Paediatric applied respiratory physiology — the essentials

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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 paper 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 inductance plethysmography. 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

What's new?

- The essentials of pediatric respiratory physiology have changed relatively little over the last few years
- New methods for the assessment of respiratory function are beginning to extend beyond the research realm and are becoming available to the bedside clinician
- Understanding normal paediatric respiratory physiology and how to diagnose and manage respiratory abnormalities remains crucial for any general paediatrician.

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 musculofibrous septum that separates the thorax from the abdominal cavity. When the diaphragm contracts, abdominal contents move 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

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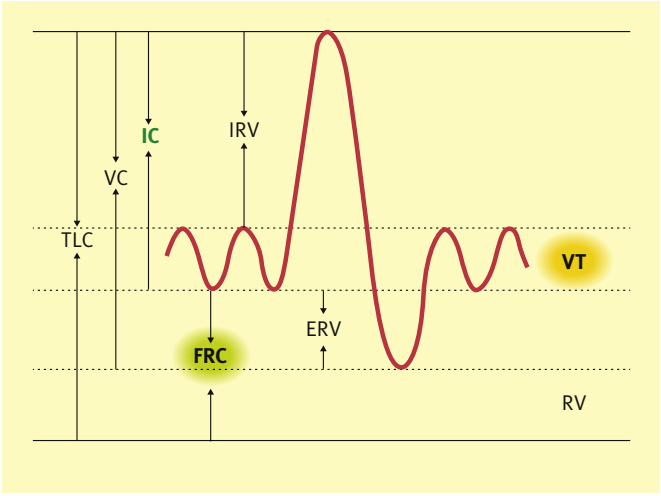

Figure 1 The various lung volumes and capacities obtained by spirometry. IC, inspiratory capacity; VC, vital capacity; V_T, tidal volume; TLC, total lung capacity; RV, residual volume; IRV, inspiratory reserve volume; ERV, expiratory reserve volume; FRC, functional residual capacity.

or more lung volumes together as capacities. The four lung capacities (Figure 1) are

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

Note that 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 or plethysmography. 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 helping supply oxygen to mitochondria. The partial pressure of oxygen in the alveolus (P_AO_2) is a primary determinant of arterial oxygen tension (P_aO_2).

$$P_AO_2 = [(P_b - P_{H_2O}) * FiO_2] - P_ACO_2/RQ$$

where P_b is barometric pressure, P_{H_2O} is the partial pressure of water vapour and FiO_2 is the fraction of inspired oxygen. P_ACO_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_AO_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_aO_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_AO_2:altitude (low P_b), hypoxic gas mixture (low FiO_2) and hypoventilation (high P_ACO_2).

Decreased P_AO_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_AO_2 and the P_aO_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 any 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, and 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_aCO_2) is directly proportional to minute ventilation (VE) where:

$$VE = \text{respiratory rate} \times \text{tidal volume}$$

$$VE = fV_T$$

Tidal volume (V_T) includes both dead-space volume (V_D) and alveolar volume. 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 accounts for approximately 20–30% of a tidal breath or approximately 1 ml/kg, although it is relatively larger in infants (approximately 3 ml/kg). Alveolar

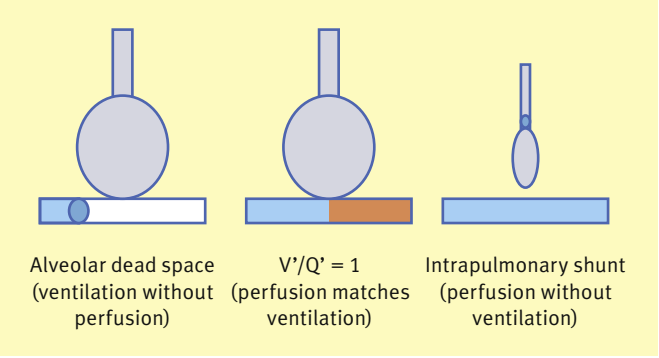

Figure 2 The continuum of V'/Q' matching from alveolar dead space to intrapulmonary shunt.

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