterial CO_2 if the child is unable to compensate with enough of an increase in minute ventilation.

The level of the pulmonary capillaries is the site of active exchange of water and solutes between the vascular space and the interstitium of the lung. This exchange is regulated across the endothelial cells by a balance of hydrostatic and oncotic forces. An increase of fluid filtration into the interstitial tissue and then into the alveoli may occur either when the microvascular pressure increases because of left ventricular failure or because of pulmonary vein obstruction or when the permeability of the endothelium is increased because of damage to the wall of the capillary vessels, which occurs in ARDS and in virtually any form of lung injury.

Oxygen demand is much higher per unit mass in infants than in adults. The higher oxygen consumption together with decreased oxygen supply in the lungs explains the inclination toward rapid hypoxemia and desaturation, with the appearance of cyanosis in infants and children during hypoventilation. This greater oxygen consumption accounts for the increased respiratory frequency, while tidal volume does not change much throughout age. In response to low oxygen, the newborn does not sustain an increase in ventilation. CO_2 response is also reduced in the young. Respiratory muscles seem to be more susceptible to fatigue in children than in adults. Thus, any condition that increases the work of breathing in children may easily fatigue the respiratory muscles and result in respiratory failure.

Paediatric Respiratory Life Support [6,7]

Cardiopulmonary arrest in infants and children is rarely a sudden event and respiratory causes predominate. Current guidelines use the term "newly born" to refer to the neonate in the first minutes to hour after birth. The term "infant" includes the neonatal period and extends to one year of age. For the purposes of resuscitation, the term "child" refers to ages 1-8 years and the term "adult" applies to all victims beyond 8 years of age.

Because most paediatric arrests are secondary to progressive repiratory failure and/or shock and because ventricular fibrillation is uncommon, immediate cardiopulmonary resuscitation ("phone fast") is recommended for infants and children in the out-of-hospital setting rather than the adult approach, which is immediate activation of the emergency system before applying initial cardiopulmonary resuscitation ("phone first"). Opening the airway must be done with caution in infants, avoiding excessive head tilt, because it is easy to collapse the airway. Between 2 and 5 rescue breaths should be delivered initially to ensure that at least two effective ventilations are provided. Mouth-to-mouth-and-nose ventilation must be used for infants. A rescuer with a small mouth may have dif-

ficulty covering both the mouth and the nose of a large infant. Under these conditions, mouth-to-nose ventilation is applied. For children, mouth-to-mouth ventilation is suitable, like in adults. The volume of each rescue breath should be sufficient to rise the chest without causing gastric distention. Improper airway opening is by far the most common cause of inadequate ventilation during resuscitation. Gastric distention can be minimized by delivering the rescue breaths slowly. Gentle pressure on the cricoid cartilage during ventilation can help decreasing the amount of air driven to the stomach. Bag and mask ventilation in infants and children must be handled without difficulty by a professional rescuer. Paediatric-size ventilation bags (450-500 ml) should be used to ventilate term newly born infants, larger infants and children. The "E-C clamp" technique provides adequate sealing to the face of the mask if applied gently but firmly, avoiding excessive head tilt, particularly in infants. Two-rescuer bag-mask ventilation (four-hands technique) may be used by unskilled rescuers.

Ventilation via a tracheal tube is the most effective and reliable method of ventilation and airway control. A pillow to flex the neck is not necessary for oral intubation of children < 2 years. For children older than one year, an estimate of tracheal tube size may be made using the following equation: tube size (mm) = (age/4) + 4.

The appropriate depth of insertion of a tracheal tube from the mouth may be estimated in children < 2 years of age using the following formula: depth (cm) = internal tube diameter (mm) x 3.

For children > 2 years of age the following equation must be used instead: depth (cm) = (age in years/2) + 12.

When it is impossible to oxygenate the child with bag-mask ventilation and when intubation cannot be accomplished, transtracheal ventilation may be attempted by needle-cannula cricothyrotomy.

Physiology and Pathophysiology of the Lateral Decubitus Position

Gravity causes a vertical gradient for pulmonary blood flow in the lateral decubitus position. Gravity also causes a vertical gradient in pleural pressure in the dependent lung, which places it in a more favourable position on the compliance curve [5]. The abdominal content exerts more pressure on the dependent lung, resulting in doming and better contraction of the diaphgram during spontaneous ventilation. The induction of anaesthesia does not change the distribution of pulmonary blood flow. There is a loss of functional residual capacity in both lungs, which may be greater in the dependent lung because of the weight of the mediastinum and the abdomen. The upper, non-dependent lung moves to the more compliant portion of the pressure volume curve, whereas the lower lung becomes less compliant. Ventilation-perfusion matching is disturbed and a decreased PaO_2 easily develops. PEEP can restore ventilation to the lower lung. As the chest is opened, the ventilation-perfusion mismatch worsens and postive-pressure ventilation becomes mandatory.

An important difference exists between adults and children regarding the lateral decubitus position in the presence of unilateral lung disease. In adults, gas exchange is optimal when the "good" lung is dependent in the lower position [8]. By contrast, infants with unilateral lung disease show better oxygenation when the "good" lung is not dependent. Infants, both those breathing spontaneously and those receiving positive-pressure ventilation, distribute more ventilation to the upper, non-dependent lung [9]. This may be due to the unstable rib cage, which does not fully support the underlying lung [10]. In children, the best side must be found watching SaO_2 changes with both lateral positions.

Respiratory Failure

Acute respiratory failure in paediatrics may be categorized into lung diseases of infants, in whom a single process involves only the lung, and those of older children, in whom pulmonary failure is often part of a multiple system dysfunction syndrome that is similar to that in adults with ARDS. Respiratory failure is defined as an alteration in arterial PaO_2 and $PaCO_2$. Respiratory failure caused by mechanical abnormalities ensues as the mechanisms that compensate for the increased work of breathing are not efficient enough to maintain normal or near-normal gas exchange. Mechanical abnormalities increase both the ventilatory requirement and the physical effort to fulfil these requirements. In addition to the increased work of breathing, many factors, such as malnutrition, electrolytes disorders and hypophosphatemia, increase the vulnerability of the ventilatory muscular pump. Even the increase of ventilatory demand caused by agitation or by an increase in temperature of the body may be sufficient to overcome the compensation mechanisms and precipitate significant hypoxemia and hypercarbia. An awake child with upper airway obstruction caused by croup or epiglottitis is much more stable in a mother's arms because the increased air flow generated by crying may easily precipitate respiratory failure. The clinical manifestations are the signs and symptoms of respiratory distress. Early recognition of respiratory distress is thus crucial to prevent escalation to respiratory failure.

By contrast, no distress is observed when respiratory failure is caused by neuromuscular or control abnormalities, and the only clinical clue is the observation of a decrease of the frequency and/or the depth of ventilation, which requires a very skilled eye and knowledge of the normal respiratory rate throughout infancy and childhood. Acute hypoxemia and hypercarbia are often associated with lethargy and confusion alternating with agitation and altered behaviour of the child.

Establishing a diagnosis and planning a therapy for children with respiratory diseases is aided by the clinician being able to distinguish between conditions that alter primarily the elastic (restrictive respiratory diseases) and the resistive (obstructive respiratory diseases) characteristics of the respiratory system [5].

Children with restrictive diseases breathe at fast rates, with shallow respiratory excursions. An expiratory grunt is common as the child attempts to raise the functional residual capacity by a partial closure of the glottis at the end of expiration. Lung percussion is usually dull and the auscultation reveals rales or crackles. Obstructive diseases are generally characterized by slower, deeper breaths. The pattern and the length of inspiration and expiration is very useful to diagnose the level of the obstruction: when the obstruction is extrathoracic from the nose up to the first half of the trachea, inspiration is more prolonged than expiration. With such conditions, an inspiratory stridor, can be easily heard. By contrast, expiration is more prolonged than inspiration when the obstruction is at the distal trachea, at the bronchi and at the level of the small airways. The child has to make use of the accessory muscles of the abdominal wall to eject the air out of the lungs. The auscultation reveals expiratory wheezes caused by turbulent flow in narrow airways.

Administration of supplemental oxygen is a safe and wise precaution for all children at risk of respiratory failure. Oxygen can be easily administered by nasal cannulas or hoods. Facial masks are not always well tolerated by infants and small children. The indication of ventilatory support is made on an individual basis considering the persistence or the worsening of gas exchange. Mechanical ventilation is life-saving, but can produce significant lung damage and adverse effects all over the body. Regardless of the underlying respiratory problem, the goal of the artificial ventilation is not to normalize arterial PaO_2 and $PaCO_2$, but rather to obtain adequate or acceptable gas exchange for the single patient, according to the underlying conditions. What can be considered adequate is very different at present from than it was some years ago. There is now a wide consensus among paediatric intensivists that some degree of hypercarbia and even some relative hypoxia is well tolerated and can reduce volutrauma and barotrauma. Permissive hypercapnia is considered an arterial CO_2 around 50-60 mmHg; its effects on pH are reduced by renal retention of bicarbonate. Moderate hypoxia, such as an arterial saturation around 85-90%, is also well tolerated if anaemia and insufficient cardiac output can be avoided. Lactate levels and mixed venous oxygen saturation may help in identifying the adequacy of cardiac output.

Inhaled nitric oxide may improve PaO_2 by reducing pulmonary vascular resistance.

Upper Airway Obstruction

The Child with a Runny Nose [11]

It is very common to evaluate a child scheduled for surgery, with symptoms and signs of a possible upper respiratory tract infection. There are many causes for a runny nose in children and each patient must be considered on an individual basis. It is very important to take a careful history and make an accurate diagnosis of the cause of the symptoms. Often, the parents state that their child always

has a runny nose. The history may point to an acute onset of coryza. The examination of the mouth, nose and throat may help in identifying the amount and type of secretions and other evidence of an acute infection. An elevated temperature may suggest an infection, but a minor increase is seen frequently and may not be associated with infections. The problem is that there is an increased incidence of intraoperative and postoperative airway complications, such as episodes of desaturation and laryngospasm, in children with upper respiratory infections. The decision to postpone surgery is not without consequences for the child and the family because of many economic and emotional implications. However, it may be appropriate to be conservative, expecially if an endotracheal intubation is planned. The risks versus the benefits of proceeding with general anaesthesia should be discussed frankly with the parents and the surgeon.

Congenital Larynx Abnormalities [12]

Congenital laryngeal webs and other anomalies of the larynx may be observed after birth. Stridor appearing in the first month of life is generally the result of laryngomalacia and tracheomalacia. The diagnosis is made by direct laryngoscopy. The symptoms of airway obstruction can be intermittent. Often they are worse when the infants lie on their backs and when an upper airway infection is superimposed on already narrowed airway. Stridor can also be caused by other malformations, such as cysts, haemangiomas and vascular anomalies. For this reason, a complete, accurate bronchoscopy is always indicated for these patients. Laryngomalacia usually resolves spontaneously during early infancy. Rarely, the infant must be intubated, and even more rarely, the patient requires a tracheostomy. Laryngeal abnormalities may require surgical intervention or laser treatment [13].

Croup (Laryngotracheobronchitis) [14]

The term croup includes a group of acute, often infectious conditions characterized by a "croupy" cough. Inspiratory stridor, dyspnea, hoarseness and signs of respiratory distress, more or less pronounced, are always evident in the most severe cases. Symptoms are usually worse at night and increase quickly during the hours following onset. Agitation and crying aggravate the symptoms and may even hasten respiratory failure. The parainfluenza viruses account for 75% of cases. Occasionally, other viruses and bacteria may be involved. A radiograph of the upper airway shows very often the typical subglottic narrowing (steeple sign). Oxygen must always be administered in the hospital. Worsening hypoxemia and fatigue may ultimately lead to respiratory failure: the child is usually cyanotic, pale or obtunded. At this time, any manipulation of the pharynx may result in respiratory arrest. In a child showing signs of progressive fatigue, endotracheal intubation may become necessary (see "Epiglottitis"). A spasmodic type of croup occurs in children of 1-3 years of age and may be caused by viruses and other allergic or psychological factors. At laryngoscopy, a posterior

laryngitis is usually seen, suggesting gastroesophageal reflux. It usually occurs in the evening or at night time and is characterized by a sudden onset, with no fever in most cases. The dyspnea is aggravated by excitement and agitation. Croup may recur several times.

Epiglottitis [14]

This is an acute, life-threatening inflammation of the entire supraglottic region with a very rapid clinical course. It is now seen much less commonly since immunization against *H.influenzae* type B. The child, usually 2-7 years old, is well until the onset of high fever, severe sore throat, and inspiratory dyspnea. Prostration and fatigue ensue shortly thereafter. Within a few hours, the clinical course is complicated by progressive complete obstruction of the airway and death unless effective treatment is provided. Dysphagia is usually associated with drooling of saliva from the mouth. Painful or frightening procedures may result in sudden complete airway obstruction and cardiac arrest. If an epiglottitis is suspected, no attempts at stabilization, such as lying the child down, establishing an intravenous line or a forced visual inspection of the pharynx must be done. Oxygen is administered, avoiding constrained manoeuvres. The child must remain in the posture chosen, usually an upright position, stay together with the parents, and immediate transport to the hospital must be arranged. In the hospital, radiographs are performed to clarify the diagnosis only if the child is stable, and in the presence of a physician able to perform endotracheal intubation. As soon as possible, it is my practice to bring the patient to the operating room, trying to maintain a calm atmosphere. Anaesthesia is induced with sevoflurane in oxygen and a laryngoscopy is performed. The anatomy may be extremely distorted and sometimes it is even difficult to identify the glottic structures. The sight of air bubbles during exhalation may be the only clue to find the vocal cords. A small endotracheal tube is then inserted into the trachea. After some time a larger tube may be positioned in the trachea, as compression of the previous tube in the oedematous tissue enlarges the glottis passage. A tracheostomy is very rarely needed. After successful airway management, the child is sedated, treated with an antibiotic (ceftriaxone 100 mg/kg iv) and transferred to the pediatric intensive care unit for 48-72 h with assisted ventilation (CPAP). Acute pulmonary edema is not unusual after the relief of airway obstruction. Usually, this complication resolves within 24 h following appropriate treatment (oxygen, CPAP, diuretics).

Lower Airway Obstruction

Bronchiolitis [15]

This is an inflammatory obstruction of the small airways which occurs in the first two years of life, with a peak incidence at 6 months of age, mainly in winter and early spring. Respiratory syncytial virus is the aetiologic agent in more than

50% of all cases. The bronchial obstruction is due to edema and accumulation of cellular debris and viscous mucus. Since the resistance to flow is inversely related to the fourth power of the radius, the thinning of the bronchioli profoundly affects the air flow. Because the radius is even smaller in expiration, the ball-valve obstruction leads to air trapping and alveolar overinflation. The infant is usually tachypneic, in clear distress, with intercostal and subcostal retractions. Wheezings are often evident at auscultation. SaO_2 is reduced. The initial treatment is placing the patient in an atmosphere of warm humidified oxygen to relieve hypoxemia. Ribavirin is an antiviral agent which is currently used only for patients with bronchopulmonary dysplasia or cardiac abnormalities. Antibiotics are used if there is an associated pneumonia. Corticosteroids are used often to reduce inflammation. Some patients require endotracheal intubation and ventilatory assistance if $PaCO_2$ rises above 50 mmHg or severe hypoxemia persists despite oxygen therapy. Mechanical ventilation must consider the pathophysiologic basis of hyperinflation (see asthma).

Asthma [16,17]

Asthma is the most common chronic disease of childhood. It is a leading cause of school absence and a common cause of hospital and intensive care admissions, too. The disease is intermittent and reversible and is characterized by narrowing of the airways due to inflammation, bronchospasm and accumulation of sticky mucus. The episodes may result in life-threatening respiratory failure. It is believed that allergy plays an important role in asthma. Airflow obstruction causes hyperinflation because of on expiratory ball-valve mechanism. Hypoxemia, hypocapnia and respiratory alkalosis are usually seen early in acute attacks. Ventilation/perfusion abnormalities result from non-uniform narrowing of the airway and changes in blood flow from the high intralveolar pressure, causing maldistributions in perfusion. Hypocapnia is due to hyperventilation. A normal level of $PaCO_2$ in the presence of respiratory distress means severe obstruction because of inadequate removal of CO_2. Elevated $PaCO_2$ occurs as the $FEV_{1.0}$ falls below 20% of the predicted value, and it indicates muscular exhaustion. Children with respiratory acidosis must be monitored closely in an intensive care unit. Intubation and mechanical ventilation must be considered when pharmacologic therapy [18] (beta-2-agonists, ipatropium, steroids, amynophilline) has failed to prevent fatigue and worsening hypercapnia. There are no absolute guidelines for initiating mechanical ventilation in status asthmaticus, except cardiopulmonary arrest; however, the following criteria are usually considered when deciding on an individual basis:

1. Coma or deterioration of mental status.
2. Progressive exhaustion.
3. Decreased wheezing associated with absence of breath sounds on auscultation.
4. Cyanosis or $SaO_2 < 85\text{-}90$ or $PaO_2 < 55\text{-}60$ mmHg, despite oxygen therapy.
5. Hypercapnia, $PaCO_2 > 50$ or rapidly increasing.

A suggested sequence for intubating an asthmatic child is as follows: pre-oxygenation with 100% oxygen, atropine 0.01 mg/kg iv, lidocaine 1-2 mg/kg iv, midazolam 0.2-0.3 mg/kg iv, ketamine 2-3 mg/kg iv, Sellick manoeuvre (cricoid compression) and an intravenous myorelaxant, such as suxametonium 1-2 mg/kg or rocuronium 1 mg/kg. Suxametonium is a better choice if a difficult intubation is suspected. However, it should be avoided if there is hyperkalemia, which is not unusual because of the beta-2 stimulation. Bag and mask ventilation with 100% oxygen prior to intubation may be difficult, but it becomes crucial to avoid dangerous hypoxemia after use of the drugs. Cricoid pressure may help in reducing gastric distention. Alternatively, halogenated agents, such as sevoflurane, may be used to induce anaesthesia during assisted ventilation. A chest radiography is always obtained after intubation to determine the position of the endotracheal tube and to get a "starting point" of x-ray assessment.

Mechanical ventilation must take account of the concept of limiting the risk of hyperinflation, barotrauma and volutrauma. Permissive hypercapnia is the recommended choice to be kept in mind in ventilating asthmatic children. Pressure-ventilation is often chosen to limit barotrauma. Expiration times must be long enough to allow air flow out of the obstructed bronchi, thus reducing air trapping. PEEP is generally not recommended, even if some centers use it, expecially during assisted spontaneous ventilation. Controlled ventilation is used initially. Then, pressure support may be chosen to facilitate weaning from the ventilator. Most chidren are ventilated for 36-72 h. The initial setting is usually as follows:

1. Peak pressure < 40 mmHg.
2. Tidal volume around 6-8 ml/kg.
3. Frequency around half the normal value for the age.
4. Inspiratory/expiratory ratio equal to 1/3 or 1/4.
5. No inspiratory pause.
6. PEEP: 0-3 cmH_2O
7. FIO_2 as needed to obtain SaO_2 90%.

Subsequent adjustment should be done on the basis of clinical adequacy of ventilation and blood gases. $PaCO_2$ is maintained around 50-60 mmHg, with pH > 7.20. Monitoring includes pressure-volume curves, intrinsic PEEP (by means of end-expiratory breath hold) and plateau pressure (by means of end-inspiratory breath hold). Sedation and sometimes myorelaxation may be required to promote synchronization of the ventilator with breathing, which is favourable in reducing oxygen consumption and CO_2 production. Midazolam and ketamine (2-8 µg/kg/min) are the most frequent drugs used. Morphine is used with caution because of the possibility of worsening bronchospasm by morphine-induced histamine release. Drugs which cause hypotension are better avoided and it may become necessary to sustain cardiovascular function by inotropes.

Inhalational anaesthetics have been used in severe asthma. Isoflurane may be used in the ventilator circuit by progressive 0.1% increases, witholding all other sedatives until bronchospasm is reduced. Hypotension is the main concern. Intravenous fluids and/or vasoconstrictive agents may become necessary. The

administration of magnesium sulfate 25-50 mg/kg iv, maximum 2 g, has gained interest in the treatment of asthma. Some improvements have been shown in recent studies. High doses of ketamine by continuous infusion, up to 40 µg/kg/min, may contribute to the succesful treatment of refractory bronchospasm.

Foreign Bodies in the Respiratory Tract [6,19,20]

Most reported episodes of choking in children occur during play or eating, when parents or care providers are usually present. A foreign body which occludes the laryngeal inlet is an immediate threat to life. When a foreign body produces signs of complete airway obstruction, only a prompt relief may allow ventilation. Smaller objects or a piece of food may lodge in the main stem or lobar bronchus; the symptoms consist of coughing and sudden respiratory distress. After the initial symptoms, which may be forgotten, there is often a symptom-free period that may last hours, days, or even weeks.

If a responsive infant demonstrates signs of complete obstruction of the airway and spontaneous coughing is not successful to clear the airway, the rescuer must provide back blows and chest thrusts until the object is expelled or the victim becomes unresponsive. For the older child (1-8 years of age) a series of Heimlich abdominal thrusts are used to force air and the foreign body out of the airway. If the infant become unresponsive, the sequence of ABC of resuscitation is followed by the professional rescuer: open the victim's airway and attempt to provide rescue breaths. If the breaths are not effective, perform 5 back blows and 5 chest thrusts. This sequence is repeated until advanced life-support facilities are available. If the child becomes unresponsive, place the victim in the supine position, open the airway, and attempt to provide rescue breaths. If the repeated artificial breaths are not effective, the Heimlich manoeuvre is performed and the sequence is continued, alternating airway opening and inspection, rescue breaths and subdiaphragmatic thrusts.

Partial obstruction of the airway is more common if the object lies in the carena or the main bronchi. A non-obstructing, non-irritating foreign body may produce few symptoms. If there is a slight obstruction, a wheeze is produced. If the obstruction is severe, either overinflation or atelectasis may ensue. A ball-valve obstruction produces overinflation of the dependent area because air flow is interrupted in expiration, as the radius of the tube becomes smaller. Complete obstruction, by contrast, produces an atelectasis, as the air distal to the obstruction is reabsorbed. If the foreign body is a vegetable, such as a peanut, a severe bronchitis results, characterized by cough, mucus production, fever and worsening dyspnea. Chronic suppuration may occur if the vegetable remains in the airway for a long time. A chest X-ray may not reveal any change and it is always advisable trying to obtain films taken in deep inspiration and deep expiration. Early inspection of the airway and possible removal by rigid open-tube bronchoscopy is always indicated even if the presence of a foreign body is only suspected.

Aspiration Pneumonia [21]

Children with gastroesophageal reflux or impaired consciousness and altered airway reflexes may regurgitate and aspirate food and vomitus. Hydrocloridric acid is the main determinant, but not the only one, of chemical lung injury. The child may show sudden distress just after the aspiration of a large amount of vomited food. Fever and tachypnea are usually present within 2 h after the episode. The chest X-ray reveals localized or, more often, bilateral infiltrates. Superimposed infection is very common. Immediate treatment consists of suctioning to reduce the chance of continuing aspiration. Oxygen is always used as needed. Endotracheal intubation and mechanical ventilation become necessary in severe cases. Clearing of infiltrates occurs within two weeks in most children. Hospitalized chronically ill children may become colonized and infected by gram-negative flora. Previously healthy children are often infected by mouth flora, predominantly anaerobes.

ARDS [22-24]

Acute respiratory failure may occur after injury to the alveolar-capillary unit after a variety of insults in children with previous healthy lungs. It is often unclear whether the precipitating events are causative or merely associated phenomena. Shock, sepsis, aspiration pneumonia and diffuse infectious pneumonia are the most common causes in published pediatric series. The pathology and pathophysiology of the syndrome described in adults are not different than those in paediatric patients. The open lung approach, now much advocated in adult patients with ARDS, was first demonstrated in the young-animal lung lavage model of acute lung injury. It has been demonstrated that, in immature lambs, as few as six large tidal-volume breaths before the first spontaneous breath, a common scenario in the delivery suite, results in significant lung damage and blunts the effect of the exogenous administration of surfactant [25]. This concept was then applied using high-frequency oscillatory ventilation (HFOV) to treat premature infants. It is now clear that cyclic lung distention delivered by positive pressure ventilation can produce changes similar to those in ARDS in the lungs and damage to entire organ systems outside of the lungs. This damage may be mitigated by using small tidal volumes and high PEEP. Thus, oscillatory ventilation has a rationale to decrease lung damage during ventilation. If the clinician is to use HFOV, early intervention before the lung sustains significant damage is crucial. This has been demonstrated in a study in which premature infants were placed on HFOV in the delivery room [26]. If conventional ventilation is used in paediatric ARDS, a tidal volume as low as 5-6 ml/kg is associated with a reduction in mortality among patients with acute lung injury despite a significant degree of hypercapnia. Liquid ventilation is a very interesting concept in the treatment of ARDS, but the comparison of partial liquid ventilation with conventional ventilation in paediatric hypoxemic respiratory failure did not show any advantage in using the liquid ventilation

approach. There are data which show improvements in gas exchange in the prone position in paediatric patients [27, 28]. The role of extracorporeal membrane oxygenation (ECMO) in the treatment of children with pulmonary failure in this new era of lung-protection ventilation strategies remains to be determined. At present, when the facilities and experience with ECMO are limited, the value of this approach may only be judged on a case-by-case basis.

Thoracic Trauma [29-31]

Trauma remains the single most common source of morbidity and mortality among children up to 14 years of age. Thoracic injuries may account for around 10% of admissions to trauma centres and they remain a substantial source of morbidity and mortality. The great flexibility of the thoracic cage in children allows the traumatic stress to propagate into the chest and thus pulmonary contusion is common, whereas rib fractures occur less frequently in children than in adults. Around 70% of chest injuries in children are the result of blunt trauma and very often there is not any external evidence of the trauma on the skin and the wall of the chest, such as rib fractures and brusing. If rib fractures are observed there is always a severe pulmonary contusion and other thoracic or abdominal organs may be involved. Chest radiographies are usually taken, but younger patients are more likely to have injuries without plain-film abnormalities. Chest computed tomography is very useful in the evaluation and management of thoracic injuries. Lung injury usually manifests as areas of consolidation. Pneumothorax may be missed by plain X-ray of the chest. Unilateral absence of breath sounds, tracheal deviation away from the affected site, jugular venous distention and hyper-resonance of the ipsilateral thorax indicate tension pneumothorax. The child with tension pneumothorax demonstrates severe respiratory distress and haemodynamic instability. The condition of the child may rapidly worsen as positive-pressure ventilation is started. Emergency treatment requires needle decompression, very often before obtaining a confirmatory chest X-ray. An over-the-needle catheter is inserted through the second intercostal space on the midclavicular line, just above the third rib. Injuries to the tracheobronchial tree are rare. They occur usually in the distal trachea or proximal bronchi. Most exhibit mediastinal air even if distal injuries may manifest as pneumothorax. Other findings include large air leak from the chest tube and cervical emphysema. Relevant hemothoraces occur in 15% of children sustaining blunt-force chest trauma. Prompt drainage of blood is necessary because pockets of blood are excellent culture media for bacteria. Antibiotic use for chest injury is controversial. However, circumstances of soft-tissue injury, pulmonary contusion with hemoptysis and need for operative intervention may well be indications for antibiotic prophylaxis.

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