Management of Respiratory Failure

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Respiratory failure is defined as the inability of the lungs to adequately deliver oxygen to the pulmonary circulation and/or remove carbon dioxide. The partial pressure of arterial oxygen (PaO_2) is therefore below the predicted normal range for the patient's age at the prevalent barometric pressure of the partial pressure of arterial carbon dioxide ($paCO_2$) is elevated above the normal range. It has been accepted that in respiratory failure the paO_2 is below 60 mm of Hg and $paCO_2$ is above 50 mm of Mg.[1]

In many instances respiratory failure is the consequence of underlying chronic pulmonary disease which gets exacerbated by acute pulmonary insults or reaches an end stage due to progressive disease. Another variety is where acute respiratory failure occurs in persons with previously normal lungs due to some precipitous insult. The onset in acute respiratory failure is relatively sudden.

Pathophysiology

The pathophysiological mechanisms in acute respiratory failure are related to (1) alveolar hypoventilation (2) ventilation perfusion mismatch (3) presence of shunt (4) diffusion limitation.

1. Alveolar hypoventilation : This results in abnormal increase in PCO_2 leading to respiratory acidosis. The arterial hypoxemia in this is due to a decrease in alveolar oxygen concentration (PAO_2).

When arterial hypoxemia is solely due to alveolar hypoventilation, the alveolo arterial difference [P (A-a) O_2] is normal. PAO_2 is calculated by alveolar gas equation i.e. $PAO_2 = PIO_2 - PaCO_2 \div R$ where R is respiratory exchange ratio and is normally 0.8 (CO_2 production $\div$ O_2 consumption). This arterial hypoxemia can be corrected by oxygen administration. However, oxygen supplementation does not correct respiratory acidosis so alveolar hypoventilation should be corrected by increasing the respiratory frequency.

2. Ventilation perfusion mismatch : Hypoxemia results when gas exchanging regions of the lungs are under ventilated with respect to their blood flow. In these regions the alveolar PAO_2 is low and alveolar $PACO_2$ is high. Hypoxemia due to ventilation perfusion mismatch rapidly responds to smaller increases in FiO_2. Appreciable improvement in arterial oxygen after a small increase in FiO_2 is useful in distinguishing V/Q mismatch and shunt as the primary mechanism of hypoxemia.

3. Shunt : The designation shunt refers to one extreme of ventilation perfusion mismatch. A shunt has no ventilation but perfusion continues whereas in V/Q mismatch some ventilation to the lung is still present. Both shunt and V/Q mismatch cause alveolar to arterial oxygen difference to increase. Small increases in FiO_2 have

relatively little effect on arterial oxygenation in presence of a shunt.

4. Severe diffusion limitation : is a relatively unimportant cause of acute respiratory failure. It is however an important factor in conditions associated with thickening of alveolo capillary membrane such as interstitial fibrosis, pulmonary alveolar proteinosis etc.

Type 1 respiratory failure

Type 1 respiratory failure represents poor oxygenation but relatively adequate ventilation. Patients in this group have a low paO_2 and a normal or low $paCO_2$. It is also termed normal ventilatory or non ventilatory respiratory failure. It is manifested by increase in p (A-a) DO_2 usually due to any pathological condition affecting alveolar space or distal airways. This results in ventilation/perfusion imbalance (VA/Q), shunting and failure of oxygenation with failure of ventilation. Since poor compliance with a low tidal volume may accompany this situation the body attempts to compensate for minute ventilation by increasing the respiratory rate.[2] Such patients have tachypnoea with low or normal $paCO_2$. Decompensated CO_2 retention leads to type 2 respiratory failure.

Type 2 respiratory failure

This represents a ventilatory defect with low paO_2 high $paCO_2$ and an imbalance in ventilation and perfusion (VA/Q). These patients need assisted ventilation. Patients can manifest with both types of respiratory failures during the course of the illness. Hypoxia or hypoxemia which is common denominator for both types of respiratory failures can potentially result in pulmonary hypertension and increased pulmonary vascular resistance. Chronic repiratory failure is characterised by chronically raised CO_2 levels.

Clinical pointers towards respiratory failure

Acute respiratory failure often ensues unpredictably and abruptly in critically ill children. Hence a high index of suspicion and knowledge of measures to prevent it is a must. When a child with a serious respiratory illness is not hyper ventilating it is a sign of imminent respiratory failure.

Following clinical features suggest presence of respiratory failure[3]

1. Presence of marked wheezing with progressive dyspnea and accessory muscle use manifested by suprasternal subcostal and intercostal retractions; 2. Decreased breath sounds; 3. Cyanosis inspite of oxygen administration; 4. Tachypnea, bradypnea, apnea and respiratory grunting; 5. Pulsus paradoxus > 15 mm Hg; 6. Marked restlessness, irritability and mental confusion; 7. In the presence of *neuromuscular disease, week or absent cough reflex, gag reflex, *incomplate *deglutition, pooled *oropharyngeal secretions and poor muscle tone entail monitoring for respiratory failure.

Laboratory diagnosis of respiratory failure in post neonatal period[4]

1. PaO_2 < 50 mm Hg breathii g room air at sea level or PaO_2 < 100 mm Hg with patient breathing 100% oxygen at sea level; 2. $PaCO_2$ > 50 mm Hg.

Management of respiratory failure

Management of respiratory failure starts with the ABC of resuscitation. (A-Airway, B = Breathing, C = Circulation). In acute cases after prompt cardiopulmonary assessment cardiopulmonary resuscitation (CPR) should be initiated in patients in cardiorespiratory arrest.

It is very important to allay the anxiety associated with hypoxemia by giving appropriate sedation to the affected child.

A. Airway patency

Maintenance of a patent airway is the first step in management of respiratory failure. This starts with proper positioning of the patients, avoiding neck flexion and splinting of the chest. Some times triple airway maneuvour is performed which includes head tilt/chin lift/jaw thrust. *Oropharyngeal secretions should be cleared by suction.

*Oropharyngeal airway may need to be inserted to prevent falling back of the tongue. This is a tubular curved device with one flanged end which protrudes from the mouth and the curved end reaches the angle of mandible.

Endotracheal Intubation is indicated (1) in upper airway obstruction with ensuing respiratory failure. (2) when excessive secretion in the airways. (3) for endotracheal clearance of aspirated material. (4) in respiratory failure requiring ventilatory support.

Endotracheal intubation can be orotracheal and nasotracheal.

Orotracheal intubation. After pre oxygen-ation with 100% oxygen orotracheal tube is passed preferably from the right corner of the mouth to avoid visual obstruction of the epiglottis. If needed, sedation is given with midazolam/diazepam/chloral hydrate. The tongue is depressed with the help of a blade of laryngoscope.

The tube position is checked by auscultation for bilateral equal air entry. Esophageal intubation is ruled out by abdominal auscultation after air insufflation into the tube. Objective assessment for this aspect can also be done with the help of hand held end tidal capnometers.

Nasotracheal intubation is performed in patients requiring artificial airway for longer periods of time. Usually it is done under general anaesthesia. It can also be performed with the help of a fibroptic bronchoscope.

Complications of endotracheal intubation can be prevented by avoiding the local injury, checking the tube position and by use of cuffed endotracheal tubes.

Tracheostomy is indicated in the presence of upper airway obstruction, vocal cord paralysis, facial burns or trauma, excessive secretions in the endotracheal tube or whenever prolonged ventilation is required. Technically it is more difficult in infants and younger children. Percutaneous tracheostomy decreases the risk of complications.[5]

B. Oxygen therapy

Hypoxemia is corrected by administration of humidified oxygen through mask, nasal prongs, nasopharyngeal catheters, hood or tent. Oxygen must be used as a drug. Higher concentration of oxygen especially

> 40% in inspired gas are potentially deleterious and should be avoided. Monitoring of a patient by pulse oxymetry to keep the hemoglobin saturation more than 90% is recommended. Saturation more than 97% may be associated with very high PaO_2 and should be avoided.[6,7]

Invasive blood gas monitoring gives the PaO_2 values which can be used to calculate arterial/alveolar oxygen (PaO_2—PAO_2) difference. Recently a patient dedicated on demand blood gas monitor has been tried which makes use of fluoroscent gas sensors known as optodes. Its performance is similar to conventional blood gas analyser.[8] This modality would avoid the need for frequent punctures and provide continuous monitoring of blood gases.

Home oxygen therapy : In patients of chronic respiratory failure, oxygen administration can be done at home with the use of oxygen concentrators. Administration of O_2 to maintain normal oxygen concentrations prevents the development of pulmonary hypertension. However careful monitoring should be done to preserve the hypoxic drive in these patients as chronic hypercarbia leads to chemo receptors becoming non responsive to changes in $paCO_2$.

C. Ventilation

Continuous positive airway pressure (CPAP) : Continuous positive airway pressure is used to improve oxygenation. This increases the functional residual capacity (FRC) and prevents the collapse of alveoli. It can be administered indigenously without the help of a ventilator.[9]

Assisted ventilation : Assisted ventilation is indicated whenever hypoxemia with paO_2 <60 mm Hg is not responding to oxygen administration (with FiO_2 more than 60%) or when $paCO_2$ is more than 50 mm Hg, in the presence of apnea, shallow breathing with tidal volume < 3.25 ml/kg.

Physiological basis of ventilation : The process of ventilation deals with movement of air inside and outside the lung and depends on the static properties of the lung like compliance of the lung and chest wall and dynamic properties determined by resistive properties of chest wall and airways.

The product of compliance and resistance determines the time constant. This is the measure of time required for a alveolar unit to reach equilibrium i.e. time to fill up and empty. One time constant fills 67% of an alveolus. There is 87%, and 95% filling by 2 and 3 time constants respectively. Three to five times constant are required for complete filling and emptying of alveolar unit.

The lung compliance varies with different phases of respiration. Time constant of alveoli increase in diseases where airway resistance increases as in asthma. Diseases associated with stiff lungs cause decreases in compliance and time constant is also decreased leading to faster emptying of alveoli.

Choice of a ventilator : Ventilators are pressure controlled and volume controlled. Most ventilators are time cycled. Pressure limited ventilator delivers air till a set pressure is released whereas volume controlled ventilator delivers a pre set tidal volume. Pressure controlled ventilators are useful in infants and volume limited in older children. Ventilators like Servo 900C

and Newport Breeze, provide both the modes. Pressure controlled ventilators are also useful in patients of ARDS or other conditions associated with decrease lung compliance.

Modes of ventilation

1. Controlled ventilation (CV) : The patient has very minimal efforts or is paralysed and ventilator controls all the ventilation.

2. Intermittent mandatory ventilation (IMV) : Ventilator delivers a preset number of controlled breaths which supplement spontaneous breathing of the patients. Sometimes patients can trigger the breaths as in synchronized IMV or SIMV.

3. Patient triggered ventilation (PTV) : The negative pressure generated by the patient triggers the ventilator. If the patient fails to trigger then the ventilator provides the breath.

4. Pressure support : This provides additional inspiratory flow through a demand valve and the patient is on continuous breathing.

5. High frequency ventilation (HFV) : This encompasses several modalities that have in common, the accomplishment of oxygenation and gas exchange with smaller tidal volumes and faster respiratory range. High frequency jet ventilation (HFJV) depends on the principle of entrapinment of gases with a high frequency velocity pulse of air or oxygen injected through a narrow canula in the endotracheal tube. Jet frequency vary from 150-900 breaths per minute. Another major mode of HFV is High Frequency Oscillatory Ventilation (HFOV). Devices that provide HFOV produce both positive and negative pressure wave forms with frequency ranges from 900-3600 cycles per minute. The tidal volume employed are 1-3 ml/kg. HFOV has met its greatest success in small infants.[10]

Initiation of ventilation

The setting of a tidal volume of 10-15cc/kg is a good starting point in initiating mechanical ventilation. The chest excursion is observed and auscultated to assess adequate inflation of lungs. Keeping in mind the compressible volume of a ventilator, a corrected tidal volume should be calculated.[11] As the tidal volume is increased there is an increase in the peak pressure generated that results in further increase in compressible volume loss. The ventilation frequency accompanied by an appropriate corrected tidal volume efficiently eliminates CO_2. Slower rates are used for obstructive airway disease to allow more time for exhalation and emptying of hyperinflated areas.[12]

The usual I : E ratio is 1 : 2. In severe lung disease, prolongation of inspiratory time allows better distribution of gas and enhances oxygen diffusion. Prolongation of IT creates more laminar flow and keeps the peak pressures low. The recommended level of PEEP depends on the type of disease process. PEEP levels should start at 3-5 cms of water and increased in increments of 1 or two cms of water. Higher values of PEEP are recommended for patients of acute respiratory distress syndrome previously termed as adult respiratory distress syndrome.[13,14] Patients on PEEP > 10 require pulmonary artery catheterization with Shwan ganz catheter and need cardiac output monitoring.

Weaning

Weaning is a step before stoppage of as-

sisted ventilation. This must be done very carefully and in a timely fashion. The patient should be in a stable pulmonary status without the evidence of any pulmonary complications like pneumonia or bronchospasm. The patient should be able to generate negative pressure and should have a stable cardiovascular status. The main principle of weaning is to decrease the peak inspiratory pressure. When the peak pressure is less than 20 cm of water ventilator rate should be reduced. Common methods used are shifting the patient to IMV, SIMV or CPAP followed by T tube entilation and extubation.

Prevention of complications

Airway pressure and high concentration of inspired oxygen leading to free radical for-mation can lead to barotrauma. Sometimes air leaks like pneumothorax may occur and attempt should be made to keep the mean airway pressure and oxygen concentration as low as possible. Airway pressure release valve also minimises peak inspiratory pressure. Surveillance for ventilator associated pneumonia should be carried out by endotracheal or bronchoscopic aspirate cultures.

D. Newer Modalities

ECMO (Extra Corporeal Membrane Oxygenation : ECMO is a chronic heart lung bypass and is used in situations associated with acute reversible lung injury. This expensive technique involves cannulating a large artery such as carotid artery and a large vein such as internal jugular vein.[15,16]

TABLE 1. Causes of Respiratory Failure in Children Beyond Neonatal Period

Acute respiratory failure	Chronic respiratory failure
Pulmonary	
Pheumonias	Bronchopulmonary dysplasia
Acute respiratory distress syndrome	Cystic fibrosis
Congenital malformations of the lung	Interstitial lung disease
Foreign body aspirations	Pulmonary fibrosis Graft vs host disease
Airway problems	
Upper airway obstruction	Bronchiolitis oblitrans
Bacterial tracheitis	Bronchiolitis oblitrans with organizing pneumonia
Bronchial asthma	
Neuromuscular disorders	
Acute Inflammatory Demyelinating Polyneuropathy (Landre Guillaine Barre Syndrome)	Myopathies
Poliomyelitis	
Snake bites	

The infant blood is pumped through a membrane oxygenator where ventilation occurs. The warmed oxygenated blood is returned to the body. The lungs can then be spared the continued pressure and oxygen toxicity and are usually maintained on low rate low pressure intermittent mandatory ventilation to prevent total lung atelectasis. Steinhorn et al reviewed 72 patients with RSV bronchiolitis treated with ECMO at 9 insitutions. Of the 58 who survived some required upto 3 weeks of ECMO support.[17]

Liquid ventilation has been tried in infants where liquid oxygen dissolved in fluorocarbon solution can be used to ventilate the lungs.[18] This mode of treatment is mainly experimental and has met with limited clinical success.

Inhaled nitric oxide

Nitric oxide has been hailed as the molecule of the nineties and has been used in conjunction with ventilation in respiratory failure associated with pulmonary hypertension.[19] It has also been tried with beneficial results in acute respiratory distress syndrome (ARDS).[20]

Lung transplantation

Lung and heart lung transplantation is now an accepted treatment option in children with end stage pulmonary and cardio-pulmonary disease.[21] Indications for pediatric lung transplantation have been expanding.[22]

Pediatric lung transplantation is performed in end stage lung disease due to cystic fibrosis, primary pulmonary hypertension, emphysema, graft vs host disease, rheumatoid lung and desquamative inter-

stitial pneumonitis. The over all survival rate has been reported to be more than 76%.

Hence it is possible to prolong the life of patients of respiratory failure with a reasonably good quality of life. However availability of these therapies in our country and their cost benefit ratio is a subject of serious consideration.

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