

# Paediatric applied respiratory physiology — the essentials

Anoopindar Bhalla  
Robinder G Khemani  
Christopher JL Newth

## Abstract

General paediatricians will frequently encounter children with a variety of respiratory abnormalities. A basic knowledge of respiratory physiology, pathophysiologic derangements during common paediatric disease states and appropriate assessment of abnormalities is essential to properly care for children with respiratory disease. This review will give an overview of normal respiratory physiology and how to assess the efficiency of gas exchange. It will also discuss common methods of respiratory monitoring including pulse oximetry, carbon dioxide monitoring, pulmonary function tests and respiratory effort and capacity tests. Finally, paediatric diseases will be used to illuminate the intersection between pathophysiology, clinical symptoms and monitoring capabilities.

**Keywords** lung volume measurements; oximetry; paediatrics; plethysmography; pulmonary ventilation; respiratory function tests

## Introduction

The diversity and prevalence of respiratory illnesses in children requires paediatricians to understand basic respiratory physiology and respiratory function monitoring. This discussion will review normal respiratory physiology and explore forms of respiratory monitoring. With this foundation, the paediatrician can accurately diagnose and assess the severity of respiratory illness.

## Brief overview of normal respiratory physiology

### Muscles of respiration

The most important and powerful muscle during the inspiratory phase of respiration is the diaphragm, a dome-shaped musculo-fibrous septum that separates the thorax from the abdominal cavity. When the diaphragm contracts, abdominal contents move

**Anoopindar Bhalla MD** is an Assistant Professor of Pediatrics, Department of Anesthesiology and Critical Care Medicine, University of Southern California Keck School of Medicine, Children's Hospital Los Angeles, Los Angeles, USA. Conflict of interest statement: none.

**Robinder G Khemani MD MScI** is an Associate Professor of Pediatrics, Department of Anesthesiology and Critical Care Medicine, University of Southern California Keck School of Medicine, Children's Hospital Los Angeles, Los Angeles, USA. Conflict of interest statement: none.

**Christopher JL Newth MD FRCPC FRACP** is a Professor of Pediatrics, Department of Anesthesiology and Critical Care Medicine, University of Southern California Keck School of Medicine, Children's Hospital Los Angeles, Los Angeles, USA. Conflict of interest statement: none.

## What's new?

The essentials of paediatric respiratory physiology have changed relatively little over the last few years. New methods for the assessment of respiratory function continue to be developed, many of which are beginning to extend beyond the research realm and are becoming more available to the bedside clinician. A thorough understanding of normal paediatric respiratory physiology and how to use these tools to diagnose and manage respiratory abnormalities remains crucial for any general paediatrician.

downward and the lung expands in the vertical and horizontal planes. During normal tidal breathing the diaphragm moves approximately 1 cm, but with forced inspiration and exhalation, it can move up to 10 cm.

During inspiration, external intercostal muscles elevate and move the ribs forward. This increases the lateral and anteroposterior diameters of the thoracic cavity. The two most common accessory muscles of inspiration are the sternocleidomastoid and scalenes. The sternocleidomastoid raises the sternum while scalenes elevate the first two ribs. During normal respiration these muscles do not participate in inspiration, but during exercise or in pathological processes they can play an important role in maintaining normal alveolar ventilation.

While exhalation is normally passive due to the elastic properties of the lungs and chest wall, both exercise and certain pathophysiological conditions invoke both the internal intercostal and abdominal muscles including the internal and external obliques, the transversus abdominis and the rectus abdominis. These muscles work to decrease the thoracic volume and assist in forcing air from the lungs.

Both the lungs and chest wall are elastic, and each component has a natural propensity; the lung to collapse inward and the chest wall to 'spring' outward. The equilibrium point of lung volume where these forces are balanced is the functional residual capacity (FRC).

## Lung volumes and capacities

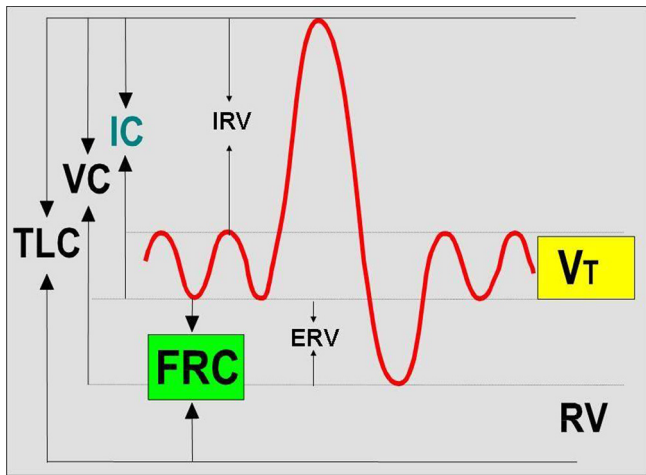
The various lung volumes and capacities can be measured during different phases of the respiratory cycle and change under different pathophysiological conditions. Spirometry is used to record the volume of air moved during respiration.

There are four lung volumes which, when added together, equal the total lung capacity (TLC) (Figure 1):

- tidal volume ( $V_T$ ) — volume of air inspired or expired during a normal breath
- inspiratory reserve volume (IRV) — the additional volume of air that can be inspired in addition to a tidal breath
- expiratory reserve volume (ERV) — the additional volume of air that can be exhaled after the end of a tidal breath
- residual volume (RV) — volume of air remaining after the most forceful exhalation.

In evaluating a patient's clinical status and understanding pathophysiology, it is sometimes advantageous to consider two or more lung volumes together as capacities. The four lung capacities (Figure 1) are:

- inspiratory capacity (IC) — equals tidal volume plus inspiratory reserve volume



**Figure 1** The various lung volumes and capacities obtained by spirometry. IC, inspiratory capacity; VC, vital capacity;  $V_T$ , tidal volume; TLC, total lung capacity; RV, residual volume; IRV, inspiratory reserve volume; ERV, expiratory reserve volume; FRC, functional residual capacity.

- functional residual capacity (FRC) – equals expiratory reserve volume plus residual volume, or the volume of air remaining at the end of a normal expiratory breath
- vital capacity (VC) – equals inspiratory reserve volume plus tidal volume plus expiratory reserve volume
- total lung capacity (TLC) – equals vital capacity plus residual volume.

Since by definition residual volume cannot be exhaled from the lungs, it cannot be measured by spirometry. FRC and TLC also cannot be measured by spirometry alone but instead are measured by gas-dilution techniques. Abnormalities in FRC characterize obstructive (increased FRC) airways disease and restrictive (decreased FRC) lung disease.

### Gas exchange

The primary purpose of the respiratory system is gas exchange to maintain cellular homeostasis. The two principal components are delivery of oxygen and removal of carbon dioxide.

**Oxygenation:** the respiratory system increases the oxygen content of blood flowing through the lungs, a necessary step for the goal of oxygen delivery to mitochondria. The partial pressure of oxygen in the alveolus ( $P_{A}O_2$ ) is a primary determinant of arterial oxygen tension ( $P_{a}O_2$ ).

$$P_{A}O_2 = [(P_b - P_{H_2O}) * F_{i}O_2] - P_{A}CO_2/RQ$$

where  $P_b$  is barometric pressure,  $P_{H_2O}$  is the partial pressure of water vapour and  $F_{i}O_2$  is the fraction of inspired oxygen.  $P_{A}CO_2$  is the partial pressure of carbon dioxide in the alveolus and RQ is the respiratory quotient. For most purposes RQ is assumed to be 0.8.

Substituting normal values for an individual breathing room air at sea level, the  $P_{A}O_2$  is approximately 100 mmHg. As oxygen crosses the alveolar membrane into the pulmonary capillary network a negligible amount of oxygen tension is lost (around 10 mmHg). Thus, the  $P_{a}O_2$  of a normal individual is approximately 90 mmHg. By examining the alveolar gas equation closely, one can see that three conditions can cause a decrease in  $P_{A}O_2$ :

altitude (low  $P_b$ ), hypoxic gas mixture (low  $F_{i}O_2$ ) and hypoventilation (high  $P_{A}CO_2$ ).

Decreased  $P_{A}O_2$  is generally a rare cause of hypoxaemia in a patient. More commonly, hypoxaemia is a result of a disturbance in the matching of ventilation and blood perfusion in alveoli ( $V'/Q'$  mismatch), which manifests with a large difference between the  $P_{A}O_2$  and the  $P_{a}O_2$  (an elevated Alveolar-arterial gradient). Patients with severe hypoxaemia typically have intrapulmonary shunt, one end of the spectrum of  $V'/Q'$  mismatch (Figure 2). In intrapulmonary shunt, some lung areas receive blood perfusion without ventilation. This results in deoxygenated blood passing through the pulmonary system without any increase in oxygen content, which can lead to significant arterial hypoxaemia. A wide variety of clinical conditions can cause intrapulmonary shunt. When intrapulmonary shunt is related to alveolar collapse, interventions such as continuous positive airway pressure (CPAP), bi-level positive airway pressure (BiPAP) or endotracheal intubation with mechanical ventilation can improve hypoxaemia by restoring normal lung volumes and improving the  $V'/Q'$  relationship.

**Ventilation:** the respiratory system also eliminates carbon dioxide from the blood through the alveolus. Arterial carbon dioxide tension ( $P_{a}CO_2$ ) is usually directly proportional to minute ventilation ( $V_E$ ) where:

$$V_E = \text{respiratory rate} \times \text{tidal volume (or } V_E = fV_T)$$

Tidal volume ( $V_T$ ) includes both physiologic dead-space volume ( $V_D$ ) and alveolar volume. Physiologic dead-space volume represents ventilation that does not participate in gas exchange and is comprised of airway dead space and alveolar dead space. Airway dead space is found within the conducting airways – nose, mouth, oropharynx, trachea, bronchi and bronchioles – and normally accounts for approximately 20–30% of a tidal breath or approximately 1 ml/kg, although it is relatively larger in infants approximately 50% of a tidal breath (3 ml/kg). Alveolar dead space represents the opposite side of the spectrum of  $V'/Q'$  mismatch from intrapulmonary shunt, where alveoli receive ventilation without blood perfusion (Figure 2). Alveolar dead space approaches zero in healthy individuals, as most lung units are equivalently ventilated and perfused. In infants and children with respiratory disease, however, total dead space may approach 60–70% of a tidal breath. The hallmark for patients with increased dead space is an elevation in  $P_{a}CO_2$ , or an abnormally high minute ventilation to maintain a normal  $P_{a}CO_2$ .

In contrast, alveolar volume is the portion of inspired breath that arrives at the alveoli and participates in gas exchange with pulmonary capillaries. Therefore, it is more accurate to state that  $P_{a}CO_2$  is proportional to alveolar minute ventilation ( $MV_A$ ):

$$MV_A = f(V_T - V_D)$$

Hence, an elevated  $P_{a}CO_2$  can arise from a decrease in respiratory rate, a decrease in tidal volume, or an increase in dead space when metabolic conditions are stable.

### Infant respiratory physiology

Infants are at particular risk for respiratory failure due to several physiologic disadvantages. The chest wall compliance of an

Download English Version:

<https://daneshyari.com/en/article/5720002>

Download Persian Version:

<https://daneshyari.com/article/5720002>

[Daneshyari.com](https://daneshyari.com)