Update for nurse anesthetists

Functional residual capacity: The human windbag

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Like the windbag of a bagpipe, the functional residual capacity (FRC) is the lung volume that acts as a reservoir of air for physiologic use. This reserve volume is particularly important during the period of apnea that occurs during induction of general anesthesia. The balance of the inward elastic recoil of the lung and the outward chest wall forces determines the FRC. Inward recoil forces are dependent on the interaction between the fibrous skeleton of the lung tissue and the alveolar surface tension regulated by pulmonary surfactant.

Positioning and the use of inhaled and intravenous anesthetics influence outward chest wall forces. Factors that affect the FRC may be altered by volume recruitment maneuvers such as administration of vital capacity breaths, the application of positive end-expiratory pressure, and/or maintenance of anesthesia with a fraction of inspired oxygen of less than 1.0.

This course reviews the basic anatomy and physiology of the FRC during the perioperative period. Understanding the processes that contribute to intraoperative loss of lung volume and knowledge of interventions that can alym them are paramount to providing a reliable and safe general anesthetic.

Key words: Alveolar architecture, anesthesia, atelectasis, functional residual capacity.

Objectives

Upon completion of this course, the reader should be able to:
1. Examine the effects of anesthesia on pulmonary mechanics.
2. Discuss the determinants of functional residual capacity.
3. Examine the effects of anesthesia on functional residual capacity.
4. Describe the effects of anesthetic agents on pulmonary mechanics.
5. Analyze the occurrence, causes, and effects of intraoperative atelectasis.

Introduction

A bagpipe is a reed instrument consisting of a melody pipe and one or more accompanying drone pipes protruding from a windbag into which the player blows a reserve of air. The windbag serves as a reservoir that is manipulated by the pressure of the musician’s arm to produce a flow of air into the pipes. The cumulative effects of these components create the unique and unusual sounds produced, and the windbag is crucial to providing the stream of continuous sound. In an analogous manner, the human respiratory system is a bagpipe comprising the laryngeal structures that provide us voice, a series of conducting airways, a reserve volume termed the functional residual capacity (FRC), and the muscles of ventilation that create the pressure-volume relationships required to produce flow. The windbag, or FRC, acts as the reservoir of air into which the tidal volume is exchanged. This is significant because the FRC serves as the patient’s only source of oxygen during the iatrogenically produced apnea that occurs while inducing general anesthesia. Because the balance of the chest wall and lung recoil forces determines the FRC, its volume is dynamic and profoundly affected by common perianesthesia events. This course
reviews the determinants of the FRC and the effects of positioning, age, induction of general anesthesia, and use of anesthesia drugs on this reserve volume.

**Determinants of the FRC**

The FRC is conventionally defined as the volume of gas that remains in the lungs at end expiration. The lung relaxation volume (Vr) is defined as the volume at which the inward elastic recoil of the lung and the outward elastic recoil of the chest wall are equal and opposing forces. These 2 volumes are essentially equal during normal quiet breathing at rest, but they may vary under specific conditions. Both the FRC and Vr decrease when moving from an upright to a supine position. However, under some conditions, FRC and Vr move in opposite directions. In a newborn with high chest wall compliance, Vr is lower than FRC, which is maintained by increasing inspiratory muscle tone and glottic braking. Differentiating FRC and Vr is important to achieve a clear understanding of pulmonary mechanics. It is the FRC that buffers the changes in alveolar gas content from the pulmonary capillary blood so that arterial gas content does not fluctuate significantly.

The volume of the FRC depends on factors that influence both lung and chest wall elastic forces. The inward lung elastic recoil comprises tissue elastic and surface active forces. Tissue forces are created by the flexible state of the fibrous skeleton of the lung parenchyma and the elasticity of the functional alveoli. Surface forces depend on alveolar size and surface tension properties. The outward chest wall elastic recoil comprises the chest wall components and respiratory muscle tone. Chest wall components include the rib cage, the abdomen, and the diaphragm that separates them. The abdominal load and respiratory muscle tone contribute to the chest wall forces that act in opposition to the lung recoil forces.

The FRC is approximately 3.0 L in a 70-kg upright adult and is equal to the total lung capacity (TLC) minus the inspiratory capacity. While the FRC increases with increases in lean body mass, it decreases in obesity, with postural changes, and in restrictive lung disease. Age has no significant effect on FRC, while for the same height, the FRC is about 10% less in women than in men. The most common methods for measuring FRC in a laboratory setting include body plethysmography, nitrogen washout, and closed-circuit helium dilution. The measurement of thoracic gas volume with plethysmography is considered the "gold standard" because it is the only method that quantifies non-communicating gas volumes. Helium and nitrogen dilution techniques measure only communicating gas volumes, but are used more commonly to measure FRC during mechanical ventilation, anesthesia, or both.

**Structure and function of surface forces**

The alveoli often are likened to a collection of grapelike clusters surrounded by a dense capillary bed. However, the alveolar capillary surface area is meticulously structured to comply with certain mechanical specifications. It must: (1) be thin enough to provide for rapid diffusion of gases, (2) contain alveolar walls that respond to minimal pressure change with large volume changes and return to their original volume with ease, (3) tolerate cyclic distention without significantly altered pulmonary capillary blood flow, and (4) possess a method to reduce potentially high surface forces. Any iatrogenic or pathologic event that affects these specifications can be expected to alter normal gas exchange processes.

The pulmonary acinus, or primary lobule, consists of a region supplied by a single first-order respiratory bronchiole, including second- and third-order respiratory bronchioles, alveolar ducts, and alveolar sacs. Thus, the primary lobule represents the Weibel generations 15 through 23. Gas exchange occurs not only at the terminal alveoli, but also across the alveoli interspersed within the respiratory bronchioles and alveolar ducts. This triad of structures forms the functional alveoli. The individual alveoli are not merely grapelike clusters of independent units. They are “wet, hollow structures” that are embedded within a complex fiber skeleton that profoundly affects acinar mechanics. In fact, elements of this fibrous system within the alveolar duct walls interact intimately with alveolar surface forces.

The fibrous framework of the lung has a tripartite organization: (1) peripheral tissue extensions from the pleura into the interlobular and intersegmental septa, (2) axial connective tissue that accompanies the airways to the acini where it continues as the strong fibrous network surrounding the alveolar ducts, and (3) a delicate net of alveolar septal fibers anchored to the axial and peripheral connective tissue and interlaced with the capillaries. The latter 2 components contribute extensively to the mechanics of the pulmonary lobule. While tissue and surface forces interact differently at the extremes of lung volume, alveolar surface tension from 40% to 70% of TLC is the same in both large and small alveoli regardless of alveolar size or location. These conditions, in a normal lung, help to prevent pendelluft, the movement of air from one area of the lung to another without participating in gas exchange. The phenomenon of pendelluft, also referred to as time-constant inequalities, has been demonstrated in anesthetized patients in the lateral decubitus position and in patients with adult respiratory distress syndrome.

The web of axial fibers that forms the alveolar entrance rings interacts with surface forces in surround-
Figure 1. Model for the structure of the alveolar duct

AF = axial connective fibers that form alveolar entrance ring
PF = fibers of the peripheral connective tissue system

Surface tension (\(\gamma\)) directly affects the geometry of alveolar structure. Under conditions of constant lung volume, increasing surface tension increases alveolar duct diameter but decreases alveolar depth and surface area. Under abnormally high surface tension (bottom panel), alveolar pleating occurs and alveolar septa are folded and compressed.

Figure 2. Demonstration of alveolar pleats hidden between apposed pulmonary capillaries in air-filled rabbit lung at 40% of total lung capacity

Pleats are observed both between capillaries (Cap) and between capillaries and the septal midplane (SM). Arrows delineate pleats that may contain small amounts of fluid covered by surfactant.

The alveolar architecture in air-filled lungs resembles irregular polyhedrons in which the walls are flat and form angles with one another. At low lung volumes, septal “pleats” can be observed, predominantly in alveolar corners (Figure 2). In the presence of increased surface tension, alveolar pleating can result in alveolar collapse, regardless of lung volume. In addition, increases in surface tension can increase the size and number of alveolar pleats to a point at which alveolar duct widening becomes prominent. Acinar morphology is markedly altered as “masses of piled-up septa are interposed between widened alveolar ducts.”

Clearly, preservation of low surface tension is an important aspect of maintaining normal acinar function. It is well known that, depending on the volume history of the lung, physiologic differences in the surface tension of functional alveoli at the same lung volume can be observed. The fact that surface tension is higher on lung inflation than lung deflation in an air-filled lung is the explanation for the hysteresis seen on pressure-volume loops. Thus, surface forces act to “derecruit” alveolar surface area. Delivery of a very large volume effectively reduces the influence of surface forces on the compliance curve, leaving mainly the tissue contribution to lung recoil. The administration of vital capacity breaths is believed to modify lung compliance by opening previously closed regions of the lung parenchyma, or “recruiting” areas of pulmonary collapse.

The effect of surface forces on surface area is marked at 40% of TLC (FRC) and minimal at 80% of TLC. At
high lung volumes, the stretched fibrous network provides the majority of the elastic recoil properties of the lung tissue. While ordinary tidal breathing punctuated with frequent breaths to TLC preserves low surface tension, prolonged breathing at FRC without “sighs” produces stiff lungs and areas of lung collapse. Because surface tension increases at constant lung volumes, alveolar ducts widen and alveolar pleats occur, contributing to further decreases in total alveolar surface area.5-7 One might deduce from these physiologic facts that the maintenance of a constant lung volume during mechanical ventilation, without regular punctuation with sighs or vital capacity breaths, ultimately decreases surface area and FRC.

**Role of pulmonary surfactant**

In addition to its micromechanical effects, decreased alveolar size influences the dynamics of the liquid subphase that lines the alveolar capillary membrane. The omission of deep breaths while the patient is mechanically ventilated under anesthesia may result in a decreased content of the active forms of alveolar surfactant.12 Under these conditions, pulmonary surfactant slowly exits from the air-water interface and becomes nonfunctional.

Important characteristics of pulmonary surfactant include its rapid adsorption and spread over the air-water interface, formation of a film that reaches surface tension values close to zero on expiration, and a half-life of more than 10 minutes.6 While the most prominent surface active agent, the phospholipid dipalmitoyl phosphatidylcholine (DPPC), is crucial to maintenance of alveolar stability, at least 3 surfactant proteins (SPs) have been described that contribute to this process. SP-A has a role in surfactant metabolism by regulating surfactant secretion and promoting surfactant reentry into type II pneumocytes. SP-B and, to a lesser extent, SP-C act to enhance the absorption of lipids to the air-liquid interface. SP-B interacts with phosphatidylglycerol in the hypophase to create a DPPC-enriched environment under conditions of lung deflation, thereby reducing surface tension.13

The pool of available surfactant is dynamic and based on the changing demands of each breath. Local factors that augment surfactant production include hyperinflation and stretch of the alveolar surface (deep breaths). Adenosine triphosphate and β-adrenergic agonists increase surfactant secretion. Clearance of surfactant from the alveolar space occurs primarily by reuptake via type II pneumocytes coupled with ingestion by alveolar macrophages and movement up the mucociliary escalator.

The phospholipid molecule has a unique characteristic that provides for the stabilizing effects of surfactant on alveoli: it is amphipathic. One portion of the molecule avoids water, or is hydrophobic, while another portion of the same molecule is water-loving, or hydrophilic. In DPPC, the hydrophobic portion projects into the alveolus, while the hydrophilic portion is oriented toward the alveolar lining fluid in the hypophase. During normal breathing, a balance is maintained among the production, release, and consumption of surfactant so that alveolar stability is maintained during both inspiration and expiration. At end expiration, surfactant molecules are compressed over a smaller alveolar area, generating a low surface tension and preventing collapse. During positive pressure ventilation, alveoli are distended with large positive transpulmonary pressures. The surfactant lining can no longer cover the enlarged alveolar surface area, and surfactant molecules are replenished from the hypophase. During expiration, the transpulmonary pressures are reduced, alveolar surface area has decreased, and some surfactant molecules are lost to the airways. Subsequent inspiration and alveolar enlargement requires additional surfactant to be deposited from the hypophase (Figure 3).14

Thus, cyclic ventilation with large mechanical breaths, particularly to end-expiratory volumes less than FRC, washes out pulmonary surfactant and contributes to surfactant deficiency. Moreover, lung stretch, due to high tidal volumes or high pressures, has been demonstrated to produce both epithelial and endothelial injury.15 Wall stress within distended pulmonary capillaries may be large enough to damage the endothelial layer, resulting in hemorrhage, pulmonary

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**Figure 3. Model of proposed surfactant mechanisms in artificial ventilation**

edema, or both. The overdistention of lung units and/or the shear forces generated due to repeated expansion and collapse of atelectatic lung tissue may initiate or exacerbate structural injury. These phenomena are termed shear stress, or ventilator-induced lung injury. It is hypothesized that “splinting” the alveoli open with a positive end-expiratory pressure (PEEP) of 10 to 15 cm of water can reduce surfactant washout and possibly prevent this type of shear stress phenomenon.

**Structure and function of chest wall components**

Chest wall elastic components traditionally include the rib cage, the abdomen, and the thin muscular diaphragm that separates them. While the diaphragm and external intercostal muscles historically have been considered primary muscles of inspiration, studies have demonstrated that both the internal and external intercostals function as postural muscles during quiet breathing. During eupnea, these muscles serve mainly to facilitate the stability of the torso.

On inspiration, upper rib cage movement is “pump-handle” in character as the anterior ribs become horizontal. The rib-sternal attachments are fairly restrictive, and most movement occurs ventrally rather than laterally. Lower rib movement is “bucket-handle” in character, with less restrictive sternal articulations and more lateral movement on inspiration. The lower ribs are also subject to diaphragmatic forces in the zone of apposition: the inner aspect of the rib cage that is directly apposed to the cylindrical portion of the diaphragm where it inserts on the inner aspect of the lower rib cage. This zone may occupy as much as 30% of the total internal surface area of the rib cage.

Inhaled anesthetics, quantified in relation to minimum alveolar concentration (MAC), affect both primary and accessory muscles of ventilation. Parasternal muscle activity is abolished at a MAC of 1 and in some subjects at a MAC as low as 0.2, which explains the paradoxical inspiratory movements that occur as anesthesia is deepened; diaphragmatic function is preferentially preserved so that diaphragmatic contraction causes expansion of the lower rib cage and abdomen while the upper rib cage collapses inward.

The abdomen acts as an incompressible liquid-filled container that contributes a significant load to chest wall recoil. Its mostly fixed boundaries include the dorsal spine, the caudal pelvis, and the iliac crests laterally. Only its cranial boundary, the diaphragm, and the central abdominal wall are compliant. An inward displacement of abdominal contents by either diaphragmatic relaxation or abdomen rectus contraction must result in an equal outward displacement elsewhere.

The relative contributions to volume displacement of rib cage motion versus diaphragmatic motion have been studied. In an upright position, the rib cage is more compliant than the abdomen and contributes more to the volume displaced. The liquid substance of the abdomen acts to stretch the abdominal wall and reduces its compliance. When a healthy person is placed supine, abdominal compliance increases markedly and allows more prominent abdominal motion. Under some conditions, prone positioning can profoundly affect abdominal load and reduce pulmonary compliance, in particular when the abdomen is not allowed to fall freely. In obese subjects, both awake and under general anesthesia, abdominal mass loading decreases FRC, respiratory system compliance, and oxygenation while increasing the work of breathing.

Respiratory muscle tone also contributes to chest wall forces. Vagally mediated reflexes activate the abdominal muscles when lung volume is increased by postural change or positive pressure ventilation. The reduction in vagally mediated abdominal muscle tone that occurs when subjects are placed supine aids in explaining the increase in abdominal motion that occurs in this position. However, under general anesthesia, active contraction of the abdominal muscles can occur during expiration, at a MAC as low as 0.2 in some patients.

It is well known that volatile anesthetics such as halothane, enflurane, isoflurane, and sevoflurane depress diaphragmatic contractility. These effects are thought to occur due to inhibition at the level of the neuromuscular junction, within the process of excitation-contraction coupling, or via depression of membrane excitability. Evidence has shown that both subhypnotic and anesthetic doses of propofol and sedative doses of midazolam decrease diaphragmatic contractility. The inhibitory effects of propofol may be due to impaired excitation-contraction coupling and/or a decrease in blood flow to the diaphragm caused by decreases in cardiac output common during the use of propofol. The exact mechanism by which midazolam decreases diaphragmatic contractility remains unclear.

**Anesthesia and the FRC**

Events at both the lung and chest wall levels influence pulmonary function during the peri-anesthesia period. The FRC acts as an air-filled cushion, not only buffering changes in pulmonary arterial gas content during ventilation, but also providing a reservoir of oxygen during periods of apnea. Decreasing the volume of this reservoir can profoundly affect oxygenation, particularly during induction of and emergence from general anesthesia. In addition, the volume loss that occurs under anesthesia may be a contributing factor to post-
operative atelectasis, pneumonia, or both.

Oxygen levels fall rapidly and carbon dioxide levels rise when apnea occurs from the end-expiration breathing room air; however, short-term increases in carbon dioxide levels are not associated with the potentially devastating sequelae of acute hypoxia. The partial pressure of dissolved oxygen falls from approximately 100 mm Hg to 60 mm Hg in about 30 seconds, 15 seconds before optimal intubating conditions occur using the ultra-short-acting neuromuscular blocking agent succinylcholine. These data support the use of a pre-oxygenation sequence in all patients before the induction of anesthesia. Physiologic modeling using computer simulation has shown that a decrease in FRC, a short duration of the denitrogenation process, and/or hypoventilation significantly shorten the time to desaturation of hemoglobin during apnea. In all modeled cases, desaturation was more rapid during apnea with a closed or obstructed airway, a condition common during induction of anesthesia than during apnea with an open, unobstructed airway.

Therefore, several events occur during general anesthesia that affect normal lung physiology. First, supine positioning has been shown to reduce the FRC by 0.5 to 1.0 L. This change in FRC occurs even in healthy, awake, spontaneously breathing persons. The FRC is markedly reduced in obese subjects, and when they are in the supine position, the FRC may fall below closing capacity. Large abdominal size can inhibit the normal diaphragmatic movement in the supine position. Other intraoperative events also reduce the FRC, including the Trendelenburg position, the supine lithotomy position, and gas insufflation of the peritoneum. Despite the fact that the FRC increases slightly with aging, after 50 years of age, the closing capacity exceeds FRC even in the upright position (Figure 4). This suggests that middle-aged and elderly people have airway closure and ventilation-perfusion mismatch at resting lung volumes, even before intraoperative events occur.

Second, induction of anesthesia causes a further reduction of FRC by 16% to 20% (400-500 mL). This decrease occurs whether the patient is breathing spontaneously or after paralysis and mechanical ventilation. Froese and Bryan demonstrated that the diaphragm shifts cranially after anesthesia induction with no further shift after paralysis. The fall in FRC occurs immediately on induction and is well established within the first few minutes of anesthesia. A return to normal FRC does not occur for several hours after the termination of the anesthetic.

Three factors may contribute to the reduction in FRC under anesthesia: chest wall configuration, diaphragmatic position, and relative distribution of blood volume between the thorax and the abdomen.

While it has been postulated that movement of blood from the periphery into the central blood compartment might influence FRC under anesthesia, this has not been demonstrated. A reduction in circumference of the chest wall occurs under anesthesia that has been estimated to account for a decrease in FRC of approximately 200 mL. This change may be due to altered rib cage position, altered diaphragmatic position or tone, or changes in spinal curvature.

Awake supine patients maintain an end-expiratory diaphragmatic tone that is thought to prevent the abdominal viscera from impinging on diaphragmatic position. This tone is lost under anesthesia, which permits either cephalad movement of the diaphragm or a change in shape of the diaphragm. The cephalad shift of the diaphragm occurs primarily in its dependent regions and is estimated to contribute to FRC losses of only 30 mL.

In addition, Hedenstierna, Hedenstierna and Tokies, and Rothen and colleagues have developed a body of research that demonstrates the regular occur-

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**Figure 4. The effects of aging on lung volumes and capacities**

- **CC** = closing capacity
- **ERV** = expiratory reserve volume
- **FRC** = functional residual capacity
- **IC** = inspiratory capacity
- **RV** = residual volume
- **TLC** = total lung capacity
- **VC** = vital capacity

(Reprinted from Levitzky. Used by permission from the American Physiological Society).
rence of atelectasis during anesthesia. Atelectasis occurs in 85% to 90% of anesthetized patients, with no correlation with age and little correlation with body configuration. Computed tomography of the chest, obtained awake and after anesthesia, elegantly reveals densities in the dependent regions of both lungs after anesthesia (Figure 5). Notably, these computed tomography scans also consistently show the dome of the right hemidiaphragm ascending in the rib cage. Both of these events could contribute to the volume loss noted after induction of anesthesia.

Atelectasis under anesthesia is unaffected by mechanical tidal volumes delivered at pressures up to 10 cm of water or by sigh breaths at pressures up to 20 cm of water. Airway pressures of 40 cm of water, or vital capacity maneuvers, are required to reopen these atelectatic areas. Recall that normal intraoperative tidal volumes are 10 mL/kg (700 mL in the ubiquitous 70-kg male), with autosigh mechanisms preset at 1.5 times set tidal volume (1,050 mL). A normal vital capacity is 65 to 69 mL/kg or 4,550 mL, 4 times the volume of an automated sigh breath. While the atelectasis can be reduced or eliminated by application of PEEP of 10 cm of water, not all patients undergoing anesthesia can tolerate the cardiovascular effects of PEEP.

Rothen et al demonstrated that the recruitment of alveolar volume after a sigh breath is quickly abated if the patient is ventilated with 100% oxygen. In these studies, the vital capacity maneuver was performed by inflating the lungs to a pressure of 40 cm of water with an inspiratory hold of 15 seconds. In patients ventilated with 40% oxygen after volume recruitment, reexpansion of densities was maintained for approximately 40 minutes, while atelectasis recurred within 5 minutes in patients ventilated with 100% oxygen. These events are consistent with the well-known model of absorption atelectasis—an oxygen-filled alveolar unit trapped behind a closed or partially closed airway will collapse more rapidly than an alveolar unit filled with an oxygen-nitrogen mixture. The mechanics of FRC reduction during general anesthesia, in which end-expiratory volume falls rapidly below closing volume, could create the conditions of partial airway closure necessary for absorption atelectasis to occur.

Last, when a patient is mechanically ventilated, ventilation typically is performed with a constant lung volume. The pulmonary acinus is now operating at a very low lung volume, and its micromechanics are primarily governed by surface forces. Under these conditions, the functional alveoli may be compromised by the formation of alveolar pleats and additional loss of alveolar surface area. By creating a volume history for the acinus, the pressure-volume relationship can be restored to a more favorable position in which tissue, not surface, forces govern lung mechanics.

Thus, on entering the operating room, the patient is subjected to a substantial reduction in FRC from supine positioning, induction of anesthesia, airway closure, ventilation with high fractions of inspired oxygen, and mechanical ventilation. These volume losses can cause clinically apparent disturbances in oxygenation, ventilation-perfusion mismatch, and increased shunting in some patients.

The fact that a majority of patients enter the operating suite without pulmonary volume loss and leave with it suggests that the conduct of the anesthetic may have a role in the development of clinically apparent postoperative atelectasis, pneumonia, or both. Known intraoperative risk factors include location of surgical incision, type of surgical procedure, baseline physical status, and the duration of anesthesia. It is possible that anesthesia duration is merely a proxy indicator of the effects of perianesthesia events on the respiratory mechanics. Coupled with preoperative risk factors such as obesity, preexisting lung disease, or smoking history, inadequate maintenance of pulmonary volume under anesthesia could account for significant postoperative morbidity.
Conclusion
For a multitude of physiologic and pharmacologic reasons, the FRC decreases under most intraoperative conditions. Atelectasis occurs in a majority of patients within minutes of anesthesia induction. While no studies in the anesthesia literature have demonstrated that intraoperative atelectasis and volume loss have long-term detrimental effects, they represent potential causes of postoperative morbidity. Given the charge of the anesthesia provider to maintain homeostasis, it would seem prudent to take steps to prevent or allay these pathologic events.

Research on the safety of induction without complete denitrogenation should be conducted, while identifying patient populations in which the technique would be appropriate or contraindicated. Periodic vital capacity breaths, when possible using a fraction of inspired oxygen of 0.3 to 0.4, should be administered with the caveat that the peak pressures generated potentially could contribute to pulmonary barotrauma. The use of low to moderate levels of PEEP should be investigated as an adjunct to alveolar recruitment efforts intraoperatively.

Due to the well-described effects of general anesthesia on the respiratory system, anesthesia providers should acknowledge that there are no conditions under which an anesthetized patient should be allowed to breathe without assistance—either assisted ventilation via the breathing bag or mechanical ventilation is warranted in all cases. In addition, periodic vital capacity breaths should be administered, regardless of the duration of surgery. Allowing the depletion of the human windbag, or FRC, can adversely affect the function of the entire bagpipe and potentially create disharmony.

REFERENCES


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