General Anesthesia and the Chest Wall

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SYMBOLS AND DEFINITIONS OF TERMS

Chest wall: Rib cage, diaphragm, abdominal contents, and abdominal wall, i.e., all structures outside the lungs that participate in the breathing movements.
Respiratory muscles: Muscles whose actions produce volume changes of the respiratory system during breathing.
Transpulmonary pressure (Ptp or P_l): Pressure difference between airway opening (mouth, nares, or endotracheal tube opening) and visceral pleural surface.
Transpleural pressure: Pressure difference between parietal pleural surface and body surface.
Transrespiratory pressure (Prs): Pressure difference between airway opening and body surface.
Pleural pressure (Ppl): Pressure between visceral and parietal pleura relative to atmospheric pressure. It can be estimated from esophageal pressure (Pes).
Static elastic recoil pressure of the lung (Pset): Transpulmonary pressure at a given lung volume at zero flow.
Pressure-volume (PV) relationship: 1) for the lung = relationship between transpulmonary pressure and lung volume; 2) for the chest wall = relationship between transpulmonary pressure and volume; 3) for the total respiratory system = relationship between transpulmonary pressure and volume.
Static compliance (C_m): Change in volume for a given change in pressure, thus, it is a measure of distensibility. Static compliance is defined as the slope of the static PV curve of the lung (C_L), chest wall (C_w), or total respiratory system (C_r).
Functional residual capacity (FRC): Gas volume remaining in the lungs at the resting expiratory level.
Intrapulmonary distribution of inspired gas: Ventilation per unit lung (gas) volume.

Changes remain unclear. However, an increasing number of studies provide evidence that general anesthetics act initially on the chest wall and that the changes in lung function during anesthesia may be secondary to alterations in chest wall mechanics. It is the purpose of this article to familiarize the reader with this concept.

Historical Notes

The concept that the chest wall is affected by general anesthesia is not new. As early as 1858 John Snow noticed that chloroform anesthesia led to a breathing pattern “... sometimes performed only by the diaphragm whilst the intercostal muscles are paralyzed.” This observation was confirmed in 1925 by Miller who noticed that the inhalation of ether resulted in “... a progressive ascending paralysis of the muscles of respiration, the extent of which provides a valuable indication of the depth of anesthesia.”

Later, muscle paralysis was used by physiologists as a tool to examine the elastic behavior of the chest wall. Because muscle paralysis without anesthesia is very uncomfortable for the subjects, muscle paralysis was combined with general anesthesia in these studies. In 1955, Nims, Conner, and Comroe were concerned that static pressure-volume curves of the respiratory system obtained in the awake state might lack reproducibility because even trained subjects might be unable to completely relax respiratory muscles. They, therefore, used anesthesia and muscle paralysis to examine what they believed was the true passive pressure-volume relationship of the respiratory system. They found that “... the thorax was less compliant when the subjects were anesthetized.” Howell and Peckett and Butler and Smith reinvestigated, in 1957, the pressure-volume relationship of the total respiratory system and, in addition, examined the compliance of its components, i.e., lung and chest wall during anesthesia-paralysis. They found that the compliance of the total respiratory system and lung was markedly decreased. Both groups of investigators hypothesized that this decrease in lung compliance resulted from a change in the distribution of inspired gas.

Campbell, Nunn, and Peckett, in 1958, took this one step further. They found pulmonary gas exchange to be

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impaired in anesthetized-paralyzed, mechanically ventilated subjects compared with awake, spontaneously breathing subjects, and they postulated that changes in the shape of the chest wall may result in an altered distribution of inspired gas. They wrote: "If the pattern of inflation is different, then the distribution of the inspired air will also be different." They also suggested that an incomplete readjustment of pulmonary blood flow to the altered gas distribution may be the cause of impaired pulmonary gas exchange seen during anesthesia: "... in the supine position with an intact chest the distribution of ventilation and blood flow are less 'ideal' during artificial ventilation than during natural breathing."

Support for the hypothesis that changes in chest wall function may result in secondary changes in the function of the lung was provided by studies performed by Caro, Butler, and DuBois,13 in 1960. They showed that chest and abdominal restriction in conscious, spontaneously breathing, seated subjects led to a reduction in lung volumes, decrease in lung compliance, increase in elastic recoil pressure of the lung, altered distribution of inspired gas, decrease in arterial oxygen tension, and increase of intrapulmonary shunting. They stated that "... forces external to the chest cage, by reducing thoracic gas volume, can affect the function of the lungs. We speculate that this effect may occur in other conditions in which there is a reduction of thoracic gas volume ..." and listed, amongst other such conditions, paralysis of respiratory muscles.

Until that time, however, no direct experimental evidence for an altered distribution of inspired gas had been provided. It was not until 1963 that intrapulmonary gas distribution was determined in anesthetized and anesthetized-paralyzed subjects and was compared with the distribution occurring in the awake state.14 Unfortunately, problems with computation and, thus, interpretation of data led to the erroneous conclusion that "enlargement of the thoracic cavity by spontaneous respiratory muscle activity produces the same pattern of lung expansion as application of external pressure to the airway." In 1965, Severinghaus and Larson,15 referring to that paper in a definitive review, concluded that "no impairment of distribution was found." In the same year, Nunn et al.,16 also referring to the same paper, stated "the distribution of inspired gas has now been found to be normal during anaesthesia." This emphasis on one paper by three leading anesthesiology researchers effectively obscured the previous suggestions throughout the 1960s.

During the last decade, interactions between lung and chest wall have regained increasing interest.17-20 New evidence has been provided that 1) in awake man an altered pattern of expansion of the respiratory system may result in an altered distribution of inspired gas;21-23 2) shape and motion of the chest wall are altered during general anesthesia, both with muscle paralysis and mechanical ventilation24,25 and with spontaneous breathing;26,27 and 3) distribution of inspired gas is altered during general anesthesia.28-32 It has been hypothesized that the initial effect of general anesthesia with or without muscle paralysis is an alteration in shape and motion of the chest wall.27,33,34 This in turn alters the distribution of inspired gas and, without appropriate adjustment of pulmonary blood flow, increases ventilation-perfusion (V/Q) mismatching and impairs pulmonary gas exchange.35

Physiology of the Chest Wall

Before considering the effect of anesthesia on the chest wall, we shall review the normal function of the chest wall and its components, including the interaction between diaphragm and thoracic wall. We will then discuss the effects of changes in chest wall motion or shape (or both) on the intrapulmonary distribution of gas. The chest wall, as defined by respiratory physiologists, consists of the rib cage and the abdomen-diaphragm, i.e., all structures outside the lung which participate in ventilation. The chest wall has two major functions, namely, a ventilatory and a stabilizing function. The ventilatory function is provided for during quiet breathing mainly by the phasic activity of the diaphragm. The efficiency of the ventilatory function of the diaphragm depends critically on the function of the rib cage muscles which must stabilize the rib cage against distortions induced by diaphragmatic contraction. During quiet breathing, the muscles of the rib cage preserve the shape of the rib cage by counterbalancing the forces of the diaphragm acting on the rib cage and, hence, allow the diaphragm to function optimally. In erect man, the diaphragm elevates and everts the rib cage during inspiration, using the abdominal contents as a fulcrum.35 The interaction between the diaphragm and the rib cage in supine subjects is less well understood, and coupling of these two components of the chest wall has been said to be less effective in the supine than in the erect position.26 As will be shown, the interaction between the diaphragm and the rib cage may be disturbed by anesthesia during spontaneous breathing, leading to paradoxical ventilation.

While the phasic activity of the respiratory muscles determine the ventilatory function, tonic activity of the respiratory muscles may alter the baseline length of the muscles between phasic contractions. Halothane suppresses the tonic activity of the diaphragm which may be the underlying mechanism for the reduction in FRC seen with general anesthesia.36 If the reduction in FRC was caused by suppression of tonic activity of respiratory
muscles, muscle paralysis would not be expected to induce any further change, as is indeed the case.

**Concept of Chest Wall/Lung Interaction**

The respiratory system functions as a unit, i.e., shape and motion of the chest wall are interrelated with the shape and motion of the lung. Thus, if the motion of the chest wall or its shape are altered, for instance by anesthesia or muscle paralysis (or both), then this may lead to a change in the shape of the lung. These interrelations are complex and will next be reviewed.

Static regional lung expansion and static pleural pressure are not uniform, but show a vertically oriented gradient. This vertical gradient in static pleural pressure was originally considered as the cause for the uneven distribution of regional lung volumes. However, more recent studies have suggested that the vertical pleural pressure gradient may be the result rather than the cause of the uneven distribution of regional lung volumes. Most of this evidence comes from the work of Agostoni, who studied the effect of a number of maneuvers on the pleural pressure gradient. His work can be summarized by stating “that the gradient in pleural pressure reflects, to a great extent, the nonuniform distribution of surface stress necessary for the lung and the thoracic cavity, which have different natural shapes, to conform to each other.” It should be remembered that another major force contributing to the distortion from the natural shape is gravity.

The balance between the ease of distortion of chest wall and lung defines their ultimate shapes. As stated by Macklem and Murphy, “according to Agostoni’s concepts, therefore, the distribution of pleural surface pressure is essentially related to the regional expansion that the lung undergoes in fitting the chest wall, whose shape in turn is determined by the action of gravity on its parts, particularly the diaphragm and the abdominal contents.” According to this hypothesis, changes in the pattern of expansion or shape (or both) of the chest wall should result in a different distribution of inspired gas. However, reports about the relationship between changes in shape of the chest wall and distribution of inspired gas are not consistent. These inconsistencies in experimental results may be explained on the basis that different changes in shape, i.e., proportional and nonproportional, were examined. Proportional changes in shape, i.e., proportional changes of all parts of the chest wall, should not affect the distributions of inspired gas, pleural pressure, or regional lung volumes. Conversely, nonproportional changes in shape may alter distributions of inspired gas, pleural pressure, and regional lung volumes. Species differences in the ease of distortion of the

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**Fig. 1.** Pressure-volume (PV) curves of total respiratory system, lung, and chest wall. Note shift of the chest wall PV curve to the left at high lung volumes and to the right at low lung volumes after induction of general anesthesia. Note marked shift to the right of the PV curves of lung and total respiratory system after induction anesthesia. No further change observed with muscle paralysis. (From Westbrooke et al. By permission of the American Physiological Society.)

**Fig. 2.** Pressure-volume (PV) curve of chest wall and lung. Solid line (Pes) represents normal chest wall PV curve of an awake, relaxed subject in the supine position. Heavy dashed line represents transpulmonary pressures (Ptp) required to balance the chest wall if FRC decreased with no change in the chest wall PV characteristics. Open circles represent static lung recoil pressures measured at FRC in anesthetized subjects in the supine position. Lightly dashed line (Ptp corrected) represents Ptp corrected for underestimation when using esophageal pressures. Note that even with this correction applied, values in anesthetized subjects are lower than the predicted, suggesting that the PV curve of the chest wall was shifted to the right with induction of general anesthesia. (From Westbrooke et al. By permission of the American Physiological Society.)
chest wall also may have contributed to the different findings. In addition, changes in the distribution of inspired gas and regional lung volume may be reduced by compensatory mechanisms such as slippage of lung lobes, mechanical interdependence of lung parenchyma, and collateral ventilation.

In summary, the chest wall consists of the rib cage and the abdomen-diaphragm. There is a close interaction between thoracic wall and diaphragm such that, if the function of one is altered, the efficiency of the other may be affected. According to current thinking, changes in inspired gas distribution are due to changes in the shape or motion (or both) of the chest wall. In contrast, the converse may not be true; changes in shape or motion (or both) do not have to result in changes in gas distribution, namely, if a proportional shape change occurred. We have found in anesthetized dogs that the regional pulmonary clearances of $^{133}$Xenon did not differ between mechanical ventilation and spontaneous breathing in spite of paradoxical ventilation.43

### Chest Wall Mechanics, Lung Mechanics, and Regional Lung Function During General Anesthesia

Having considered the physiology of the chest wall, including the coupling of diaphragm and rib cage and the interaction between chest wall and lung, the disturbances in chest wall function induced by general anesthesia and muscle paralysis will now be reviewed. Induction of general anesthesia in supine subjects decreases FRC and compliances of the lung and total respiratory system38,44 (fig. 1). The pressure–volume (PV) curve of the chest wall tends to shift to the left at higher lung volumes and to the right at lower lung volumes, i.e., at low lung volumes its normal tendency to increase lung volume is reduced (fig. 2). Muscle paralysis induces no further change. These findings led Westbrook et al.34 to postulate that “... the initial effect of our anesthetic procedure was to cause a change in the PV characteristic of the chest wall such that a reduction in FRC occurred. In turn, as the subject breathed at an abnormally low FRC, the lung became stiff, possibly because of a combination of a change in the surface compliance and the occurrence of atelectasis.” This hypothesis is supported by the observation that the decreases in FRC and lung compliance observed in recumbent, anesthetized man do not seem to be due to a direct pharmacologic effect of the anesthetic agent on the lung, in that they do not occur with the same anesthetic regimen in seated subjects.45 In interpreting the study by Westbrook et al., however, it must be remembered that a shift in the PV curve of the chest wall may reflect, at least in part, an incomplete muscular relaxation of the subjects while they were

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**Fig. 3.** Position of the diaphragm and its displacement during tidal breathing in a subject in supine position. Dashed lines represent position of the diaphragm at awake functional residual capacity. Stippled areas represent diaphragmatic excursions during tidal breathing. The cephalad border of the stippled areas thus represents the end-expiratory position of the diaphragm. Note cephalad shift of the diaphragm at end-expiration in the anesthetized and awake-paralyzed states. Note also reversal of the pattern of diaphragmatic excursions with subject awake-paralyzed, mechanically ventilated compared to the spontaneously breathing awake or anesthetized subject. (From Froese and Bryan.44 By permission of the American Society of Anesthesiologists, Inc.)

**Fig. 4.** Intrapulmonary inspired gas distribution determined with $^{133}$Xenon, expressed as ventilation index ($V_i$, per cent), plotted as a function of vertical distance down the lung. Note more uniform distribution of inspired gas after induction of anesthesia-paralysis in the supine position, totally uniform distribution in the right lateral decubitus position, and less uniform distribution in the sitting position. No difference is seen between the awake and anesthetized-paralyzed states in the prone position. (From Rehder et al.29 By permission of the American Physiological Society.)
awake, although this was considered not to be a major factor. Furthermore, in supine subjects, estimates of pleural pressure from esophageal pressure may be affected by mediastinal artifacts and, finally, the elastic behavior of the esophagus may change following induction of anesthesia. We believe that none of these potential problems invalidate the conclusions of Westbrook and associates. First, in anesthetized recumbent dogs, esophageal pressure closely reflects the surrounding pleural pressure, and, second, in supine dogs, esophageal elastance does not change following induction of anesthesia.

Further evidence implying a change in the mechanical properties of the chest wall following induction of general anesthesia is the finding of a cephalad shift of the dependent parts of the diaphragm in recumbent subjects (fig. 3). Loss of tonic activity of the diaphragm during halothane-induced anestheisa has been demonstrated and may be responsible for the cephalad shift of the diaphragm following general anesthesia in subjects in recumbent positions. This argument is further supported since this shift is not exaggerated by additional muscle paralysis. The pattern of diaphragmatic motion is also different between spontaneous breathing and mechanical lung inflation after muscle paralysis (fig. 3). These findings of both altered shape and motion of the diaphragm during general anesthesia and muscle paralysis confirm and extend previous observations that gas distribution in anesthetized-paralyzed, mechanically ventilated subjects is altered from that in the awake state (fig. 4). The finding of an altered distribution of regional lung volumes after induction of anesthesia-paralysis is also consistent with an initial effect of anesthesia on the shape of the chest wall. That changes in the shape of the chest wall can alter the distribution of inspired gas has been shown in awake man in the lateral decubitus position. Voluntary relaxation of the diaphragm during intermittent positive-pressure breathing in the lateral decubitus position results in an altered gas distribution that is strikingly similar to the distribution observed in anesthetized-paralyzed subjects.

Support for an effect of general anesthesia on the chest wall also comes from the observation that, in man, halothane anesthesia without muscle paralysis and, in dogs, enflurane anesthesia without muscle paralysis decreases expansion of the rib cage and may lead to paradoxical ventilation (fig. 5). A major component of the ventilatory depression associated with halothane anesthesia results from the preferential suppression of intercostal muscle function with relative sparing of the

Fig. 5. Chest wall motion in anesthetized supine dog. Note marked paradoxical motion during anesthesia and spontaneous respiration (heavy lines), i.e., inward motion of lateral rib cage (right upper panel) and anteroposterior abdomen (left lower panel) during early part of inspiration; outward motion of lateral rib cage (right upper panel) during early part of expiration. (From Schmid et al.) By permission of the American Physiological Society.)

Fig. 6. Intrapulmonary distribution of blood flow at FRC determined with $^{133}$Xenon, expressed as perfusion index ($Q_l$, per cent), plotted as a function of vertical distance down the lung. Note less uniform blood flow distribution in the right lateral decubitus position and more uniform $Q_l$ in the sitting position. Following anesthesia-paralysis, $Q_l$ is not significantly different between the awake and anesthetized-paralyzed states in the supine position. (From Rehder.) By permission of the Canadian Anaesthetist Society.)
diaphragm.26 However, one cannot conclude, for reasons outlined above, that every "reduction in amplitude of rib cage movement results in a redistribution of $V_{A}/Q$ ratios within the lung.27"

Additional evidence that alterations in chest wall function may lead to secondary changes in lung function has come from studies using chest or abdominal (or both) restriction in conscious seated man. Chest or abdominal restriction (or both)13,48-56 lead to a reduction in FRC, increased lung recoil pressure, decreased closing capacity, decreased lung compliance, and altered pulmonary gas exchange—changes similar to those seen with general anesthesia.34,51,52 The mechanism for the increase in lung recoil during chest restriction is unknown, but it is probably due to a decrease in the compliance of the alveolar surface lining layer as a result of breathing at low lung volumes56 rather than due to "airway closure," gas trapping, or atelectasis.

Altered function of the chest wall leading to an altered inspired gas distribution during general anesthesia, both with spontaneous breathing53 and with muscle paralysis and mechanical ventilation,28-32 may cause increased ventilation to perfusion ($V_{A}/Q$) mismatching because intrapulmonary distribution of perfusion fails to adjust to the altered gas distribution32,54 (figs. 6–8). In sitting subjects regional intrapulmonary distribution of perfusion becomes more uniform after induction of general anesthesia and paralysis at a time when regional distribution of

Fig. 8. Ventilation to perfusion ($V_{A}/Q$) relationship in a subject in the supine position while awake and after induction of anesthesia-paralysis. Top Panels, Retention vs. blood/gas partition coefficient ($\lambda$) for six inert gases. Dashed lines represent theoretical retentions for lungs with homogeneous $V_{A}/Q$ equal to mean $V_{A}/Q$. Measured retentions are indicated by (•). Middle and Bottom Panels, Distributions of perfusion and ventilation relative to $V_{A}/Q$ ratio. Note increased dispersions of both distribution of perfusion and distribution of ventilation after induction of anesthesia-paralysis. (From Kehrer et al.26 By permission of the American Physiological Society.)
inspired gas has become less uniform. In subjects in the lateral decubitus position, the regional distribution of perfusion becomes less uniform, while inspired gas distribution becomes more uniform. In contrast, in subjects in the supine position, distribution of regional perfusion remains unchanged, while distribution of ventilation becomes more uniform.

The failure of the regional distribution of pulmonary blood flow to adjust to the altered distribution of inspired gas may be due to an altered responsiveness of pulmonary blood vessels during general anesthesia. Such a change in responsiveness, with impairment of hypoxic pulmonary vasoconstriction, with anesthetics has been demonstrated. However, impaired hypoxic pulmonary vasoconstriction is not necessary to explain the failure of regional pulmonary blood flow to adjust to the altered gas distribution. Similar increases in mismatching of regional \( V/Q \) as observed during anesthetia-paralysis, also occur in awake man if the diaphragm is relaxed voluntarily during passive inflation of the lungs.

In summary, there is increasing evidence that general anesthesia initially alters the mechanical properties of the chest wall, such that a reduction in FRC occurs. Breathing at low lung volumes may then alter the alveolar surface lining layer, causing the elastic recoil of the lung to increase and lung compliance to decrease, i.e., the changes in lung function are secondary to the changes in chest wall function, changes very similar to those seen with chest restriction. Alterations of the mechanical properties of the chest wall may be largely responsible for increased \( V/Q \) mismatching during general anesthesia. However, anesthesia also may have an effect on the airways of the lung which could contribute to the altered lung function.

**Summary and Conclusions**

The mechanisms underlying impairment of pulmonary gas exchange during general anesthesia with or without muscle paralysis and mechanical ventilation are becoming clearer. It appears that induction of general anesthesia, with or without muscle paralysis, results in an altered shape and motion of the chest wall which changes the mechanical properties of both the chest wall and, secondarily, the lung. The latter is indicated in some patients by a reduction in FRC, an increase in lung recoil, and a decrease in lung compliance. These changes result in an altered distribution of inspired gas. An inadequate adjustment of pulmonary blood flow to the altered distribution of inspired gas leads to an increase in \( V/Q \) mismatching and to impairment of both oxygenation (due to the development of lung regions with low \( V/Q \) ratios) and CO\(_2\) elimination (due to the development of lung regions with high \( V/Q \) ratios). These changes also are seen in awake man when chest wall function is altered by chest wall or abdominal (or both) restriction sufficient to reduce his FRC by a similar amount.

Knowledge of the concept that changing the shape and motion of the chest wall may alter the distribution of inspired gas helps in understanding why and how pulmonary gas exchange may be altered, not only with general anesthesia but also under other conditions. Changes in body position, neuromuscular disease affecting respiratory muscle function, altered chest wall motion following rib fractures (flail chest), altered chest wall motion with deformation of the chest wall (as is seen with kyphoscoliosis), and restriction of chest wall motion after abdominal or thoracic incision may all contribute to an altered distribution of inspired gas and, hence, may result in impaired pulmonary gas exchange.

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