Spontaneous Breathing

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Vinod K. Bhutani

I. Introduction
   A. Air, like liquid, moves from a region of higher pressure to one with lower pressure.
   B. During breathing and just prior to inspiration, no gas flows because the gas pressure within the alveoli is equal to atmospheric pressure.
   C. For inspiration to occur, alveolar pressure must be less than atmospheric pressure.
   D. For expiration to occur, alveolar pressure must be higher than atmospheric pressure.
   E. Thus, for inspiration to occur, the gradient in pressures can be achieved either, by lowering the alveolar pressure ("negative," "natural," spontaneous breathing) or, raising the atmospheric pressure ("positive," "pressure," mechanical breathing).
   F. The clinical and physiologic implications of forces that influence inspiration and expiration are discussed in this section.

II. Signals of Respiration
   A. Each respiratory cycle can be described by the measurement of three signals: driving pressure (P), volume (V), and time (Fig. 4.1).
   B. The rate of change in volume over time defines flow ($\dot{V}$).
   C. The fundamental act of spontaneous breathing results from the generation of P, the inspiratory driving force needed to overcome the elastic, flow-resistive, and inertial properties of the entire respiratory system in order to initiate V.
      1. This relationship has been best described by Röhrer using an equation of motion in which the driving pressure (P) is equal to the sum of elastic (P_E), resistive (P_R) and inertial pressure (P_I) components, thus:

$$P = P_E + P_R + P_I$$

      2. In this relationship, the elastic pressure is assumed to be proportional to volume change by an elastic constant (E) representing the elastance (or elastic resistance) of the system.
      3. The resistive component of pressure is assumed proportional to airflow by a resistive constant (R) representing inelastic airway and tissue resistances.

V.K. Bhutani, M.D. (✉)
Department of Pediatrics, Lucille Packard Children's Hospital, Stanford University, Palo Alto, CA, USA
e-mail: Bhutani@stanford.edu

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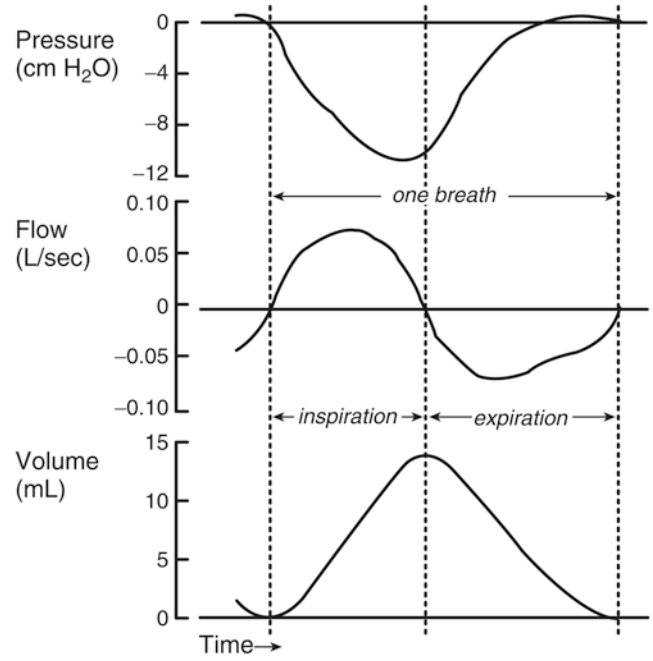

Fig. 4.1 Graphic representation of a respiratory cycle demonstrating pressure, flow, and volume waveforms. Volume is obtained by integration (area under the curve) of the flow signal (Modified from Bhutani VK, Sivieri EM, Abbasi S: Evaluation of pulmonary function in the neonate. In Polin RA, Fox WW [Eds.]: *Fetal and Neonatal Physiology*, second edition, Philadelphia, W.B. Saunders, 1998, p. 1144, with permission.)

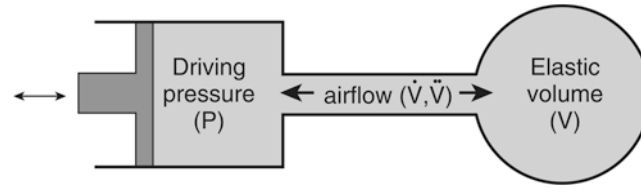

Fig. 4.2 Linear, first order model of the respiratory system, where applied pressure causes gas to flow through a tube

4. In addition, the inertial component of pressure is assumed to be proportional to gas and tissue acceleration ($\ddot{V}$) by an inertial constant (I). Therefore:

$$P = EV + R\dot{V} + I\ddot{V}$$

5. This is a linear, first order model in which the respiratory system is treated as a simple mechanical system (Fig. 4.2), where applied pressure P causes gas to flow through a tube (the respiratory airways) which is connected to a closed elastic chamber (alveoli) of volume V. In this ideal model E, R, and I are assumed to be constants in a linear relationship between driving pressure and volume.
6. Under conditions of normal breathing frequencies (relatively low airflow and tissue acceleration) the inertance term is traditionally considered negligible, therefore:

$$P = EV + R\dot{V}$$

7. In respiratory terminology, elastance is usually replaced by compliance (C), which is a term used to represent the expandability or distensibility of the system. Since compliance is simply the reciprocal of elastance, the equation of motion can be rewritten as:

$$P = V / C + R\dot{V}$$

8. This simplified form of the Röhrer equation is the basis for most evaluations of pulmonary mechanics where measurements of P, V, and $\dot{V}$ are used to compute the various components of respiratory system compliance, resistance, and work of breathing.

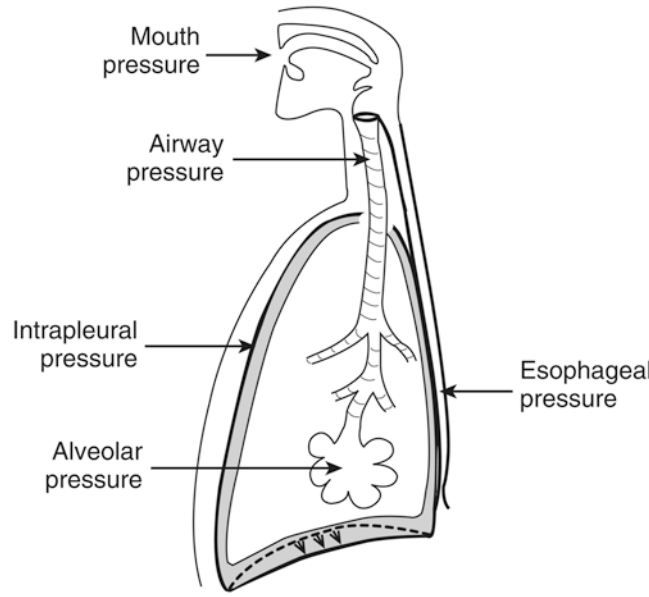

Fig. 4.3 Schematic representation of components of respiratory pressures used in pulmonary function studies. Esophageal pressure approximates intrapleural pressure (Modified from Bhutani VK, Sivieri EM, Abbasi S: Evaluation of pulmonary function in the neonate. In Polin RA, Fox WW [Eds.]: *Fetal and Neonatal Physiology*, second edition, Philadelphia, W.B. Saunders, 1998, p. 1153, with permission.)

D. One can further study the nonlinear nature of the respiratory system using more advanced nonlinear models and by analyzing two-dimensional graphic plots of P–V, V–$\dot{V}$ and P–$\dot{V}$ relationships.

E. Because the inherent nature of the respiratory signals is to be variable (especially in premature infants), it is imperative that the signals are measured in as steady state as feasible and over a protracted period of time (usually 2–3 min).

III. Driving Pressure

A. During spontaneous breathing the driving pressure required to overcome elastic, airflow-resistive, and inertial properties of the respiratory system is the result of intrapleural pressure (P_{IP}) changes generated by the respiratory muscles (Fig. 4.3).

B. During a respiratory cycle both the intrapleural and alveolar pressures change.

1. Just before the commencement of an inspiratory cycle, the intrapleural pressure is subatmospheric (−3 to −6 cm H_2O) because of the elastic recoil effect of the lung.
2. At this time, the alveolar pressure is atmospheric (zero), because there is no airflow and thus no pressure drop along the conducting airways. At this time, the alveolar pressure is atmospheric zero, because there is no airflow and thus no pressure drop along the conducting airways.
3. During a spontaneous inspiration, forces generated by the respiratory muscles cause the intrapleural pressure to further decrease producing a concomitant fall in alveolar pressure so as to initiate a driving pressure gradient which forces airflow into the lung.
4. During a passive expiration, the respiratory muscles are relaxed and the intrapleural pressure becomes less negative.
5. Elastic recoil forces in the now expanded lung and thorax cause alveolar pressure to become positive and thus the net driving pressure forces air to flow out of the lungs.
6. With forced expiration, the intrapleural pressure rises above atmospheric pressure.
7. The magnitude of the change in the alveolar pressure depends on the airflow rate and the airway resistance but usually varies between 1 and 2 cm H_2O below and above atmospheric pressure during inspiration and expiration, respectively.
8. This range of alveolar pressure change can be markedly increased with air trapping or airway obstruction.

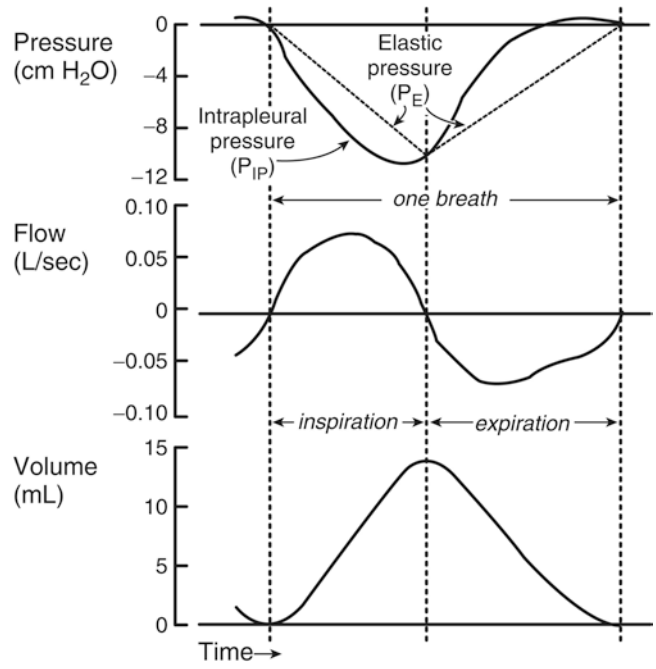

Fig. 4.4 During tidal breathing, airflow is zero at end-inspiration and end-expiration, where it reverses direction. The pressure difference between these two points represents the net elastic pressure at end-inspiration. The elastic component of intrapleural pressure at other points can be approximated by a straight line connecting points of zero flow

C. Following are some physiologic observations of changes in intrapleural pressure during spontaneous breathing
   1. Under some conditions respiratory airflow is zero or very close to zero:
      a. During tidal breathing, airflow is zero at end inspiration and end expiration where it reverses direction (Fig. 4.4).
      b. During slow static inflation, airflow can be approximated as zero.
      c. In both cases the resistive component of driving pressure as described above is zero or $RV=0$ and P_{IP} is equal to elastic pressure only:

$$P_{IP} = P_E = V / C$$

   2. The elastic component of intrapleural pressure can be estimated on the pressure tracing by connecting with straight lines the points of zero flow at end-expiration and end-inspiration. The vertical segment between this estimated elastic pressure line and the measured intrapleural pressure (solid line) represents the resistive pressure component (Fig. 4.5).
   3. Resistive pressure is usually maximum at points of peak airflow, which usually occurs during mid inspiration and mid expiration.
   4. Transpulmonary pressure (P_{TP}) is the differential between intrapleural pressure and alveolar pressure. This is the portion of the total respiratory driving pressure which is attributed to inflation and deflation of the lung specifically.

D. With mechanical ventilation, of course, the driving pressure is provided by the ventilator. In contrast to spontaneous breathing, where a negative change in intrapleural pressure is the driving pressure for inspiration, the mechanical ventilator applies a positive pressure to an endotracheal tube. Nonetheless, in both cases there is a positive pressure gradient from the mouth to the alveoli. In both cases the transpulmonary pressure gradient is in the same direction.

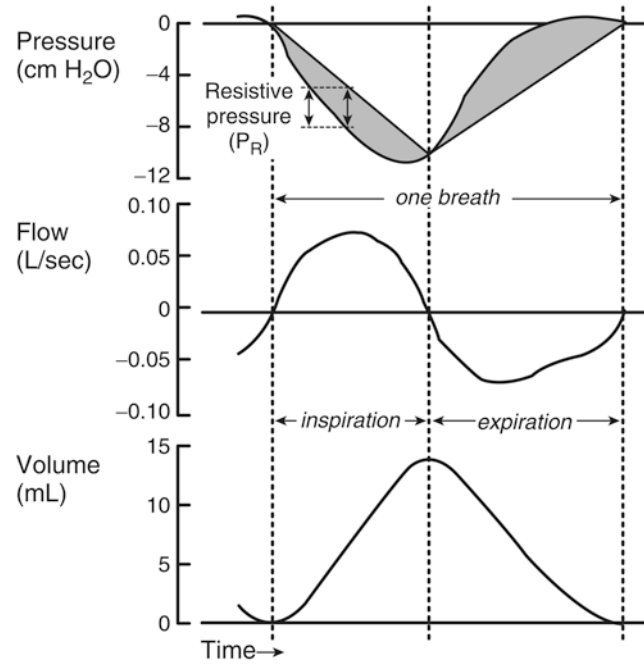

Fig. 4.5 The elastic component of intrapleural pressure can be estimated on the pressure tracing by connecting points of zero flow at end-expiration and end-inspiration with a straight line. The vertical distance between this estimate and the measured intrapleural pressure is the resistive pressure component (*solid line*)

IV. Factors that Impact Mechanics of Airflow

Factors that influence the respiratory muscles and respiratory mechanics have an effect on how air flows in and out of the lungs. These are characterized by physical, physiologic, and pathophysiologic considerations.

A. Physical Factors

1. The pattern of airflow is affected by the physical properties of the gas molecules, the laminar or turbulent nature of airflow, and the dimensions of the airways, as well as the other effects described by the Poiseuille equation (Chap. 8).
2. The elastic properties of the airway, the transmural pressure on the airway wall, and structural features of the airway wall also determine the mechanics of airflow.
3. In preterm newborns, the airways are narrower in diameter and result in a higher resistance to airflow. The increased airway compliance increases the propensity for airway collapse or distension. If a higher transmural pressure is generated during tidal breathing (as in infants with bronchopulmonary dysplasia, or, during positive pressure ventilation), the intrathoracic airways are likely to be compressed during expiration (Fig. 4.6).
4. During forced expiration, the more compliant airways are also likely to be compressed in the presence of a high intrathoracic pressure.
5. Increased distensibility of airways, as when exposed to excessive end-distending pressure, can result in increased and wasted dead space ventilation.
6. Turbulence of gas flow, generally not an issue in a healthy individual, can lead to a need for a higher driving pressure in the sick preterm infant with structural airway deformations as encountered in those with BPD.

B. Physiologic

1. The tone of the tracheobronchial smooth muscle provides a mechanism to stabilize the airways and prevent collapse.
2. An increased tone as a result of smooth muscle hyperplasia or a hyper-responsive smooth muscle should lead to a bronchospastic basis of airflow limitation.

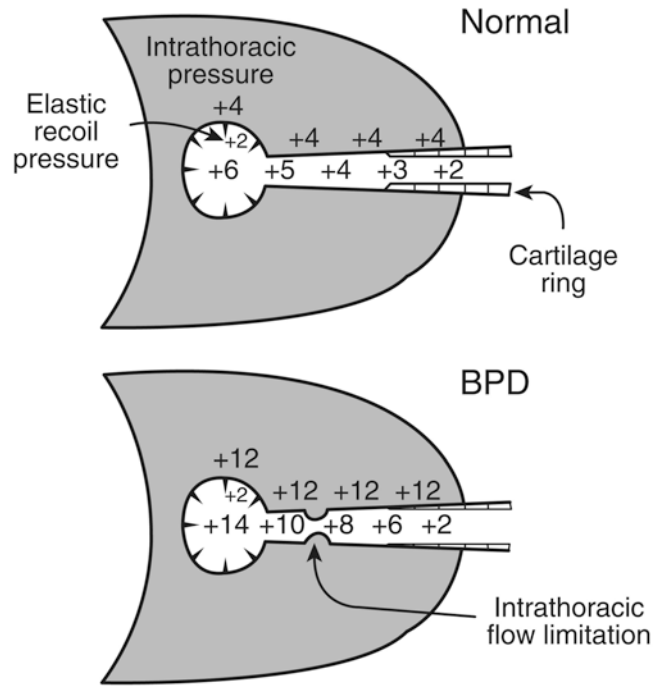

Fig. 4.6 Schematic comparison of normal and abnormal airflow. Infant with bronchopulmonary dysplasia (BPD) has higher transmural pressure generated during tidal breathing and thoracic airways are likely to be compressed during expiration, resulting in a flow limitation (Modified from Bhutani VK, Sivieri EM: Physiological principles for bedside assessment of pulmonary graphics. In Donn SM [Ed.]: *Neonatal and Pediatric Pulmonary Graphics: Principles and Clinical Applications.* Armonk, NY, Futura Publishing Co., 1998, p. 63, with permission.)

3. The bronchomalactic airway may be destabilized in the presence of tracheal smooth muscle relaxants.
4. The effect of some of the other physiologic factors, such as the alveolar duct sphincter tone, is not yet fully understood.

C. Pathophysiologic states
1. Plugging of the airway lumen, mucosal edema, cohesion, and compression of the airway wall lead to alterations in tracheobronchial airflow.
2. Weakening of the airway walls secondary to the structural airway barotrauma and the consequent changes of tracheobronchomalacia also result in abnormal airflow patterns.
3. BPD related airflow effects have also been previously described.

V. Lung Volumes

Ventilation is a cyclic process of inspiration and expiration. Total or minute ventilation (MV) is the volume of air expired each minute. The volume of air moved in or out during each cycle of ventilation is the tidal volume (V_T) and is a sum of the air in the conducting zone (V_D, or dead space) and the respiratory zone (V_A, or alveolar space). Thus,

$$\text{MV} = \left(V_A + V_D\right) \times \text{Frequency}$$

The process of spontaneous breathing generally occurs at about mid total lung capacity such that about two-thirds of the total capacity is available as reserve.

A. Ventilatory Volume:
1. Tidal Volume (V_T): volume of air inspired with each breath.
2. Minute Ventilation: (MV): product of frequency (F, the number of tidal volumes taken per minute) and V_T.
3. Dead Space (V_D): volume in which there is no gas exchange.
   a. Dead space refers to the volume within the respiratory system that does not participate in gas exchange and is often the most frequent and unrecognized cause for hypercapnia.

b. It is composed of several components.
   (1) Anatomic dead space is the volume of gas contained in the conducting airway.
   (2) Alveolar dead space refers to the volume of gas in areas of "wasted ventilation", that is, in alveoli that are ventilated poorly or are under-perfused.
   (3) The total volume of gas that is not involved in gas exchange is called the physiologic dead space. It is the sum of the anatomic and alveolar dead space.
c. In a normal person, the physiologic dead space should be equal to the anatomic dead space. For this reason, some investigators refer to physiologic dead space as pathological dead space.
d. Several factors can modify the dead space volume.
   (1) Anatomic dead space increases as a function of airway size and the airway compliance. Because of the interdependence of the alveoli and airways, anatomic dead space increases as a function of lung volume. Similarly, dead space increases as a function of body height, bronchodilator drugs, and diseases such as BPD, tracheomegaly, and oversized artificial airways.
   (2) Anatomic dead space is decreased by reduction of the size of the airways, as occurs with bronchoconstriction, tracheomalacia, or a tracheostomy.

4. Alveolar Volume (V_A): volume in which gas exchange occurs:

$$V_A = V_T - V_D.$$

5. Alveolar Ventilation (V_A): product of frequency and V_A

B. Lung Reserve Volumes

Reserve volumes represent the maximal volume of gas that can be moved above or below a normal tidal volume (Fig. 4.7). These values reflect the balance between lung and chest wall elasticity, respiratory strength, and thoracic mobility.

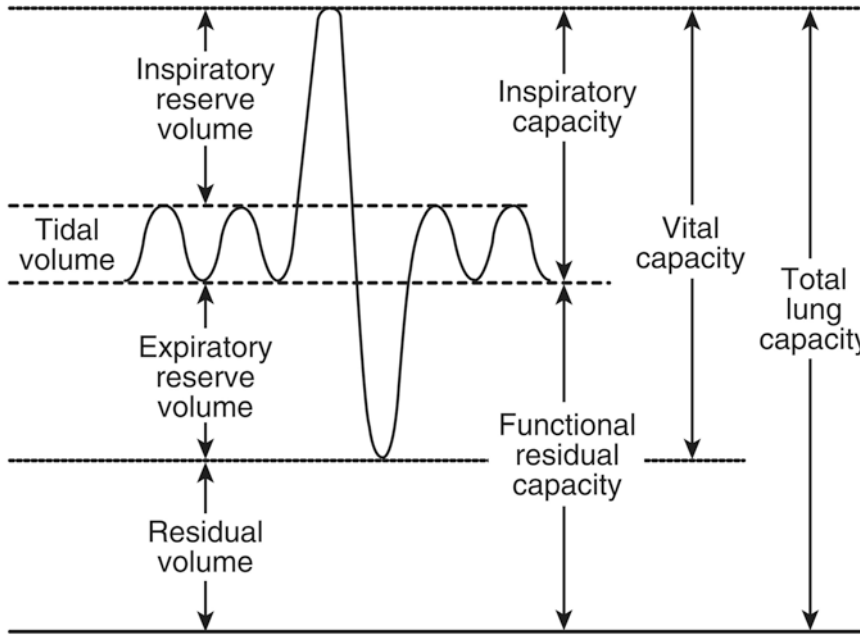

Fig. 4.7 Graphic representation of lung volumes and capacities (Modified from Bhutani VK, Sivieri EM: Physiological principles for bedside assessment of pulmonary graphics. In Donn SM [Ed.]: *Neonatal and Pediatric Pulmonary Graphics: Principles and Clinical Applications.* Armonk, NY, Futura Publishing Co., 1998, p. 67, with permission.)

Table 4.1 Lung volumes in term newborns

Ventilatory volumes	Normal values for term newborns	Static lung volumes	Normal values for term newborns
V_T	5–8 mL/kg	RV	10–15 mL/kg
F	40–60 b/min	FRC	25–30 mL/kg
V_D	2–2.5 mL/kg	TGV	30–40 mL/kg
MV	200–480 mL/min/kg	TLC	50–90 mL/kg
V_A	60–320 mL/min/kg	VC	35–80 mL/kg

1. Inspiratory reserve volume (IRV) is the maximum volume of gas that can be inspired from the peak of tidal volume.
2. Expiratory reserve volume (ERV) is the maximum volume of gas that can be expired after a normal tidal expiration. Therefore, the reserve volumes are associated with the ability to increase or decrease tidal volume. Normal lungs do not collapse at the end of the maximum expiration.
3. The volume of gas that remains is called the residual volume (RV).

C. Lung Capacities

The capacity of the lungs can be represented in four different ways: total lung capacity, vital capacity, inspiratory capacity, and functional residual capacity (Fig. 4.7).

1. Total lung capacity (TLC) is the amount of gas in the respiratory system after a maximal inspiration. It is the sum of all four lung volumes. The normal values as well as the values of static lung volumes for term newborns are shown below in Table 4.1.
2. Vital capacity (VC) is the maximal volume of gas that can be expelled from the lungs after a maximal inspiration. As such, the vital capacity is the sum of IRV + TV + ERV. Inspiratory capacity (IC) is the maximal volume of gas that can be inspired from the resting end-expiration level; therefore it is the sum of TV + IRV.
3. Functional residual capacity (FRC) is the volume of gas in the lung when the respiratory system is at rest; that is, the volume in the lung at the end of a normal expiration that is in continuity with the airways. The size of the FRC is determined by the balance of two opposing forces:
   a. Inward elastic recoil of the lung tending to collapse the lung
   b. Outward elastic recoil of the chest wall tending to expand the lung. Functional residual capacity is the volume of gas above which a normal tidal volume oscillates. A normal FRC avails optimum lung mechanics and alveolar surface area for efficient ventilation and gas exchange.
4. Residual volume (RV): volume of air remaining in the respiratory system at the end of the maximum possible expiration.

$$\text{Expiratory Reserve Volume}\left(\text{ERV}\right) = \text{FRC} - \text{RV}.$$

D. It is important to note that thoracic gas volume (TGV) is the total amount of gas in the lung (or thorax) at end-expiration. This value differs from FRC and the difference would indicate the magnitude of air trapping.