Paediatric aeromedical transport and hypoxia

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Introduction

Paediatric patients have different anatomical and physiological parameters when compared to the adult population. These differences are consistent and well described[1], with some of them rendering the infant more susceptible to hypoxia[2]. Aeromedical staff require a sound knowledge of these differences to properly treat the paediatric patient.

Paediatric anatomy and physiology

Paediatric body mass and morphology

Paediatric patients have lower body mass, less fat and connective tissue, and are morphologically different to adults[1]. In addition, their vital organs are in close proximity to the skin and the head is proportionally larger. The surface area to volume ratio is highest at birth and decreases as the child grows. This high ratio results in greater loss of thermal energy, and the paediatric patient is more prone to hypothermia[1].

Respiratory

Paediatric patients also have significantly different respiratory parameters to adults. They have higher respiratory rates, ranging from 40 to 60 breaths per minute in the infant, whereas an older child breathes 20 times per minute[1]. Spontaneous tidal volumes vary from six to eight mL/kg[1]. For the apnoeic child, mechanical ventilation is required and in this case tidal volumes are between 10 to 15 mL/kg and the minute volume is approximately 100mL/kg/min[3]. The paediatric patient also has an immature tracheobronchial tree. The developing respiratory system is relatively fragile and more prone to barotrauma than the adult. Hypoxia is the commonest cause of cardiac arrest in the child[1].

Cardiovascular

The circulating blood volume of the child is also different to the adult. The child has a circulating blood volume of approximately 80ml/kg, whereas in the adult it is 70 ml/kg[1]. The increased physiologic reserve of the paediatric patient allows for preservation of most vital signs in the normal range, even when the child is shocked[1]. Pulse rates up to 160 may be normal in the neonate. This figure decreases with age until it drops below 100 by the onset of adolescence.

Other considerations

Venous access is more difficult to achieve in the paediatric patient, and must always be secured prior to emplaning. The retrieval team should also be aware that the infant urinary output is higher than that of the adult. (2.0 mL/kg/hr in the infant decreasing to 1.0mL at age three to five years, and 0.5mL by adolescence[1]). Paediatric patients also have different psychologic needs when compared to adults.

An increased susceptibility to hypoxia

The anatomical and physiological differences between infants and adults are marked, and the response to the hypoxic environment encountered during flight is different[3]. The susceptibility to hypoxia is most marked in newborns and infants in the first year of life. Table 1 contains a summary of the factors increasing the susceptibility of infants and young children to hypoxaemia.

Hypobaric hypoxia of aeromedical retrieval

Atmospheric pressure falls in an approximately exponential manner with increasing altitude[12]. With increasing altitude there will be a corresponding decrease in barometric pressure. The barometric pressure at Mean Sea Level (MSL) in standard atmospheric conditions is 760mmHg, and this falls to 565mmHg at 8,000feet[12]. Dalton's Law of Partial Pressures states that each gas in a mixture exerts the same pressure as if it were present, alone, in the same volume[13]. Oxygen partial pressure (PO_2), or oxygen tension, is the portion of the total pressure that is exerted by the oxygen alone. The PO_2 difference between two areas determines the direction and rate of flow (diffusion) of oxygen molecules, including when the oxygen is in solution. A PO_2 gradient exists in different locations within the respiration cycle, allowing oxygen flow to occur, from regions with higher levels of PO_2 to regions with lower levels of PO_2 – this is called the oxygen cascade[12].

At the maximum cabin altitude of 8,000 feet, the atmospheric pressure is 565 mm Hg, giving an atmospheric PO_2 of 118 mm Hg. This is equivalent to reducing the fraction of inspired oxygen (FiO_2) at sea level to 15.5%. It can be seen that many of the anatomical and physiological parameters observed in the infant predispose them to an increased tendency to ventilation-perfusion mismatch. This results in infants being particularly susceptible to hypoxaemic episodes[14].

Anatomical or Physiological parameter	Mechanism of hypoxaemia
Reduced surfactant - preterm	Atelectasis and hypoxia
Rib cage more compliant	Less support of lung volume - more marked during sleep
	Negative intrathoracic pressures are less effective for inspiring air
Predisposition for paradoxical inhibition of respiratory drive < 2 months of age	Infections and hypoxia may present with apnoeic episodes and hypoventilation
Increased proportion of muscular arterioles in the pulmonary vascular bed (early infancy)	Airway or alveolar hypoxia causes pulmonary vasoconstriction[4] (Hypoxia may arise from hypobaric hypoxia, chest infection or chronic lung disease)
	Rise in pulmonary vascular resistance contributes to right to left shunting, ductal opening (in the early neonatal period), further ventilation-perfusion mismatch, and hypoxia
Increased airway reactivity in response to hypoxia (infancy)	Airway or alveolar hypoxia in infants can cause bronchoconstriction[5,6]
	Infants at 26 weeks of age show greater desaturation on histamine challenge than infants 4 weeks old[7]
Lung volume at end expiration similar to closing volume[8] (early infancy)	Small airway closure, and hence non-ventilated units, occur more readily, e.g. during active sleep, feeding, and crying
Reduced upper and lower internal diameters of the airways	Airway conductance falls from birth to 2 months of age[9]
	Reduction in diameter reduces airway patency sooner[10] e.g. respiratory infection
Fewer alveoli (early childhood)	Growth in the alveolar region greater than that in the airways in early infancy[11] Increases the susceptibility to mismatch between ventilation and perfusion
Foetal haemoglobin present up until 3 to 6 months of age	Oxygen dissociation curve is shifted to the left,so oxygen is given up less readily to the tissues. For a given PO_2, the SaO_2 is higher

Table 1: Factors increasing the susceptibility of infants and young children to hypoxaemia. Based on: Samuels M. The effects of flight and altitude. Archives of Disease in Childhood 2004;89:448-455 Table 2.

Level of respiration cycle	PO_2 Sea level	PO_2 8,000 feet
Ambient air	**160**	**118**
Inspired (tracheal)	148	108
Alveolar	103	64
Arterial	95	56
Capillary	51	30
Mitochondrial	**1-10**	**1-5**

Table 2: The Oxygen Cascade showing the PO2 at different stages of the respiratory cycle, in the resting subject breathing dry air: at Sea level – atmospheric pressure 760mmHg; and, 8,000feet – atmospheric pressure 565mmHg. Adapted from Ernsting, Nicholson, Rainford. Aviation Medicine 4th edition. Hodder Arnold 2006. Fig 2.14 p39, Table 3.2 p 45, Table 31.13 p 501.

Respiratory considerations during aeromedical retrieval of the infant

Decreased surfactant

Pre-term infants will often have decreased levels of lung surfactant predisposing to atelectasis. Atelectasis decreases ventilation, leading to a ventilation-perfusion mismatch and hypoxia[2]. Positive end-expiratory pressure may be necessary to overcome the tendency to atelectasis. Aeromedical transfer of the neonate should be performed using dedicated equipment suited to the physiology and anatomy of the newborn. In particular ventilators should have parameters that are appropriate for the patient, including safety valves to prevent pulmonary barotrauma during assisted ventilation.

Increased rib cage compliance

Neonates and young children display increased rib cage compliance[2]. Negative intrathoracic pressure generated during caudal diaphragmatic excursion is thus less effective at inspiring a volume of air. This is because the negative pressure generated is partly offset by a slight decrease in ribcage volume. Aeromedical staff should also be aware that infants predominantly use their diaphragm for respiration.

Paradoxical inhibition of respiratory drive

In infants younger than one to two months of age, the normal stimulus to ventilation caused by hypoxia

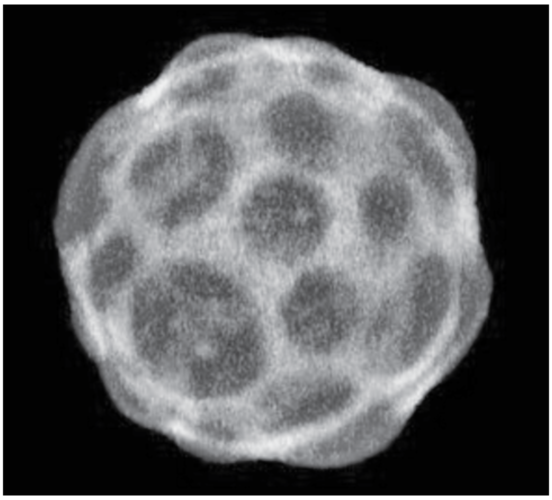

Figure 1: Fluorescent microscopy of lung surfactant. The lipid from the lung forms a liposome with bulges. The diameter of the liposome is approx. 17um. Fluorescent microscopy by Professor Luis Bagatolli Membrane Biophysics and Biophotonics group, University of Southern Denmark.

is sometimes followed by paradoxical inhibition of respiration[2]. Infants, who may have a tendency to become hypoxic due to prematurity or concurrent lung infection, may display hypoventilation or apnoea when exposed to hypobaric hypoxia during aeromedical transfer. Therefore, it is vitally important in very young infants that the aeromedical staff ensure their patient is not exposed to hypobaric hypoxic conditions. Methods to minimise this would include: the use of supplemental oxygen to increase the FiO_2, the maintenance of a sea-level cabin pressure, and by ensuring there are no intervals of disconnection of supplemental oxygen - especially when emplaning and deplaning the neonate.

Increased preponderance of muscular arterioles in pulmonary vasculature

Exposure to hypoxic conditions, such as those encountered during aeromedical transfer will result in vasoconstriction of the pulmonary vessels[4]. In early infancy this leads to a marked decrease in perfusion of the respiratory system. The raised pulmonary vascular resistance may lead to increased right to left shunting through a patent foramen ovale, resulting in non-oxygenated blood returning to the systemic circulation. In addition, exposure to hypoxia during flight may prolong the patency of the ductus arteriousus[3].

Increased airway reactivity in response to hypoxia

Some infants display increased airway reactivity when exposed to hypoxic conditions[5,6]. This reactivity actually increases following birth, to the point where at 26 weeks of age an infant shows greater desaturation upon histamine challenge when compared to a four week old neonate[7]. Aeromedical transfer of an infant with bronchiolitis (which is characterised by bronchoconstriction) is thus made particularly hazardous if the infant is exposed to hypoxia. For this reason hypoxic exposure should be minimised as much as possible.

End expiratory lung volume approximates closing volume

Closing volume (CV) is the volume of gas in the lungs in excess of the residual volume (RV) at the time when small airways in the dependent portions of the lungs close during maximal exhalation. The closing capacity (CC) is equal to CV plus RV. The closing volume is greater in young children in whom the elastic supporting structure of the lung is incompletely developed. Infants are at greater risk for atelectasis as airway closure can occur even during tidal breathing[15]. A complete explanation can be found in Fact Box A[6, 16].

Fact box A - Closing volumes and the alveolar unit

The existence of an air volume maintained within an alveolus is dependant on that unit obeying the laws of physics. The ambient pressure within the alveolus must be greater than the opposing combined forces of surface tensionand elastic stretch of the alveolar tissue. Surface tension increases as the distance between adjacent alveolar cells decreases. (The tension force is inversely proportional to the square of the alveolar radius). If the alveolar walls are very close, such as occurs in end expiration, the surface tension may be high enough to effect closure (via apposition) of some alveolar units. In other words, if the end expiratory lung volume is low enough some of the ventilatory units will close off completely[8]. Low end-expiratory volumes are potentiated during active sleep. In addition, there are lower alveolar pressures during feeding phases (sucking) and when the infant cries (at the immediate beginning of deep and forceful inspiration). This is one reason why some infants may become cyanosed during feeding and crying episodes. The aeromedical staff should provide supplemental oxygen and also facilitate a calm restful environment for the infant. The addition of 5cm H_2O of Positive End-Expiratory Pressure (PEEP) is sufficient to recruit.

Decreased airway diameter

The internal diameter of the airway in infants is proportionally smaller than the adult. During the first two months of life, when most physical parameters in the newborn are increasing rapidly (such as weight and length), the conductance of the airway actually decreases[9]. From the age of two months conductance begins to increase. Any reduction in airway diameter will effect a dramatic decrease in ventilation[10]. This is because resistance to flow is inversely proportional to the fourth power of the radius of the airway (Poiseuille's equation)[13]. Aeromedical staff should be aware that airway compromise in the neonate and infant can occur precipitously due to this physical fact. Aeromedical staff should have appropriate equipment available, such as suction devices to help clear an airway that suddenly becomes occluded.

Fewer alveoli

The respiratory system of the newborn displays significantly different morphology to the adult system. In particular there are proportionally fewer alveoli in the infant[11]. This can predispose to ventilation – perfusion mismatch, and thus hypoxia.

Foetal haemoglobin

Most types of normal haemoglobin, including haemoglobin A, haemoglobin A2, haemoglobin S, and haemoglobin F, are tetramers composed of four protein subunits and four heme prosthetic groups. Whereas adult haemoglobin is composed of two alpha and two beta subunits, foetal haemoglobin is composed of two alpha and two gamma subunits, commonly denoted as a_2y_2. Because of its presence in foetal haemoglobin, the gamma subunit is commonly called the "foetal" haemoglobin subunit. Foetal haemoglobin (HbF) persists in significant amounts up to three months of age and shifts the oxygen dissociation curve to the left[3]. The effect of foetal haemoglobin on the oxygen dissociation curve will be to enhance loading of oxygen in an hypoxic environment, and possibly to decrease unloading in peripheral tissues[17].

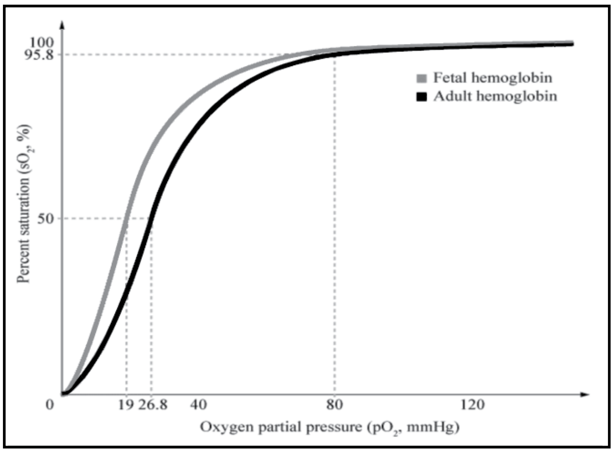

Figure 2: The oxygen saturation curve for foetal haemoglobin (grey) appears shifted to the left when compared to adult haemoglobin (black) since foetal haemoglobin has a higher affinity for oxygen.

Foetal haemoglobin's affinity for oxygen is substancially greater than that of adult haemoglobin. Notably, the P50 value for foetal haemoglobin (the partial pressure of oxygen at which the protein is 50% saturated - lower values indicate greater affinity) is 19 mmHg, wheras adult haemoglobin has a value of approximately 26.8 mmHg. As a result, the oxygen saturation curve, which plots haemoglobin saturation against pO_2 is shifted to the left for foetal haemoglobin.

This greater affinity for oxygen is explained by foetal haemoglobin's interaction with 2,3-diphosphoglycerate (2,3-DPG). In adult red blood cells, this substance decreases the affinity of haemoglobin for oxygen. It is also present in foetal red blood cells, but does not interact with foetal haemoglobin, leaving its affinity for oxygen unchanged. Adult haemoglobin alone actually has a higher affinity for oxygen than its foetal equivalent.

Barometric pressure changes

The volume of a fixed mass of gas is inversely proportional to the pressure to which it is subjected (Boyle's law). At the highest cabin altitude usually encountered in aeromedical retrieval flight (8,000 feet), gas will increase in volume by approximately 30 per cent. Gas trapped in a body cavity of a paediatric AME patient will expand at altitude and restrict diaphragmatic motion, compromising respiration and leading to hypoxaemia. Aeromedical staff should consider placing an orogastric or nasogastric tube to decompress the stomach prior to flight.

Hypoxia secondary to congenital pulmonary anomalies and cyanotic heart disease

Infants with congenital pulmonary anomalies are at risk for the development of spontaneous pneumothorax during flight[3]. In the paediatric AME patient there may be no obvious symptoms of a developing pneumothorax other than the development of unexplained variations in vital signs and abnormal movements. Paediatric AME staff should thus be vigilant for sudden changes in vital signs, and be aware of the risk of pneumothorax.

Physical phenomena causing hypoxia

The low humidity found in aircraft cabins can increase airway reactivity in some patients. In addition, bronchial secretions may thicken, leading to mucous plugging and atelectasis[3]. The resultant ventilation-perfusion mismatch will lead to hypoxia. Mucous plugging is of special concern in paediatric patients with bronchiectasis and cystic fibrosis.

Figure 3: This cyanosed neonate is just two hours old. The infant has been diagnosed with transposition of the great vessels and a ventricular septal defect. Aeromedical transfer of this neonate is a difficult and complex undertaking. The infant may exhibit dramatic episodes of desaturation when exposed to the flight environment [3].

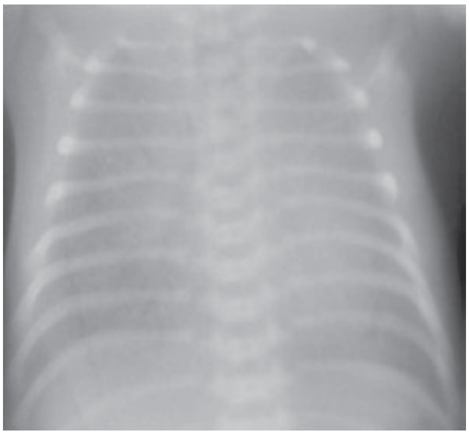

Figure 4: The characteristic CXR appearance of respiratory distress syndrome of the newborn, (previously hyaline membrane disease), showing the "ground glass" appearance. Usually due to low lung surfactant levels from prematurity. The alveoli are lined with cellular debris, fibrin, and inflammatory cells. The debris blocks gaseous exchange and renders the infant hypoxaemic. Many of the alveoli are distended and prone to barotrauma. Supplemental oxygen is provided, usually with continuous positive airways pressure (CPAP).

Helicopters produce more stress from vibration and noise than do fixed wing aircraft. Excess vibration may particularly disturb the sick neonate, producing hypoxaemia, apnoea, or bradycardia[18]. Temperature regulation should be maintained, as hypothermia and shivering increase oxygen consumption, and may aggravate metabolic acidosis and hypoglycaemia in sick infants. As humidity decreases with altitude, additional means to moisten the inspired air should be provided; for example by using nebulised saline. This will help temperature control, fluid balance, and reduce the tenacity of secretions.

Figure 5: Intubation of the right main bronchus. More severe forms of atelectasis develop when an endotracheal tube occludes one of the main stem bronchi. Significant hypoxaemia will result.

As infants are obligate nose breathers any obstruction to the nasal passages will cause respiratory distress. The developing trachea is prone to kinking and extension of the neck may lead to airway compromise and subsequent hypoxaemia. The child should be placed with the shoulders slightly supported. The trachea is also relatively short in the infant and it is easy for the operator to intubate the right main bronchus, with significant complications and hypoventilation3.

Management of the paediatric aeromedical retrieval

Background considerations

It is important that the aeromedical staff are aware of the physical, physiological and psychological stressors of flight. In addition to hypoxia, the aeromedical team should also consider other stressors, including the expansion of trapped gasses, noise, vibration and motion. The personnel planning an aeromedical transfer of the paediatric patient should have an holistic approach to the transfer. This approach should consider all aspects of the transfer, including clinical, flight and aeromedical staff. Secondary aeromedical transfer should only occur if it is likely to improve the patient's clinical outcome[19,20]. Further , the transfer should be undertaken in a manner that does not jeopardise the level and quality of care being given.[21,22].

There are published guidelines for the expected standard of care during transportation, with a typical guideline being that provided by the Australian and New Zealand College of Anaesthetists and Australasian College of Emergency Medicine[23].

Paediatric patient assessment & overcoming hypoxia

In paediatric patients who are receiving supplemental oxygen, the fractional inspired oxygen concentration may be increased to account for the hypoxia at altitude. This can be titrated during the journey by continuous pulse oximetry[24]. Another complementary technique is to lower cabin altitudes (maximum cabin pressure of 3700 feet) to ensure haemoglobin SO_2 levels of at least 80%. In infants who are ventilated, it would also be possible to increase the positive end expiratory pressure to help oxygenation. There are a variety of formulae available for predicting hypoxaemia at altitude and these are shown in Fact Box B[3,25,26].

An alternative method for pre-flight assessment involves hypoxic challenge testing. A convenient method to do this is by titration of the extra oxygen requirement of the infant or young child via whole body plethysmography in a body box, as described in Fact Box C[27].

Communication and documentation

Prior to departure, communication between the originating medical facility and the receiving facility is essential. The aeromedical staff should be very clear as to the nature of their tasking, and in addition, communication between the aeromedical staff is

Fact box B - Formulae for calculating hypoxaemia in adults at altitude

1. This relates Pao_2 at altitude (Alt) to Pao_2 at sea level (Ground)[25].

   PaO_2 *Alt (mmHg) = 0.410 x* PaO_2 *Ground (mmHg) = 17.652*

2. This relates Pao_2 Alt to Pao_2 Ground and includes FEV_1 in litres[25].

   PaO_2 *Alt (mmHg) = 0.519 x* PaO_2 *Ground (mmHg) + 11.855 x* FEV_1 *(litres) - 1.760*

3. This relates Pao_2 Alt to Pao_2 Ground and includes FEV_1 as % predicted[25].

   PaO_2 *Alt (mmHg) = 0.453 x* PaO_2 *Ground (mmHg) + 0.386 x (*FEV_1 *% pred) + 2.44*

4. This relates PaO_2 Alt to PaO_2 Ground and includes flight or destination altitude[3,26].

   PaO_2 *Alt (mmHg) = 22.8 - [2.74 x alt. (000's ft)] + 0.68 x* PaO_2 *Ground (mmHg)*

important for crew resource management reasons as it helps to minimise error. Documentation should always accompany the paediatric patient. The documents should be a complete summary of the care to date, and importantly there should also be a summary for rapid reference. Recorded observations should be copied and provided to the receiving medical facility.

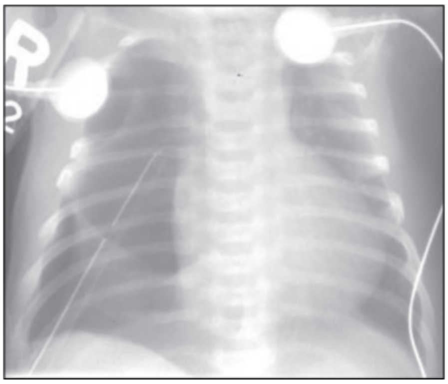

Figure 6: A right sided pneumothorax in an infant with pulmonary atresia. a chest tube has been inserted; however the lung is only partially inflated. The aeromedical staff must be alert for expansion of trapped gases and sudden deterioration in clinical status.

Selection of aeromedical staff and training

For the aeromedical transfer of paediatric patients, dedicated paediatric retrieval teams have been shown to be safer and more effective than standard aeromedical teams[28,29] The UK Paediatric Intensive Care Society recommends the use of dedicated paediatric retrieval teams[30].

Pre-transfer care

Ideally paediatric patients should be physiologically stable prior to transfer[31,32-34]. This requires careful pre-transfer assessment and optimisation of the

Fact box C - Titration of oxygen requirements at 8,000 ft altitude using whole body plethysmography at ground level

For oxygen dependent children, including premature infants with chronic lung disease where aeromedical transfer is considered essential, oxygen requirements can be titrated by plethysmography in a body box as follows[27].

Children are exposed to an hypoxic challenge with an FiO_2 of 15% while sitting on the lap of a carer in a whole body plethysmograph (body box). The infant, receiving oxygen via nasal cannulae, is placed in the body box in the company of a parent or carer, and SO_2 monitored via a probe attached to the child's finger. After measuring SO_2 of the child in air, nitrogen is passed into the body box at approximately 50 1/min* to dilute the oxygen content of the air to 15% over a period of 5 minutes. Oxygen and carbon dioxide concentrations are measured via continuous flow sampling. The SO_2 can take up to 20 minutes to reach a stable value. Any fail in SO_2 is restored to the original value by titration of the flow of oxygen through the nasal cannulae. This flow of oxygen should then be supplied during the flight.

*This high rate of flow ensures carbon dioxide does not build up beyond 0.5% within the body box.

Figure 8: A whole-body plethysmograph. For infants it is necessary that a parent or carer sits in the box nursing the infant. the healthy parent will tolerate SO2 of 15% with no difficulty. Photo Joe Mabel-the swedish Hospital Ballard Campus, seattle, Washington. http://en.wikepedia.org/wiki/File:Body_Plethysmography_chamber_01.jpg

child's status. Lack of anticipation of potential events during the transfer can adversely affect outcome[32,35]. Appropriate aeromedical equipment including monitoring devices, pumps, defibrillators, ventilators and humidicribs should be prepared and checked prior to departure. The aeromedical service should have a system in place so these activities are in a high state of preparedness at all times. Power sources should be examined closely and redundancies calculated for power and oxygen supplies. The parents of the child can be of enormous assistance to the team caring for the paediatric patient. It is important to be inclusive and considerate of their needs at all times. It is also worthwhile for your Desk Officer to ensure that Customs and Quarantine have been informed of the planned mission. Medical and nursing staff should have appropriate medical indemnity insurance for the location and type of activity they are underatking[31].

During transport

With good preparation and planning there should be little requirement for any active intervention during the flight itself[3]. The patient should be continually reassessed en-route, with the level of monitoring and the frequency of measurement of physiological parameters at least the same or greater than the originating medical facility. The paediatric patient should be monitored with continuous pulse oximetry and ECG. In addition, intermittent non-invasive blood pressure monitoring and temperature at an appropriate interval is indicated. For the ventilated patient a capnographic tracing is recommended. The equipment and therapeutic schedule should be extensive and appropriate for a paediatric patient. The schedule should be stowed in a logical and accessible manner and consumables kept in-date.

Post-transfer care

The paediatric aeromedical patient remains the responsibility of the transferring team until formal nursing and medical handover has occurred. Ideally this should be at the destination medical facility. The patient should then be reassessed using the ABC method. Monitors and ventilators are then changed to the receiving facility equipment. At this stage it is vital that all connections, lines and tubes are carefully re-evaluated. Documents are then handed over to the receiving medical facility - including blood results and radiographs. It is important to introduce the parents of the paediatric patient to the receiving staff. A post-mission debrief should occur amongst the aeromedical team.

Conclusion

Hypoxia presents one of the greatest challenges when performing aeromedical transfer of the paediatric patient. An appreciation of the anatomical and physiological differences that pre-dispose the infant to hypoxaemia should be requisite knowledge for aeromedical staff. Aeromedical transfer of paediatric patients should be performed whenever possible by dedicated paediatric transfer teams. These teams should have equipment and therapeutic schedules appropriate to the paediatric patient. The aeromedical transfer will be conducted with greater safety and efficiency for patient and staff if crew resource management practices are adopted. This includes, but is not limited to, planning and preparation, communication, briefings, error minimisation and mitigation and thorough equipment checks. Every attempt should be made to ensure the infant being transported is not exposed to any hypoxic interval – especially during those critical stages involving changes of oxygen and ventilators, and emplaning and deplaning.

Disclaimer

The views, opinions, and/or findings in this report are those of the author and should not be construed as an official policy of the Royal Australian Air Force or the Australian Defence Force.

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