# Respiratory support in children

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## Abstract

Respiratory failure is defined by the inability of the respiratory system to adequately deliver oxygen or remove carbon dioxide from the pulmonary circulation resulting in hypoxemia, hypercapnia or both. A wide variety of disease processes can lead to respiratory failure in children. Multiple interventions can support the pediatric patient with respiratory failure, from simple oxygen delivery devices to high frequency oscillatory ventilation and Extracorporeal Membrane Oxygenation. This article will review available devices to improve oxygenation and ventilation, their advantages and disadvantages, and help guide physicians in the management of children with respiratory failure.

**Keywords** anoxia; artificial; hypercapnia; paediatrics; respiration; respiratory insufficiency

## Respiratory support in children

Respiratory illness accounts for approximately 1 in 5 hospital admissions and respiratory failure is the leading cause of cardiac arrest in children. Specifically, respiratory failure is the inability of the respiratory system to adequately oxygenate or remove carbon dioxide from the pulmonary circulation, resulting in hypoxemia, hypercapnia or both. Any abnormality of the respiratory system can lead to respiratory failure (Table 1). Due to several anatomical and physiological considerations, in any given medical situation infants and young children are at greater risk of respiratory failure than older children or adults.

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### What's new

Over the last few years, the mainstays of respiratory support in children have remained the same. Characterizing the type of respiratory failure is crucial to choose the appropriate respiratory support. New objective tools are emerging to help the bedside clinician with assessment of the child with respiratory failure. Non-invasive modalities of respiratory support are also gaining popularity. The response to the chosen method of support and underlying pathophysiology of the disease should guide decision making in a child with respiratory failure.

## Overview of respiratory physiology

### Gas exchange

The content of oxygen in the blood leaving the lungs depends on several aspects of lung function; the partial pressure of oxygen in the alveoli, diffusion of oxygen across the alveolar wall, and the degree of pulmonary shunt. Pulmonary shunt is the blood flow through the lungs that does not encounter areas of ventilation and therefore does not participate in gas exchange.

The alveolar gas equation describes the partial pressure of oxygen present in individual alveoli.

$$P_AO_2 = FiO_2(P_B - P_{H20}) - P_ACO_2/RQ$$

where $P_AO_2$ is the partial pressure of oxygen in the alveolus, $FiO_2$ is the fractional concentration of inspired oxygen, $P_B$ is the barometric pressure, $P_{H20}$ is the partial pressure of water vapor, $P_ACO_2$ is the partial pressure of carbon dioxide in the alveolus (assumed to equal the partial pressure of arterial $CO_2$, the $PaCO_2$) and RQ is the respiratory quotient (represents the ratio of oxygen consumption to carbon dioxide production and is usually approximated at 0.8).

The difference between the $P_AO_2$ and the arterial partial pressure of oxygen ($P_aO_2$) is minimal in healthy lungs (10−15 mmHg). In diseased lungs, this Alveolar−Arterial (A−a) $PO_2$ gradient represents the severity of pulmonary shunt and ventilation-perfusion mismatch or rarely, a diffusion abnormality. Although an elevated $PaCO_2$ from hypoventilation can lead to hypoxemia as demonstrated by the alveolar gas equation, a modest increase in $FiO_2$ will easily increase the $PaO_2$. On the other hand, improving the hypoxemia related to pulmonary shunt or severe ventilation-perfusion mismatch is generally accomplished only with interventions that lead to resolution of the shunt.

Carbon dioxide removal from the pulmonary circulation is somewhat dependent on the minute ventilation (Minute ventilation = Tidal Volume × Respiratory Rate). However, this tidal volume includes alveolar volume as well as physiologic dead space volume, (i.e. volume that is distributed to areas of the respiratory system that are ventilated but do not receive perfusion and therefore do not participate in gas exchange). The physiologic dead space volume is composed of both airway dead space, the mouth and conducting airways, and alveolar dead space, (alveoli that are ventilated but not perfused with blood). Normally, physiologic dead space volume is approximately 30% of each breath, with alveolar dead space being close to zero.

**Common causes of respiratory failure in children**

| Site of respiratory failure | Examples of disease processes |
|---|---|
| Upper airway disorders | Anaphylaxis |
| | Foreign body |
| | Infection (Croup, Epiglottitis, Bacterial Tracheitis) |
| | Laryngotracheomalacia |
| Obstructive lower airways disease | Asthma |
| | Bronchiolitis |
| | Cystic Fibrosis |
| Restrictive lung disease | Abdominal compartment syndrome |
| | Acute respiratory distress syndrome |
| | Chronic lung disease |
| | Pleural effusion |
| | Pneumonia |
| | Pulmonary edema |
| Central nervous system disorders | Intracranial Injury (Hemorrhage, Hypoxic Ischemic Injury) |
| | Metabolic encephalopathy |
| | Pharmacologic agent (Central Nervous System Depressant) |
| Disorders of the muscles of respiration and peripheral nervous system | Guillain Barré syndrome |
| | Infant botulism |
| | Muscular dystrophy |
| | Myasthenia gravis |
| | Scoliosis |
| | Spinal cord injury |

**Table 1**

However, in children with significant lung disease physiologic dead space can approach 60–70% of each breath. Because the physiologic dead space volume does not participate in gas exchange, it does not aid in carbon dioxide removal. Therefore, the alveolar minute ventilation determines carbon dioxide removal.

$$\text{Alveolar Minute Ventilation} = (\text{Tidal Volume} - \text{Physiologic Dead Space Volume}) \times \text{Respiratory Rate}$$

It is important to note that in children, particularly infants, airway dead space is proportionally larger than in adults due to differences in the anatomy of the oropharynx. Methods to decrease airway dead space, such as washout with high flow rates of air (for example with high flow humidified nasal cannula), can preserve alveolar minute ventilation whilst decreasing the minute ventilation and therefore reduce the effort of breathing required for appropriate gas exchange. Carbon dioxide diffuses rapidly; consequently abnormalities in alveolar diffusion do not generally affect ventilation.

### Respiratory mechanics

Inspiration is an active process and exhalation in normal lungs is passive. Given their elastic properties, the lungs and the chest wall have a tendency to move in opposite directions, the lungs collapse and the chest wall expands outward. The balance point of these two forces occurs when the lung volume is at functional residual capacity (FRC). At FRC, the compliance of the respiratory system is the greatest.

$$\text{Compliance}(\text{of the respiratory system}) = \Delta\text{Volume}/\Delta\text{Pressure}$$

Restrictive lung disease, such as pneumonia or a pleural effusion, is characterized by decreased respiratory system compliance with an end-expiratory lung volume that is below normal FRC. These patients develop atelectasis and subsequent hypoxemia predominantly due to intrapulmonary shunt. While minimal atelectasis and shunt can be overcome with supplemental oxygen, patients with significant restrictive disease often require additional support with positive pressure to re-expand areas of lung collapse and consolidation.

In obstructive airways diseases, such as bronchiolitis or asthma, patients develop air trapping with an end-expiratory lung volume above normal FRC. They have mismatching between areas of ventilation and perfusion in the lung and are prone to the development of regional atelectasis and over distension. These patients may require assistance with ventilation secondary to muscle fatigue or less frequently due to hypoxemia related to shunt.

## Respiratory support devices

The management of respiratory failure is largely based on symptomatic support until the underlying disease process abates. Therapies should be applied in a manner that addresses the pathophysiology behind the respiratory failure (Table 2).