5 Physiology of the Lateral Decubitus Position, Open Chest and One-Lung Ventilation

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Key Points

- Ventilation and perfusion matching is optimized for gas exchange.
- Induction of anesthesia, one-lung ventilation (OLV) and opening of the chest progressively uncouple ventilation–perfusion (*V*/*Q*) matching.
- Hypoxic pulmonary vasoconstriction (HPV) improves *V*/*Q* matching during OLV, but can be impaired by anesthetic interventions.

Introduction

Early attempts at intrathoracic surgery in nonventilated patients were fraught with rapidly developing respiratory distress and a fast moving operative field. The difficulty with performing a thoracotomy in a spontaneously breathing patient, for both the patient and the surgeon, is easily explained by two phenomena: *Pendel-luft* and *Mediastinal shift* (Fig. 5.1) [1]. Both phenomena can be explained by the fact that the pleural interface has been disrupted in the open hemithorax so that no negative intrathoracic pressure is being created in response to a spontaneous inspiratory effort and chest-wall expansion. In the closed hemithorax, on the other hand, chest-wall expansion and the resulting negative intrathoracic pressure will produce gas flow into the lung via the mainstem bronchus. However, inspiratory gas flow will not only come from the trachea, but also from the operative lung, which is free to collapse due to the surgical pneumothorax. Inspiration therefore results in nonoperative lung expansion and operative lung retraction. The reverse process occurs during expiration, where bulk expiratory gas flow, from the nonoperative lung, not only escapes via the mainstem bronchus into the trachea, but also back into the re-expanding operative lung. This process results in the "pendular" motion of the lung with inspiration and expiration. Mediastinal shift occurs due to a similar process. The negative inspiratory pressure in the closed hemithorax is equally applied to the mediastinum, which is secondarily pulled away from the open thorax during inspiration. The reverse is true during expiration, where positive intrathoracic pressure pushes the mediastinum across into the open thorax. When combined, these two mechanisms explain the difficult exposure for the operating surgeon due to a fast moving operating field, and the rapidly developing respiratory distress in the patient secondary to inefficient to-and-fro ventilation with limited CO_2 elimination and fresh gas entrainment (Fig. 5.1). Interestingly, awake thoracic surgery is being re-popularized in certain high-risk individuals, but the use of video-assisted thoracoscopic surgery (VATS), which avoids opening of the hemithorax, minimizes the above stated issues [3].

Selective ventilation of one lung was first described in 1931 and quickly resulted in increasingly complex lung resection surgery [4]. While infinitely better tolerated than spontaneous respiration, hypoxia was a frequent occurrence during the early years of OLV. Extensive research over the ensuing decades has clarified the basic physiology governing pulmonary perfusion (*Q*) and ventilation (*V*), as well as the disturbances that are

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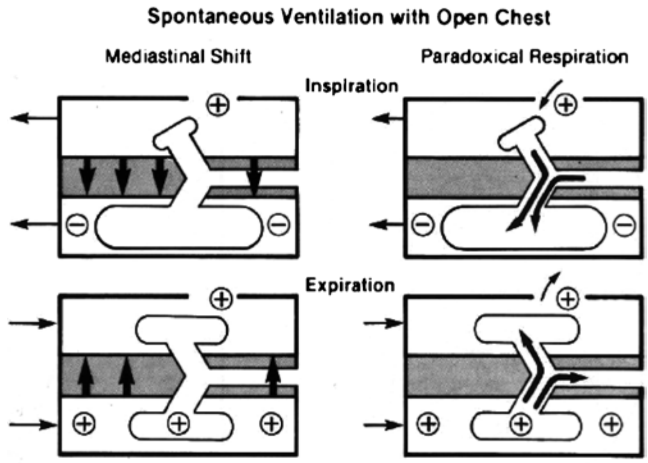

FIG. 5.1. Schematic representation of "Pendel-luft" and mediastinal shift. See text for details (modified from Benumof [2]. © Elsevier 1995).

caused by anesthetic and surgical interventions. Knowledge of the basic physiology is necessary to appreciate ventilation/perfusion (*V*/*Q*) disturbances during OLV.

Perfusion

Pulmonary blood flow is essential for multiple processes. Pulmonary arterial blood carries carbon dioxide to the alveoli for removal and exhalation. Pulmonary venous blood provides filling and oxygen to the left heart to support systemic perfusion and metabolic oxygen demand, respectively. Because of the closed nature of the circulatory system, the entire cardiac output (CO) has to pass through the pulmonary circulation. Pulmonary perfusion pressures are significantly lower than systemic perfusion pressures and become further reduced by 1-cm H_2O for each centimeter of elevation that blood flow has to travel above the level of the heart. Perfusion is therefore not uniform across the lung, as pulmonary arterial (P_{pa}) and venous (P_{pv}) pressures are dependent on the relative elevation above the heart, whereas the extrinsic compressive force of the alveolar distending pressure (P_A) is relatively constant. The interplay of pressures across the lung results in distinct territories of lung perfusion, which are known as the West Zones (Fig. 5.2a) [7, 8]. Zone 1 exists in the most superior aspect of the lung and is characterized by alveolar pressures that exceed intravascular pressures ($P_A > P_{pa} > P_{pv}$). This results in capillary collapse and secondary complete obstruction of blood flow. Zone 1 therefore represents alveolar "dead space." While Zone 1 is minimal under normal circumstances, it may increase in the presence of increased P_A (positive pressure ventilation) or decreased P_{pa} (decreased CO). Moving inferiorly in the lung, P_{pa} values increase due to the lesser elevation above the heart and begin to exceed P_A. This characterizes Zone 2 ($P_{pa} > P_A > P_{pv}$) where P_{pa} exceeds P_A resulting in capillary blood flow. As P_A continues to exceed P_{pv},

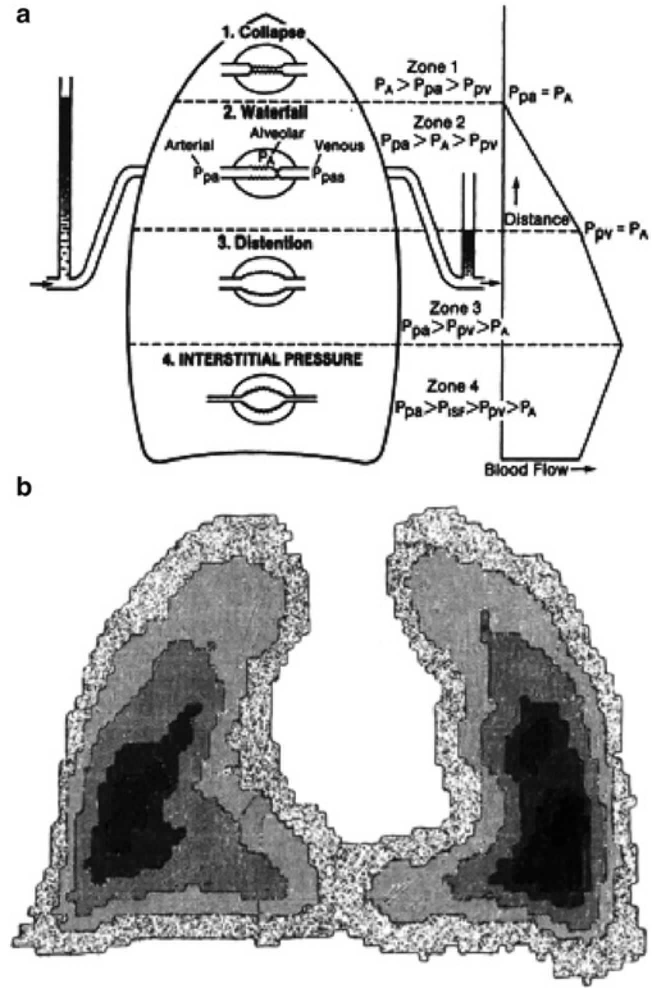

FIG. 5.2. (**a**, **b**) Pulmonary blood flow distribution as it relates to the alveolar pressure (P_A), the pulmonary arterial pressure (P_a), the pulmonary venous pressure (P_v) and the interstitial pressure (P_{is}) at various gravitational levels. Classic West Zones of blood flow distribution in the upright position (**a**). In vivo perfusion scanning illustrating central-to-peripheral, in addition to gravitational blood flow distribution, in the upright position (**b**). See text for further details ((**a**) modified from West [5] and (**b**) Hakim et al. [6], with permission).

capillary blood flow remains dependent on the differential between P_{pa} and P_A. This relationship has been likened to a waterfall, as the amount of flow is dependent on the upstream "water" pressure (P_{pa}), relative to the level of the mountain ledge or dam (P_A), but independent of the downstream "water" level (P_{pv}). Zone 3 ($P_{pa} > P_{pv} > P_A$) is reached when P_{pv} begins to exceed P_A, resulting in pulmonary perfusion independent of P_A and only determined by difference between P_{pa} and P_{pv}. Zone 4 ($P_{pa} > P_{is} > P_{pv} > P_A$) is that portion of the lung where interstitial pressure P_{is} is higher than venous pressure P_{pv}, resulting in a reduction in blood flow relative to the pressure differential between P_{pa} and P_{is}. This is analogous to the patient with increased intracranial pressure (ICP) due to cerebral edema, where the "interstitial" pressure (ICP) exceeds the venous outflow pressure (CVP) and therefore reduces the cerebral perfusion pressure. Zone 4 can exist in the most inferior portions of the lung, or may alternatively be created by exhalation to low lung volumes or increased interstitial pressures such as in volume-overload [8]. One should keep in mind that the West zones are an oversimplified static picture of a dynamic, cyclical system, as lung regions may move through various zones depending on the stage of the cardiac and respiratory cycle that they are in. For example, a given zone 2 lung region may become zone 1 during diastole (low P_{pa}) and positive pressure inspiration (high P_A) or may become zone 3 in systole (high P_{pa}) and mechanical expiration (low P_A). The gravitational model of the West zones helps to illustrate the basis of *V*/*Q* mismatch in the lungs, but only partially reflects human physiology. In vivo perfusion scanning, with tagged albumin micro-aggregates in healthy volunteers, has demonstrated a combination of gravitational distribution and an "onion-like" layering, with reduced flow at the periphery of the lung and higher flow toward the hilum (Fig. 5.2b) [6]. It has also been shown that the perfusion of the left lung, in the dependent left lateral decubitus position, is lower than would be expected based simply on gravity redistribution. Compression and/or distortion elicited by the heart and mediastinum is the likely cause for this reduction [9].

The pulmonary vascular bed is a low-resistance conduit and possesses significant recruitable territory, which helps to offset any increases in pressure. Mild increases in P_{pa} cause progressive recruitment of previously nonperfused vasculature. Once recruitment is complete, further increases in P_{pa} distend the pulmonary vessels, which accommodates increases in blood flow and helps to minimize increases in right ventricular afterload. These modifications allow pulmonary pressures to stay low, even when CO is increased to levels as high as 30 L/min during exercise [10]. At extreme levels of P_{pa}, distention of blood vessels will fail to decrease intravascular pressures resulting in transudation of fluid into the interstitium [11]. Vascular resistance within the pulmonary circulation is also influenced by the degree of lung inflation. There are two populations of pulmonary vessels that exhibit opposing responses to lung inflation. Alveolar capillaries are exposed to intra-alveolar pressures and therefore experience increasing resistance to flow, or may actually collapse, as lung volumes increase. Intraparenchymal, extra-alveolar vessels, on the other hand, experience outward radial traction with lung expansion, which progressively decreases their resistance. The cumulative effect is a parabolic resistance curve, with minimal pulmonary vascular resistance (PVR) at functional residual capacity (FRC) and progressive increases in resistance at extremes of lung volume (Fig. 4.5).

HPV

Oxygen-sensing mechanisms are active throughout the human body (placenta, ductus arteriosus, carotid body and pulmonary arteries) and have been reviewed in detail [12]. HPV of the pulmonary arterial bed is one such mechanism. In the fetus HPV-induced high PVR results in diversion of blood flow across the

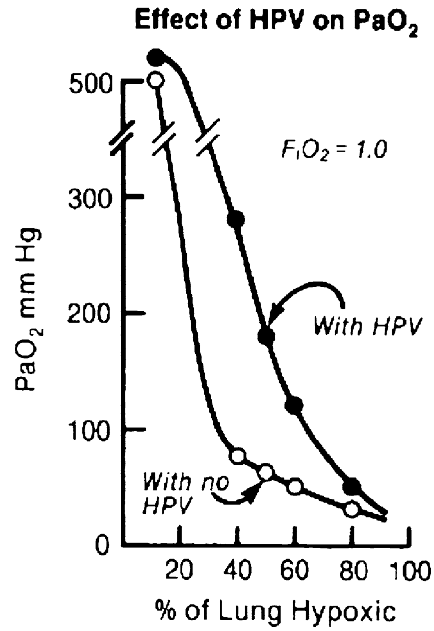

FIG. 5.3. Model of the effect of hypoxic pulmonary vasoconstriction (HPV) on P_aO_2 as a function of the percent of lung that is hypoxic. The model assumes an F_iO_2 of 1.0, normal hemoglobin, cardiac output and oxygen consumption. The HPV benefit is maximal when 30–70% of the lung are hypoxic (modified from Benumof [2]. © Elsevier 1995).

foramen ovale and ductus arteriosus. HPV remains important ex utero, as it allows *V*/*Q* matching by reducing perfusion to poorly oxygenated lung tissue. HPV is active in the physiologic range (P_AO_2 40–100 mmHg in the adult) and proportional to not only the severity of the hypoxia, but also the amount of hypoxic lung. HPV is maximal if between 30 and 70% of the lung are hypoxic (Fig. 5.3). Low partial pressure of oxygen results in inhibition of potassium currents, which leads to membrane depolarization and calcium entry through L-type calcium channels. Extracellular calcium entry, plus calcium release from the sarcoplasmic reticulum, culminates in smooth muscle contraction, primarily in low-resistance pulmonary arteries with a diameter less than 500 μm [12]. The primary stimulus for HPV appears to be the alveolar partial pressure of oxygen (P_AO_2); however, the pulmonary venous partial pressure of oxygen (P_vO_2) is also involved. HPV is maximal at normal P_vO_2 levels, but is inhibited at high or low levels. Low P_vO_2, for example in low CO states, results in a decrease in P_aO_2 and therefore generalized, competing vasoconstriction. Conversely, high P_vO_2 in the setting of sepsis will decrease the vasoconstrictor response in hypoxic areas due to the generalized increase in P_aO_2. Vasoconstriction occurs in a biphasic temporal fashion. The early response occurs within seconds and reaches an initial plateau at 15 min, followed by a late response resulting in maximal vasoconstriction at 4 h [13–16]. HPV reduces the shunt flow through the operative lung by roughly 40%, facilitating the safe conduct of OLV (Fig. 5.4), although some have questioned its true clinical importance [17].

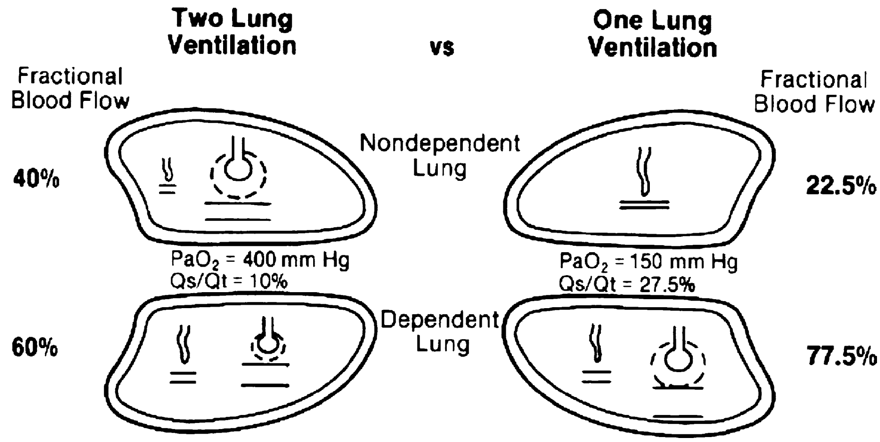

FIG. 5.4. Schematic representation of two- vs. one-lung ventilation. Typical values for fractional blood flow to the nondependent and dependent lungs as well as P_aO_2 and Q_s/Q_t for the two scenarios are shown. The Q_s/Q_t during two-lung ventilation is assumed to be distributed equally between the two lungs (5% each). The main difference between two- and one-lung ventilation is the obligatory shunt through the nonventilated lung. HPV is able to reduce the shunt flow through the nondependent lung by 50%. Total shunt fraction in the one-lung ventilation setting consists of the residual shunt flow through the nondependent lung plus the baseline 5% shunt through the dependent lung (modified from Benumof [2]. © Elsevier 1995).

Extremes of HPV may cause harm. Over-activity, particularly during exercise at high altitudes, may result in high-altitude pulmonary edema [14]. The opposite is true in thoracic anesthesia where inhibition of HPV may result in intra-operative hypoxemia. Many studies have attempted to identify agents or interventions that potentiate or inhibit the pulmonary vasoconstrictor response to hypoxia. Most research has been performed on animals, as interventions are more easily standardized. Perioperative HPV modifiers are summarized in Table 5.1.

Anesthetic Modifiers

Inhibition of HPV by inhalational anesthesia has long been recognized. Ether, halothane and nitrous oxide inhibit HPV in a dose-dependent fashion, and the underlying intracellular mechanisms have been described for halothane [64]. The effect of the newer inhalation anesthetics such as isoflurane, desflurane and sevoflurane is less certain. They appear to be neutral toward HPV, or at least not cause significant depression in clinically relevant doses. Intravenous anesthesia with propofol has been proposed as a means of avoiding HPV modulation, but the improvement in oxygenation is clinically insignificant, except in marginal patients. Results on the influence of thoracic epidural anesthesia (TEA) on oxygenation have been conflicting. Garutti et al. showed an increase in pulmonary venous admixture and secondary worse oxygenation, which may have been due to a drop in CO [65]. Multiple other studies failed to demonstrate an effect of TEA on oxygenation during OLV, when hemodynamic variables were maintained [34–36]. Traditional thoracic teaching has emphasized to keep patients warm and dry, which is supported by the fact that hypothermia and both, hemodilution and increased left atrial pressure, inhibit HPV. Almitrine and nitric oxide (NO) have been shown to provide a potential avenue for HPV modulation. Almitrine, a respiratory stimulant that causes pulmonary

TABLE 5.1. Peri-operative modifiers of hypoxic pulmonary vasoconstriction.

	Effect	References
Patient factors		
COPD	–	[18]
Cirrhosis	–	[19]
Sepsis	–	[20][a]
Pregnancy	–	[21][a]
Female sex	–	[22][a]
Exercise	–	[23][a]
Systemic HTN	+	[24]
EtOH	+	[25][a]
Physiologic changes		
Metabolic acidosis	+	[26][a]
Respiratory acidosis	0	[26][a]
Metabolic alkalosis	–	[26][a]
Respiratory alkalosis	–	[26][a]
Hypercapnea	+	[13]
Hypocapnea	–	[13]
Hyperthermia	+	[27][a]
Hypothermia	–	[27][a]
Increased LAP	–	[28][a]
Increased P_vO_2	–	[29][a]
Decreased P_vO_2	+	[29][a]
Peri-operative interventions		
Trendelenburg	–	[30]
Lateral decubitus	+	[31]
Supine position	0	[31]
Surgical lung retraction	+	[32]
Hemodilution	–	[33]
Epidural anesthesia	0	[34–37]
Inhaled NO	0	[37]
Pharmacologic agents		
Inhalational anesthetics		
Nitrous oxide	–	[38]
Halothane	–	[39]
Enflurane	0	[40]
Isoflurane	0/–	[41]
Desflurane	0	[42]
Sevoflurane	0	[43]

(continued)

Table 5.1. (continued)

	Effect	References
Intravenous anesthetics		
Propofol	0/+	[43, 44][a]
Ketamine	0	[44][a]
Opioids	0	[45][a]
Calcium channel blockers		
Verapamil	–	[39]
Diltiazem	0	[46]
Adrenergic blockers		
Propranolol	+	[47][a]
Phenoxybenzamine	–	[47][a]
Phentolamine	–	[48]
Clonidine	+	[49][a]
Vasodilators		
Hydralazine	–	[48]
Nitroglycerin	–	[50][a]
Nitroprusside	–	[51]
Sildenafil	0	[52]
Vasoactive agents		
Dopamine	?	[53][a]
Isoproterenol	–	[54][a]
Norepinephrine	–	[54][a]
Phenylephrine	+	[55]
Vasopressin	0?	[56][a]
Other		
Losartan (ARB)	–	[57]
Lisinopril (ACE-I)	–	[58]
Methylprednisolone	0	[59]
Indomethacin	+	[50][a]
ASA	+	[50][a]
Prostacyclin	–	[60]
PGE_1	–	[61][a]
Salbutamol	+	[62]
Atrovent	+	[62]
Lidocaine	+	[38][a]

[a]Animal data

Modified from Lohser [63], with permission

vasoconstriction when given intravenously, has been shown to potentiate HPV and improve oxygenation. Endogenous NO causes vasodilation and thereby inhibits HPV; however if given by the inhalational route to the ventilated lung during OLV, NO causes localized vasodilation and thereby decreases shunt fraction. The combination of intravenous almitrine with inhaled NO results in synergistic improvement in *V*/*Q* matching and oxygenation. Almitrine, however, is not widely available and is associated with the potential for significant toxicity. Although clearly efficacious, the focus on HPV manipulation with potentially dangerous agents such as almitrine has been called a distraction from more common reasons for desaturation, such as hypoventilation of the dependent lung [17].

Other Modifiers of HPV

Surgical retraction can assist HPV by increasing PVR in the operative lung [32]; however, the release of vasoactive substances secondary to the manipulation may conversely result in an inhibition of HPV [66]. Ligation of pulmonary vessels during lung resection results in the permanent exclusion of vascular territory and thereby a reduction in shunt flow [66]. The side of surgery influences the extent of shunt flow, as the larger right lung receives a 10% higher proportion of CO than the left lung. Positioning is important as the lateral decubitus position allows for a gravity-induced reduction in shunt flow to the nondependent lung. Procedures that call for supine positioning, on the other hand, are hampered by higher shunt flow to the nondependent lung and may have higher rates of intra-operative desaturations [31]. Similarly, addition of a head-down tilt to the left lateral position has been shown to worsen oxygenation during OLV, likely due to dependent lung compression by abdominal contents [30].

Cardiac Output and Arterial Oxygenation

Arterial oxygen content (CaO_2) is influenced by end-capillary oxygen content (CcO_2), oxygen consumption (VO_2), CO (Q_t) and shunt flow (Q_s). CaO_2 can be calculated using Eq. (5.1), and the interaction of the various factors on CaO_2 is illustrated in Fig. 5.5 [67].

$$CaO_2 = CcO_2 - (VO_2 / Q_t) \times \left(\frac{Q_s / Q_t}{10 \times (1 - Q_s / Q_t)} \right) \tag{5.1}$$

The influence of CO on arterial oxygenation during OLV has been studied repeatedly. Slinger and Scott showed a direct correlation between increasing CO and improving oxygenation in patients during OLV [68]. Similarly, CO augmented by a small dose of dobutamine (5 μg/kg/min) has been shown to improve arterial oxygenation and decrease shunt fraction [69, 70]. However, larger doses of dobutamine have been shown to adversely affect arterial oxygenation in a porcine model of OLV. Russell and James increased CO to supranormal levels (two to three times normal) with dopamine, dobutamine, adrenaline or isoproterenol [71, 72]. They demonstrated that while high CO increases mixed venous oxygenation, this benefit is overridden by an increase in shunt fraction, resulting in impaired arterial oxygenation. The shunt fraction is likely increased due to weakened HPV in the face of increases in pulmonary arterial pressure [28, 73]. Animal studies have similarly shown that high doses (20–25 μg/kg/min) of dopamine and dobutamine inhibit HPV response in dogs with left lower lobe hypoxia [74] and one-lung atelectasis [53]. At low CO, oxygenation will therefore be impaired secondary to a low mixed venous oxygen saturation, despite a relatively low shunt fraction. At supranormal CO, on the other hand, oxygenation will be impaired due to an increased shunt fraction, despite the high mixed venous saturation (Fig. 5.6). This interplay bears some resemblance to the opposing effects of alveolar and parenchymal vascular resistance on PVR (Fig. 4.5). Maintenance or restoration of "normal" CO is therefore important for oxygenation during OLV. The availability of noninvasive monitoring devices makes CO data more readily available and allows for appropriate titration of inotropes when required.

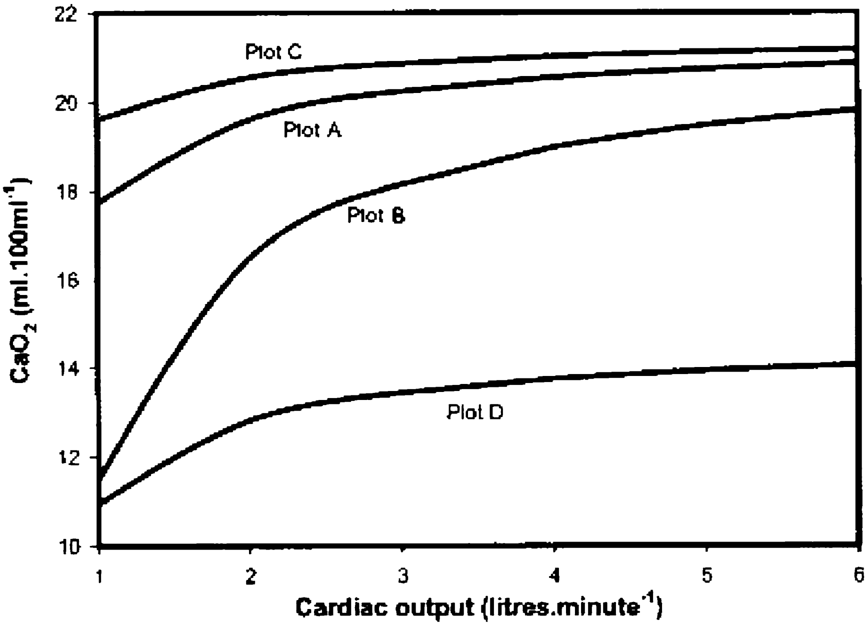

Fig. 5.5. The influence of cardiac output on arterial oxygen content (CaO_2. Plot A: hemoglobin concentration 15 g/dL, oxygen consumption (VO_2) 150 mL/min, shunt fraction (Q_s/Q_t) 0.2). Plot B: hemoglobin concentration 15 g/dL, VO_2 150 mL/min, Q_s/Q_t 0.4. Plot C: hemoglobin concentration 15 g/dL, VO_2 75 mL/min, Q_s/Q_t 0.2. Plot D: hemoglobin concentration 10 g/dL, VO_2 150 mL/min, Q_s/Q_t 0.2 (from Levin et al. [67], with permission).

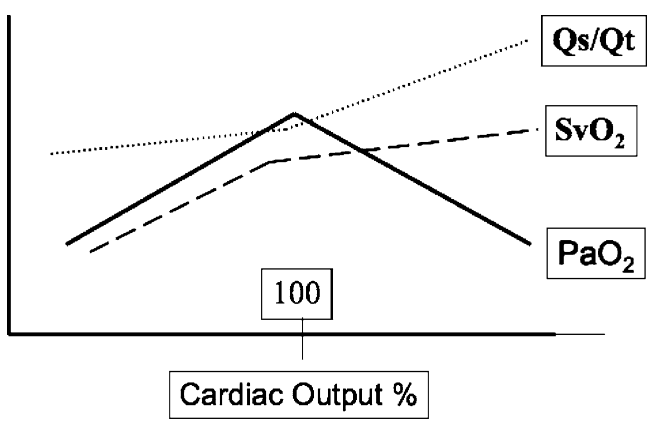

Fig. 5.6. Effect of cardiac output on P_aO_2 during OLV (on the basis of the data from Slinger and Scott [68] and Russell and James [71]).

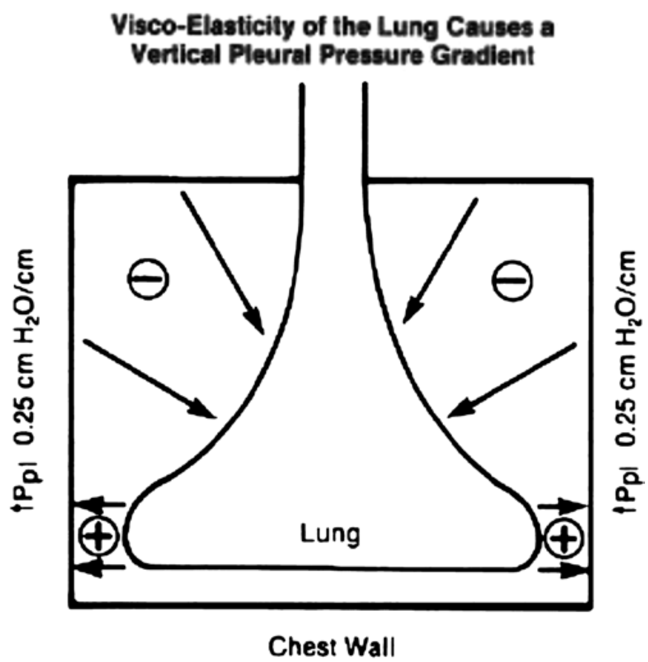

Fig. 5.7. Schematic diagram of the lung within the chest wall illustrating the tendency of the lung to assume a globular shape because of the lung's viscoelastic nature. The tendency of the top of the lung to collapse inward creates a relatively more negative pressure at the top of the lung. Thus, pleural pressure increases by 0.25 cm H_2O per centimeter of lung dependency (modified from Benumof [75]. © Elsevier 1983).

Ventilation

Similar to pulmonary perfusion, gravitational forces also affect the distribution of ventilation throughout the lung. The negative pressure of the visceral–parietal pleural interface forces the lung to maintain the shape of the hemithorax. Disruption of that interface (as in a pneumothorax) results in recoil deflation of the lung, which, analogous to a fluid filled balloon, will take on a more globular shape (Fig. 5.7). The same forces are active even with an intact pleural interface and affect the cumulative transpulmonary pressure. The inherent tendency of the lung to want to collapse away from the upper chest-wall adds to the negative pleural pressure at the top of the lung, while the tendency of the dependent lung to want to push outward reduces negative pleural pressure at the bottom of the lung. The resulting vertical pressure gradient accounts for a change of 0.25 cm H_2O per centimeter of vertical distance along the lung. On the basis of a height of 30 cm of the upright lung this corresponds to a change in transpulmonary pressure (P_{pl}) of $30 \times 0.25 = 7.5$ cm H_2O between the top and the bottom of the lung [76]. The distending force (P_A) is the same for all alveoli; however, P_{pl} becomes less negative toward the bottom of the lung. The net effect is that the transalveolar distending pressure ($P_A - P_{pl}$) is higher at the top of the lung, resulting in a larger alveolar volume compared to the bottom of the lung. In fact, this difference in size can be as much as fourfold. While the dependent alveoli are relatively small and compressed, they fall on the steep (compliant) portion of the volume–compliance curve and receive a disproportionately larger amount of the alveolar ventilation. The larger alveoli of the upper lung fall on the flat (noncompliant) portion of the volume–compliance curve and therefore change little during tidal respiration (Fig. 4.3) [77].

Ventilation–Perfusion Matching

Efficient gas exchange hinges on matching of perfusion and ventilation. Both ventilation and perfusion increase progressively from nondependent to dependent areas, but the change in perfusion is more extreme and ranges from zero flow to high flows. As a result, nondependent areas tend to be relatively underperfused ($V/Q >> 1$), whereas the dependent areas are relatively overperfused ($V/Q << 1$) (Fig. 4.6). Postcapillary blood from the under-ventilated, dependent lung zones ($V/Q << 1$), therefore, tends to be relatively hypoxic and slightly hypercapnic. Nondependent lung zones, which are relatively over-ventilated ($V/Q >> 1$), are able to compensate by removing excess CO_2, but due to the flat O_2- hemoglobin curve, they are less capable of increasing oxygen uptake. High *V*/*Q* areas therefore compensate for carbon dioxide, but not for oxygen, exchange. As a result, the alveolar–arterial (*A*–*a*) gradient, in the setting of significant *V*/*Q* mismatch, is large for oxygen and relatively small for carbon dioxide (Fig. 5.8) [78].

OLV provides a significant challenge to *V*/*Q* matching. Once lung isolation has been established, residual oxygen is gradually absorbed from the nonventilated lung until complete absorption atelectasis has occurred. At that point, pulmonary blood flow to the operative lung is entirely wasted perfusion. The resulting right-to-left shunt through the nonventilated lung is in addition to the normal 5% of shunt in the ventilated lung. As blood flow to each lung is roughly equal (right lung 55% of CO, left lung 45% of CO), this mathematically results in a shunt fraction of at least 50%. Observed shunt fractions are fortunately much lower (Fig. 5.4). Both passive and active mechanisms decrease the blood flow through the operative lung. Surgical manipulation and, in the lateral position, gravity passively reduce the blood flow to the nonventilated lung. In addition, HPV actively increases vascular resistance in the nonventilated lung, resulting in a gradual decrease in shunt fraction.

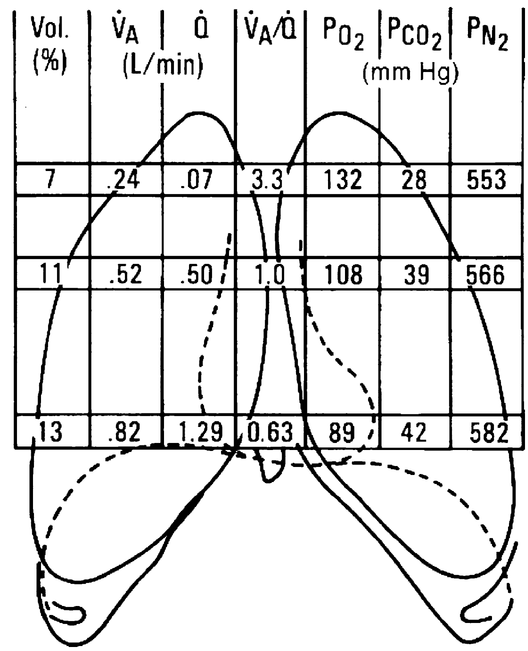

FIG. 5.8. The ventilation–perfusion ratio and the regional composition of alveolar gas. Compared with the top of the lung, the bottom of the lung has a low ventilation–perfusion ratio and is relatively hypoxic and hypercarbic (from West [78], with permission).

V/*Q* Matching in the Lateral Position

Awake

The distribution of alveoli on the compliance curve is maintained when an awake, spontaneously breathing patient assumes the lateral position. Dependent alveoli remain small and compliant, whereas nondependent alveoli stay large and noncompliant. Because of the position change, however, different areas of the lung are now dependent and nondependent. While caudal regions are small and compliant in the upright position, in the lateral position it is the dependent (down) lung, which receives most of the ventilation. Additionally, the cephalad displacement of the dependent diaphragm by abdominal contents results in more effective diaphragmatic muscle contraction. The net result is preferential ventilation of the dependent lung in the lateral position relative to the nondependent lung (Fig. 5.9) [2, 15].

Perfusion is similarly affected in the lateral decubitus position. The gravity dependent distribution of flow is maintained, with a roughly 10% shift of CO to the dependent lung. A dependent right lung will therefore receive 65% of CO, compared to the 55% it receives in the upright or supine position. For a dependent left lung this will result in an increase from the normal 45% of CO towards 55% of CO (Fig. 5.10) [79]. When combined, the lateral position favors the dependent lung in ventilation and perfusion, and *V*/*Q* matching is maintained similar to the upright position.

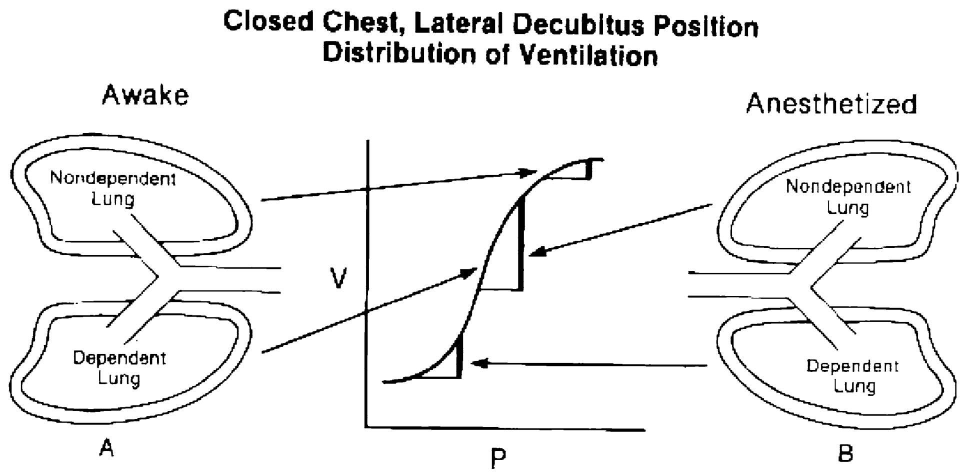

FIG. 5.9. Schematic diagram of a patient in the lateral decubitus position. The change in the distribution of ventilation with the transition from the awake state to the anesthetized state is illustrated (modified from Benumof [2]. © Elsevier 1995).

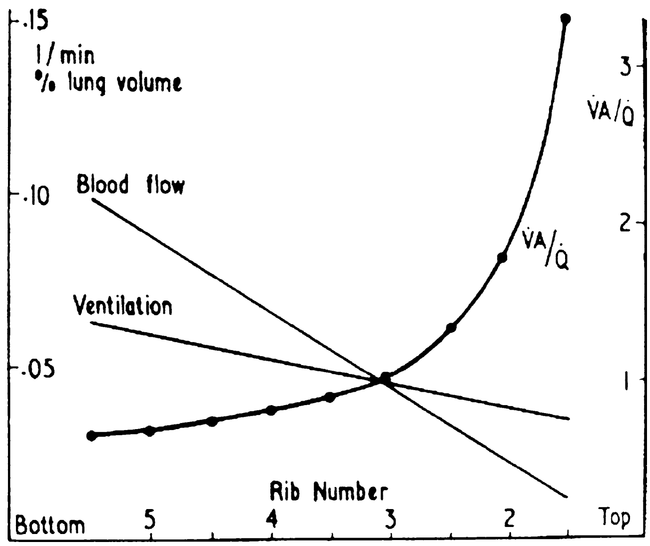

FIG. 5.10. Schematic representation of the effects of gravity on the distribution of pulmonary blood flow in the lateral decubitus position. The vertical gradient in the lateral decubitus position is less than that in the upright position (Fig. 5.2). Consequently there is less zone 1 and more zones 2 and 3 blood flow in the lateral decubitus position compared with the upright position (alveolar pressure (P_A), pulmonary arterial pressure (P_a), pulmonary venous pressure (P_v)) (modified from Benumof [2]. © Elsevier 1995).

Anesthetized

Induction of anesthesia decreases diaphragmatic and inspiratory muscle tone, which results in a 15–20% drop in FRC in both lungs. The change in lung volume alters the relative position of each lung on the compliance curve. The dependent lung drops from the steep portion of the volume–pressure curve, to the flat, noncompliant position. The nondependent lung on the other hand drops from the shallow position of the curve into the steeper portion previously occupied by the dependent lung (Fig. 5.9). As a result, the nondependent lung is now more compliant than the dependent lung and becomes preferentially ventilated [2, 80, 81]. The distribution of perfusion, on the other hand, is not affected by the induction of anesthesia. Consequently, ventilation and perfusion have become uncoupled with the nondependent lung receiving the bulk of ventilation (but little perfusion) and the dependent lung receiving the majority of perfusion (but little ventilation) [2, 15].

Paralyzed/Ventilated

Muscle relaxation, which entirely removes diaphragmatic and inspiratory muscle tone, further alters the distribution of ventilation. Diaphragmatic contraction played a more dominant role due to the favorable, higher resting position in the lateral decubitus (Fig. 5.11). Once paralyzed, static displacement of the relaxed diaphragm by abdominal contents and the gravity force of the mediastinum further restrict the lower lung, resulting in an additional decrease in its compliance (Fig. 6.3). Coupled with the institution of positive pressure ventilation, this further favors nondependent lung ventilation. Pulmonary perfusion is unaffected by muscle relaxation. However, the increase of P_A due to the institution of positive pressure ventilation will increase zone 1 ($P_A > P_{pa}$) and zone 2 territory ($P_A > P_{pv}$). The combination of reduced ventilation of the dependent lung and reduced perfusion of the nondependent lung disrupts *V*/*Q* matching beyond what was seen for the anesthetized, spontaneously breathing patient [2].

Open Chest

Opening of the chest, as well as the resulting loss of negative intrapleural pressure, releases the mediastinal weight onto the dependent lung, further worsening its compliance. The nondependent lung on the other hand is now free to move independent of chest-wall constraints, solely based on parenchymal compliance. Consequently, the lung will collapse, if lung

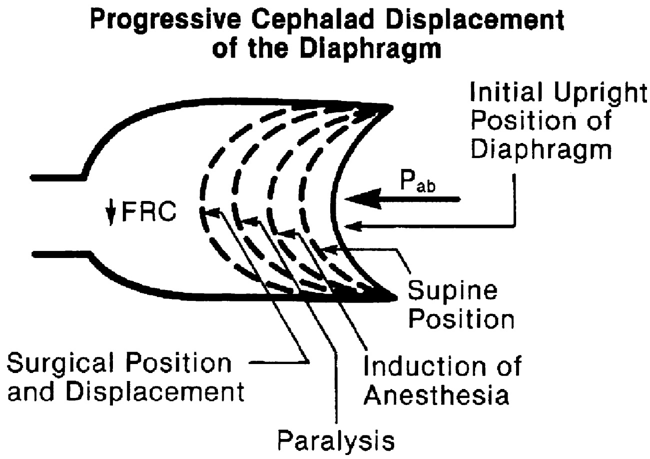

FIG. 5.11. Anesthesia and surgery result in progressive cephalad displacement of the diaphragm. All of, assuming the supine position, induction of anesthesia, paralysis, surgical positioning and retraction act to displace the diaphragm and decrease FRC (modified from Benumof [2]. © Elsevier 1995).

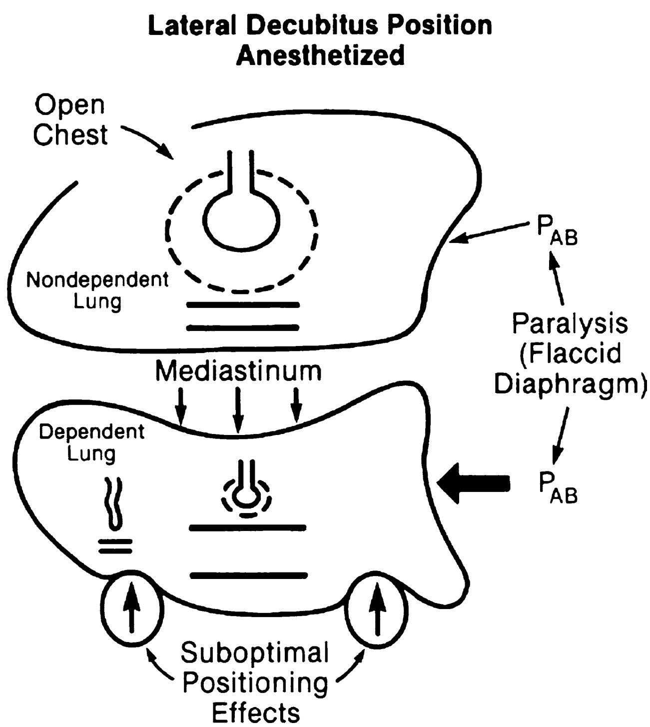

FIG. 5.12. Schematic summary of ventilation–perfusion relationships in the anesthetized patient in the lateral decubitus position. P_{AB} transmitted abdominal pressure (modified from Benumof [2]. © Elsevier 1995).

isolation has been applied, or will be able to herniate through the thoracotomy incision if still ventilated. The distribution of pulmonary blood flow will not be affected by opening the chest unless there is distortion of the mediastinal structures (Fig. 5.12). *V*/*Q* matching will depend on whether lung isolation is being employed. During TLV, opening of the chest will result in a deterioration of *V*/*Q* matching, due to increase in zone 1 ventilation when the nondependent lung is allowed to herniate through the thoracotomy incision. Application of lung isolation, however, will divert all ventilation to the dependent lung, which already receives most of the perfusion, and therefore dramatically improves *V*/*Q* matching.

Most thoracic procedures are accomplished in the anesthetized, paralyzed and mechanically ventilated patient. As we have seen in the preceding sections, induction of anesthesia, lateral decubitus positioning, paralysis and mechanical ventilation result in progressive disruption of the close *V*/*Q* matching that is part of normal physiology. Pulmonary perfusion has remained rather undisturbed, with preferential perfusion of dependent areas. Conversely, ventilation has become progressively diverted to the nondependent lung, as the dependent lung experiences extrinsic compression by mediastinum and abdominal contents. The application of lung isolation forces ventilation back into the dependent lung and re-establishes relative *V*/*Q* matching in the dependent lung, at the expense of true shunt in the nondependent lung [2, 15].

Positions Other Than Lateral

Supine

Although not routine for thoracic surgery, a certain number of OLV cases are being performed in the supine position (e.g., chest-wall resections, sympathectomy, minimally invasive cardiac procedures). Lung compliance changes occur with induction of anesthesia, paralysis and mechanical ventilation, as previously described, however, unlike the lateral decubitus position, now affect each lung equally. Abdominal, and to some degree mediastinal, compression affects each lung. Pulmonary perfusion gradients are maintained in the supine position with preferential perfusion of dependent areas. As gravity affects both lungs equally the percentage of CO perfusing each lung is unaffected. *V*/*Q* matching is disturbed, with dependent areas receiving more perfusion, but less ventilation. Because of the minimal vertical distance from anterior to posterior compared to the lateral position, this disruption is relatively minimal in the supine position. However, initiation of OLV in the supine position is less well tolerated than in the lateral position. Because of the lack of gravity redistribution of blood flow, the shunt through the nonventilated lung is substantially larger than in the lateral decubitus position, resulting in worse oxygenation [31].

Prone

OLV in the prone position is rare; however, isolated reports of lung resection and minimally invasive esophagectomy in the prone position have been published [82–84]. The effects of prone positioning during TLV have been extensively investigated [85]. In contrast to the supine position, *V*/*Q* matching and FRC are better maintained, with secondary marked improvement in P_aO_2 values. Lung compliance is improved, in part due to the lack of compression of lung tissue by mediastinal structures [86]. The prone position lacks gravity redistribution of pulmonary blood flow similar to the supine position. The shunt fraction and oxygenation during OLV should therefore be comparable or better than the supine position, but worse than for the lateral position.

Summary

OLV is a well-established anesthetic technique, and it is increasingly being used to improve surgical exposure for a myriad of pulmonary and nonpulmonary intrathoracic procedures. Although well tolerated in the majority of patients, lung compliance and oxygenation are significantly impaired and may complicate the care of some patients. A thorough knowledge of pulmonary physiology explains the majority of the intra-operative trespasses that one encounters during OLV and enables appropriate interventions.

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