Position changes and their physiological consequences

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TURNING PATIENTS from side to side every two hours is a common nursing practice for bedridden and critically ill patients in intensive care units. Changing body positions is important to prevent pressure sores, to promote ventilation and perfusion in the lungs, to prevent muscle contractures, and to prevent hemostasis.

Changes in body position have different effects on ventilation and perfusion distribution in normal subjects and in patients with diseased lungs. There are a number of approaches that the nurse can use in doing a systematic assessment of ventilation and perfusion. There are also many areas in which future nursing research is needed to develop a full picture of the effects of diseases and treatments on pulmonary function.

NORMAL PHYSIOLOGY

The primary function of the lung is gas exchange. Gas exchange occurs across an

air-blood barrier called the alveolar capillary membrane by a process of passive diffusion. Gas exchange has two components: ventilation (air) and perfusion (blood). Gas exchange includes the volume and distribution of the gas (air) within the lung, the volume and distribution of blood flow in the pulmonary circulation, and the diffusion of oxygen (O_2) and carbon dioxide (CO_2) across the alveolar capillary membrane.[1,2]

Ventilation

In the normal lung, air is inhaled through the nose or mouth and is passed through the conducting airways, the bronchi, and the bronchioles to the terminal respiratory unit. The volume of inhaled air can be divided into two parts, the anatomical dead space volume and the alveolar gas volume. The anatomical dead space volume includes the air in the conducting airways, the bronchi, and the bronchioles. This air does not participate in the gas exchange process. The alveolar gas volume is the air that is involved in the exchange of O_2 and CO_2 as the blood passes through the capillaries. In general, the alveolar gas volume is composed of two functional units: tidal volume (Vt) and functional residual capacity (FRC). Tidal volume is defined as the volume of gas inspired or expired during a normal respiratory cycle. Functional residual capacity is the volume of gas remaining in the lung following a normal expiration. In a normal person, Vt is approximately 500 ml and FRC is approximately 2400 ml.

The estimated volume of air remaining in the lung following a normal expiration is approximately five times greater than the amount of fresh alveolar air inhaled by a normal breath (Vt). Thus, Vt can be considered as the dilutional volume; that is, it is the amount of fresh air that is inhaled to dilute the FRC. Many breaths are required to exchange the bulk of the alveolar gas volume. It has been estimated that with normal alveolar ventilation, approximately half of the alveolar gas volume can be exchanged in five breaths.[3] Thus, frequency of ventilation is a major determinate of alveolar gas exchange.

Distribution of ventilation depends on distensibility of the lung and chest wall (compliance), body position, and airway resistance. Distensibility of the lung is largely determined by regional differences in intrapleural pressure and chest muscle work.[4,5] Intrapleural pressure varies from the apex to the base of the lungs. Using a radioactive gas, West studied ventilation distribution and lung volumes. He demonstrated that in the upright position, the base is better ventilated than the apex because alveoli at the base can increase their volume more than alveoli at the apex.[5]

Changes in body position affect ventilation distribution. In the upright position, ventilation distribution is related to regional differences in intrapleural pressure, which is a function of gravity and the weight of the lung. In normal, healthy subjects in the supine, prone, and lateral decubitus positions, the intrapleural pressure gradient between the apex and the base is less, and ventilation becomes more uniform but still better in the base.[6]

Airway resistance also influences ventilation distribution. Airway resistance is determined by airway anatomy, chest wall, and lung tissue recoil properties. Large

bronchioles fill alveoli earlier and quicker during inspiration than small bronchioles. Similarly, on expiration, large alveoli empty sooner and quicker than alveoli connected to small bronchi/bronchioles.[7]

Perfusion

Distribution of perfusion to the lungs is affected by the volume of cardiac output, body position, and pulmonary vascular resistance (PVR).[5,8-10] Almost the entire cardiac output normally perfuses the lung vasculature; thus, a change in the volume of cardiac output affects lung perfusion.[3]

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Body position also affects perfusion to the lung. In the standing, normal subject, blood flow is less in the apex than in the base because of hydrostatic pressure differences. Hydrostatic pressure gradients largely disappear when the subject is placed in the supine position, and blood flow becomes more even.[3] In the prone position, perfusion becomes more evenly distributed in the lung, but the anterior portion becomes the dependent lung, which is affected most by gravity. In the right or left lateral decubitus position, perfusion is slightly greater in the base of the lung dependent on gravity.[6]

Pulmonary vascular resistance also regulates perfusion. PVR is determined by passive or active processes. Passive processes involve alterations in the caliber of the pulmonary blood vessel resulting from changes in mechanical or hemodynamic events in the systemic circulation. Passive processes include changes in pulmonary arterial pressure, left atrial pressure, pulmonary blood volume, and whole blood viscosity. Active processes that affect PVR are changes in neural stimuli (such as sympathetic and parasympathetic stimulation), humoral stimuli (presence or absence of catecholamines, angiotension, acetycholine, and bradykinin), or chemical stimuli (alveolar hypoxia, alveolar hypercarbia, acidemia).[2]

Gas exchange occurs at the alveolar capillary membrane. It involves oxygenated gas on the alveolar side and mixed venous blood containing CO_2 on the vascular side. To accomplish ideal gas exchange, ventilated alveoli are equally ventilated and perfused. However, in the lung there are three types of lung units: ideal (unit 1); alveoli not adequately ventilated but adequately perfused (unit 2); and alveoli adequately ventilated but inadequately perfused (unit 3). The ventilation-perfusion (V/Q) ratio in unit 1 is 1. In unit 2, V/Q is low or less than 1; in unit 3, V/Q is high or more than 1. Since the lung is normally composed of all three functional types of units, the overall V/Q ratio has been calculated to be approximately 0.8 to 0.9.[5,6] Increased supply of unit 2 results in venous admixture and shunt; an increased supply of unit 3 results in increased physiological dead space.

Assessing ventilation and perfusion

Standard pulmonary function tests and chest x-rays provide little information regarding regional lung ventilation and

perfusion. Distribution of regional lung perfusion can be more accurately studied by the use of intravenous macroaggregated albumin or microspheres labeled with technetium-99m or with iodine-131.[11,12] Regional ventilation is determined through the use of xenon-133, a radioactive gas. Xenon-133 is inhaled and regional ventilation is estimated from the washout of the gas using a scintillation camera.[13]

Imbalances in the V/Q ratio occur in the normal lung. Imbalances in V/Q result in an impairment of gas exchange.[14] Basically, there are 12 elements that are clinically available to assess V/Q imbalances and thus, gas exchange. They include (1) respiratory frequency, (2) breath sounds, (3) lung volumes, (4) lung compliance, (5) blood gases, (6) alterations in mental status, (7) heart rate, (8) cardiac output, (9) pulmonary vascular resistance, (10) arrythmias, (11) mechanical assisted ventilation, and (12) changes in body positions. These 12 assessment variables and their relationship to V/Q are summarized in Table 1.

These 12 elements, however, do not discriminate among the cause(s) of the V/Q impairment. To distinguish regional lung units with a low ventilation-perfusion ratio from those with other gas exchange problems such as right-to-left shunt, 100% O_2 can be admininstered to patients at the bedside. When 100% O_2 is given to patients with low V/Q ratio units, the blood perfusing the unit should equilibrate with a higher partial pressure of alveolar oxygen level (PAO_2) and should yield a higher systemic partial pressure of arterial oxygen level (PaO_2) than when the patient breathes a lower O_2 concentration. Thus, the 100% O_2 appears to "correct" the hypoxia of blood that perfuses lung units with a low V/Q ratio, if ventilation to these units is adequate (nonobstructive). However, if obstruction to ventilation is present, or if blood from the pulmonary artery mixes with the pulmonary venous blood (zero V/Q), the PAO_2 will be high, but the PaO_2 will remain low despite the administration of 100% O_2. This is a pulmonary arteriovenous shunt.

If a cardiac right-to-left shunt is present, the administration of 100% O_2 will also yield an elevation in the PAO_2; however, the hypoxia is not "corrected" because mixed venous blood mixes with arterial blood in the heart. This lowers the PaO_2 aerating the lung unit, thus causing a large difference between alveolar (A) and arterial (a) oxygen levels ($P(A\text{-}a)O_2$ difference). Therefore, if the difference in the $P(A\text{-}a)O_2$ remains high even after a properly conducted trial use of 100% O_2, one cannot differentiate a zero V/Q (pulmonary shunt) from a true right-to-left cardiac shunt. The degree of difference between the PaO_2 value obtained on 100% O_2 and PaO_2 value on room air (or on a lower O_2 concentration can be used to determine the proportion of the cardiac output that is being shunted. Approximately 1% shunt exists for each 15 mm Hg difference from the ideal PAO_2.[2]

A V/Q inequality also produces differences in arterial (a) and alveolar (A) carbon dioxide levels $P(a\text{-}A)CO_2$. The difference, however, is not as marked as with the $P(A\text{-}a)O_2$ difference.[15] This is partly due to the dissociation properties of CO_2 and to the small difference that exists between mixed venous and arterial CO_2. Differences in $P(a\text{-}A)CO_2$ usually develop because of a perfusion distribution abnormality. Thus, high V/Q ratios and widened $P(a\text{-}A)CO_2$

Table 1. Assessment variables and their relationship to ventilation/perfusion distribution

Variables	Relationship to ventilation/perfusion
Ventilatory	
Respiratory frequency	Determines amount of ventilation (tidal volume) Determines rate of alveolar gas filling Determines composition of alveolar gas
Breath sounds	Indicates alterations in alveolar ventilation Indicates changes in airway resistance
Lung volumes	
Tidal volume	Affects FRC Affects composition of alveolar gas Affects blood pH
Functional residual volume	Affects amount of Vt Affects composition of alveolar gas
Lung compliance	Affects regional lung volumes Affects airway distensibility Affects lung tissue recoil properties Affects rate and volume of alveoli filling
Blood gases	
PaO_2	Determines the overall gas exchange
$PaCO_2$	Determines the overall gas exchange
Alterations in mental status	Determined by the severity of PaO_2 and/or $PaCO_2$
Perfusion	
Heart rate	Regulates pulmonary circulation volume
Cardiac output	Determines pulmonary blood volume
Pulmonary vascular resistance	Regulates blood distribution through vasoconstriction and dilatation mechanism Conversely related to cardiac output
Arrhythmias	Indicates impaired gas exchange
Extraneous	
Mechanical assisted ventilation	
IPPB	Affects intrapleural pressure Affects cardiac output
PEEP	Affects intra-alveolar pressures Affects cardiac output
Changes in body position	Affects respiratory rate Affects lung volumes (Vt, RV, FRC, TLC) Affects amount of physiological dead space

gradients are hallmarks of increased physiological dead space.

The behavior of nitrogen (N_2) differs from that of $P(a\text{-}A)CO_2$ and $P(A\text{-}a)O_2$ in several ways: Nitrogen is not absorbed or excreted by the lung, thus there is no net gas exchange, and no amount of venous shunting in the lung can contribute to a $P(a\text{-}A)N_2$ difference. Therefore, any difference in $P(a\text{-}A)N_2$ must be the result of a low V/Q ratio. For this reason, nitrogen is the gas of choice for distinguishing low V/Q from zero V/Q.[16]

Changes in body positions

There appears to be a degree of natural imbalance between blood flow and ventilation in the lung. Factors that influence or cause this imbalance have been studied. In 1955, Blair and Hickam studied the relationship of blood flow and ventilation to body positions and lung volumes. In general, they found that changes in body position from sitting to standing to supine significantly affected the rate of ventilation as well as the amount of lung volume. They found that the residual volume (RV) (the volume of gas remaining in the lung at the end of maximal expiration) remained relatively stable in the sitting and supine positions but was significantly larger in the standing position. In contrast, the functional residual capacity (FRC) varied drastically in the three positions. Mean FRC (in liters) ranged from 3.3 in the sitting to 3.79 in the standing to 2.69 in the recumbent positions. The total lung capacity (TLC) (the volume of gas in the lungs after a maximal inspiration) varied from 6.36 to 6.77 to 6.22 in the sitting, standing, and supine positions, respectively. In summary, they found that changes in body position significantly affected the rate of ventilation as well as the amount of lung volume that was ventilated at the slower rates. Therefore, distribution of blood flow and distribution of ventilation varied at different lung volumes and at different ventilatory rates.[7]

Blood flow and ventilation distribution were also examined in relation to the volume of physiological dead space in both the supine and upright positions. These studies concluded that when a subject was in the upright position, there was a large dead space and decreased alveoli perfusion. This leads to the conclusion that "the postural change caused an important alteration in ventilation-perfusion relationships in the lungs."[17(p339)]

In 1964, Bryan et al studied regional lung distribution of ventilation and perfusion in normal, sitting subjects. They found the base of the lung had nearly two and a half times the ventilation and five times the blood flow of the upper lung area. As a result, the ventilation-to-perfusion ratio was higher in the upper than the lower lung area.[18] Similar findings were also reported by Hughes et al, who also studied the distribution of pulmonary blood flow in relation to various subdivisions of lung volumes in normal subjects. They found that blood flow to the base of the lung was greatest following a maximal inspiration (ie, TLC) than following either a normal expiration (FRC) or a maximal expiration (RV).

Effects of intermittent positive pressure

The effect of intermittent positive pressure breathing (IPPB) on V/Q in different positions has been studied. Chevrolet et al

studied V/Q distribution using xenon-133 in five normal subjects in the right lateral decubitus position, during normal ventilation and during IPPB. Their findings revealed that Vt, respiratory frequency, and flow rates were similar during IPPB and normal breathing. However, there was a measurable variation in the V/Q ratio between the nondependent and the dependent lung regions. In the right lateral position, the V/Q ratio of the nondependent lung region to the dependent lung region changed from 1.09 ± 0.18 during normal breathing as compared to a ratio of 1.52 ± 0.14 during IPPB. Perfusion distribution remained unchanged. These data indicate that the dependent lung regions are underventilated, whereas the nondependent lung regions are overventilated; thus the V/Q ratio becomes less homogeneous during IPPB. This result was attributed mainly to the effect of gravity on chest wall distensibility and chest muscles.[19]

In normal subjects in the supine position, IPPB has been found to significantly alter ventilation-perfusion distributions in the base of the lung at normal Vt. During IPPB, ventilation and perfusion are lower in the basilar lung regions than in the upper regions. This effect is the result of diminished respiratory muscle contraction in the supine position.[20]

These studies raise important issues regarding the clinical use of IPPB on patients with impaired gas exchange. The prolonged use of IPPB on these patients can further accentuate impairment of gas exchange by contributing to atelectasis. Caution should be exercised in the routine changing of body positions in patients receiving IPPB because of enhanced V/Q ratios in various positions.

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APPLICATIONS TO PATHOPHYSIOLOGY

The basic principles of ventilation and perfusion described above are significantly altered under conditions of primary cardiac and lung disease.

Mitral stenosis

Mitral stenosis is the most common valvular heart defect. Stenosis of the mitral valve impedes blood flow from the left atrium to the left ventricle in diastole. This causes an increase in left atrial pressure and, in turn, an elevation in the pulmonary artery pressure and in the pulmonary capillary wedge pressure. In severe mitral stenosis, chronic pulmonary hypertension develops and cardiac output is generally fixed.[21]

It has been shown that patients with mitral stenosis have altered pulmonary perfusion distributions as a result of pulmonary hypertension. In normal, sitting subjects, the base is better perfused than the apex, [22-25] whereas in patients with mitral stenosis in the sitting position, the apex is better perfused than the base.[26-28] The reversal of the perfusion gradient has been attributed to the hemodynamic changes caused by this lesion.[28-29]

Dawson et al studied V/Q ratio differences between the left and right lungs in patients with mitral stenosis, using xenon-

133. In the sitting position, the left lung was slightly less ventilated per unit volume than in normal, sitting subjects and the V/Q ratio was significantly less than normal. It was suggested that this condition may be the result of compression or distortion of the left main stem bronchus. There was no significant difference in the ratio of left-to-right lung ventilation in the apex and base when the patient was laced in the supine position.[30]

Ventilation-perfusion studies of normal patients have demonstrated several factors: (1) Vertical perfusion gradients from apex to base are alleviated in the supine and lateral decubitus positions; (2) respiratory rate, the amount of tidal volume, and functional residual capacity vary significantly with body position changes from sitting to standing to supine; and (3) the amount of physiological dead space is larger in the upright than in the supine position. The ventilation-perfusion ratio in patients with severe mitral stenosis is altered in two ways: (1) Pulmonary hypertension causes a reversal of the perfusion distribution, and (2) the left lung may be less ventilated than the right lung.

Therefore, when a patient with severe mitral stenosis is placed in the supine position, it can be surmised that ventilation and perfusion will become more evenly distributed between the apex and the base, and that the tidal volume, functional residual capacity, and the physiological dead space will become smaller. If the patient is turned to the left lateral decubitus position, the vertical perfusion gradient should be alleviated if the cardiac output remains fixed, but perfusion in the dependent lung (left lung) will be greater than in the nondependent (right) lung. Ventilation can be further compromised if the left main stem bronchus is compressed, causing a wider $P(A\text{-}a)O_2$ difference and an increase in the respiratory and heart rates. Thus, turning patients with severe mitral stenosis may accentuate V/Q impairment.

By utilizing the 12 elements identified in Table 1, the nurse will be able to systematically assess the patient for any V/Q imbalances. However, at present, there are no case presentations in the literature to indicate that nurses are systematically assessing patients with mitral stenosis or any other disease, before or after changing their positions from standing to sitting to lateral decubitus to supine.

Myocardial infarction

Disturbances in arterial blood gases have been reported in patients following an acute myocardial infarction. Such patients frequently demonstrate hypoxemia and significant $P(A\text{-}a)O_2$ gradients. It has been assumed that these conditions are associated with myocardial damage and pulmonary congestion.[31,32] A follow-up study by McNicol demonstrated that such conditions may persist up to six months after the acute infarction.[31] Because it is a well-known fact that myocardial damage can effect cardiac function and output, and in turn, pulmonary congestion, one can assume that V/Q distributions will be affected in the lung.[33] Therefore, the nurse should be acutely aware that changes in body position affect ventilation and perfusion ratios in acute patients and those who have had myocardial infarctions.

Pulmonary embolism

Diseases that directly affect the pulmonary arterial system will cause alterations in

perfusion; one such problem is pulmonary embolism. A pulmonary embolus can cause partial or complete obstruction of blood flow to the pulmonary vasculature.[25] The result is a low PaO_2. A study conducted on anesthetized dogs in which a large thrombus was released into the pulmonary circulation demonstrated that ventilation and blood flow to the embolized region were impaired, yielding an abnormally low mean V/Q ratio of 0.64 as compared to 1.43 before embolism.[32] This V/Q change accounted for the hypoxia. Other complications such as reduced cardiac output and development of pulmonary edema secondary to pulmonary embolism will have additional effects on V/Q distribution. Therefore, the nurse must be cognizant of the location of the pulmonary embolus and be aware of the effects of turning on furthering ventilation and perfusion impairment.

Chronic obstructive lung disease

Patients with chronic obstructive lung disease (COPD—emphysema, bronchitis, and asthma) have abnormal distribution of ventilation due to abnormal airway obstruction. The significance and pattern of the distribution of ventilation-perfusion ratios increases with increased obstruction. The type of ventilation abnormality may be the result of obstruction due to parenchymal tissue loss, bronchodilatation, and/or bronchoconstriction. Patients with COPD may also have abnormal distributions of perfusion. The type of perfusion abnormality may be due to the compensatory mechanisms secondary to the ventilation distribution defects of systemic release of vasodilators and vasoconstrictors. However, if the distribution of ventilation and perfusion are affected equally, gas exchange may not be affected early in the disease.[34]

Early in a disease such as emphysema, the uneven distribution of gas is compensated by a redistribution of blood flow.[35] When compensation is not complete, the V/Q ratio widens. A later pattern in emphysema shows ventilation of high ventilation-perfusion ratio units that is consistent with an increase in physiological dead space with relatively little venous admixture. This pattern is explained by West as a result of parenchymal destruction of alveolar tissue and capillary bed tissue.[32]

In patients with severe chronic bronchitis that includes bronchial hypersecretion, the V/Q patterns demonstrate high blood flow to nonventilated lung units that may not result in large physiological shunts in these patients. The explanation of high blood flow to low V/Q units has been collateral ventilation; that is, alveoli receive inspired gas through pores of Kohn. Through pores of Kohn, alveoli communicate with each other and allow collateral ventilation to occur.[36,37]

Advanced emphysema and chronic bronchitis may share a common V/Q problem because of a narrowing of intrapulmonary airways that occurs during expiration. In these conditions, low V/Q units occur, and in turn, lead to hypoxia at rest and an increase in PVR.[34]

Altered distributions of ventilation occur secondary to changes in bronchomotor tone. Bronchomotor tone may be affected by systemic factors such as prostaglandins and histamines or locally inhaled factors such as organic dust and other substances.[2] Therefore, patients with bronchial asthma may have abnormal V/Q

ratios. Wagner et al found that in patients with asthma, 25% of the total blood flow went to lung units with low V/Q ratios, but no pulmonary arteriovenous shunt resulted.[38] After bronchodilator therapy, blood flow tends to increase to low V/Q ratio units without enhancing gas exchange effects.[39]

Previous studies have also shown that body posture affects V/Q ratios and gas exchange in patients with COPD.[40-42] Generally, in patients with COPD, $P(A\text{-}a)O_2$ differences do not occur in sitting positions. Thus regions with low V/Q ratios were less prominent sitting than in supine positions.

Interstitial lung disease

The distribution of ventilation-perfusion ratios has been studied in patients with interstitial lung disease at rest and during exercise to allow for the separation of the effects of V/Q imbalance and diffusion impairment on hypoxia. Limited inspiratory capacity, secondary to decreased elastic recoil and the presence of interstitial fibrosis, results in variations in expansion ratios and low V/Q units. The advanced states of the disease also demonstrate vasoconstriction of small pulmonary arteries and increased physiological dead space.[34] After exercise, Wagner et al found that hypoxia resulted from both V/Q inequalities and diffusion impairment.[43] Position changes affecting V/Q ratios have not been systematically assessed in this group of patients.

Acute respiratory failure

Acute respiratory failure resulting from the adult respiratory distress syndrome (ARDS) causes several important alterations in V/Q ratios. Airways narrow and sometimes close; alveoli are filled with edema fluid and intra-alveolar hemorrhage; areas of atelectasis develop; and ventilation imbalances result in areas of no ventilation.[44] The coexistence of physiological shunts and low ventilation-perfusion units have been documented by West.[45] West also demonstrated that during periods of breathing 100% O_2, units with a low V/Q ratio were converted to physiological shunt units.[32]

The ARDS also causes perfusion problems and thus increases in physiological dead space, due to the presence of microthrombi in the pulmonary vasculature.[44] Overall, the patient with acute ventilatory failure has hypoxia resulting from low V/Q units and physiological shunt units.

Patients with ARDS have benefited from the addition of positive end expiratory pressure (PEEP), because it has been demonstrated that the addition of 15 cm H_2O PEEP improves hypoxia by improving the distribution of ventilation and perfusion and decreasing shunting.[46] The addition of PEEP greater than 15 cm H_2O results in a large volume of ventilation going to high V/Q units and an increase in ventilation of nonperfused lung units. Increased physiological dead space may result from a diversion of blood flow away from well-ventilated areas of the lung because of high alveolar pressures.[47] The addition of PEEP greater than 15 cm H_2O may also cause a decrease in cardiac output and thus impair pulmonary perfusion. It may also cause pneumothorax and impair ventilation in general, leading to a paradoxical decrease in gas exchange. This may occur because PEEP at levels greater than

15 cm H_2O increases intra-alveolar pressure and shifts blood flow to low-ventilation and nonventilated lung units, thereby causing an increase in physiological shunts.[48]

Standard care of a patient with acute respiratory failure has been described as including the supine position with periodic turning to the right and left lateral decubitus positions.[49] Studies have documented the effects of this standard of care on V/Q ratios and gas exchange. Piehl and Brown found a PaO_2 increase of 47 mm Hg in five patients with ARDS who were turned 180° from supine to prone positions on a CircOlectric bed. They ascribed the enhanced gas exchange to better ventilation-perfusion matching, as well as improved airway toilet.[50] Zack et al found improved PaO_2 when patients with disease that is predominantly confined to one lung are turned from the supine to the lateral decubitus position, with the diseased lung being uppermost. That is, the diseased lung should not be the dependent lung when turning patients to the lateral decubitus positions. They speculated that the improvement in PaO_2 was due to improved ventilation-perfusion relationships. In patients with bilateral disease, PaO_2 was generally less when they were placed in the left lateral decubitus position. In addition to the left lung being smaller, the heart probably compressed the left lung volume when the patient was on the left side. The result would be to decrease ventilation to the lung and lower V/Q ratios.[51] It was not until Douglas et al studied body position in patients with acute ventilatory failure that tidal volume, inspired O_2, and PEEP were controlled. In their study, PaO_2 increased 69 mm Hg when patients were turned from supine to prone positions initially and increased 35 mm Hg after subsequent turns from supine to prone. The elevation in the PaO_2 has been thought to be due to a substantial increase in the FRC when the subject is turned to the prone position with the weight of the torso being supported by the upper thorax and pelvis.[49]

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DISCUSSION

Studies of regional ventilation-perfusion distributions have also been done in patients with other conditions; however, the eight examples chosen here describe the underlying principles of V/Q distribution affected by primary cardiac and primary lung disease. These examples identified substantive needs for assessing V/Q distributions resulting from changes in body position. Although a limited number of studies were cited, few systematic assessments of V/Q ratios and gas exchange have been done clinically.

Clinical nursing research is needed in the following areas to systematically assess V/Q ratios and gas exchange in patients in sitting, supine, and lateral decubitus positions:

- primary cardiac disease,
- primary lung disease,
- complex ventilatory failure,

- neurological defects,
- orthopedic complications,
- postoperative thoracic and abdominal surgery with splinting,
- diseases that cause increased intrathoracic and intra-abdominal pressures such as ascites,
- anesthesia.

In summary, the physiological basis for nurses to change patients' positions may be related to the distribution of ventilation and perfusion in the lungs. The nurse at the bedside has assessment tools available to evaluate position changes in patients with normal lungs and those with cardiac or lung disease. Future research in nursing needs to identify systematic changes that occur in groups of patients with different diseases when body positions are changed.

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