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THE EFFECT OF BODY POSITION CHANGES ON ARTERIAL OXYGEN SATURATION IN OBESITY AS MEASURED NONINVASIVELY BY PULSE OXIMETRY

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A Thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in Nurse Anesthesia at Virginia Commonwealth University

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Abstract

THE EFFECT OF BODY POSITION CHANGES ON ARTERIAL OXYGEN SATURATION IN OBESITY AS MEASURED BY PULSE OXIMETRY

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This study utilized a quasi-experimental pretest-posttest design that compared the effect of body position changes on arterial oxygen saturation in obesity as measured noninvasively by pulse oximetry. The null hypothesis, that there would be no difference between oxygen saturation values measured by pulse oximetry with position change from sitting to supine in obese subjects as compared to nonobese subjects, was tested. Fourteen obese, American Society of Anesthesiologist (ASA) physical class status II subjects and 14 nonobese ASA physical class status I subjects were included in the sample population. Thirteen of the obese subjects were further classified as morbidly obese. After agreeing to participate, a pulse oximeter monitoring probe was placed on the subjects index finger. Under spontaneous room air ventilation, a pretest sitting arterial oxygen

saturation was measured by a Nelcor® N-100 pulse oximeter (SpO_2). Subjects were then placed supine on a stretcher for 10 minutes. Pulse oximetry values were recorded immediately following position change and then serially for 10 minutes. Macro analysis of the means across all measurements between both groups failed to reject the hypothesis. Repeated measures analysis of covariance (ANCOVA) revealed that the means across both groups for each SpO_2 measurement approached significance ($p = .089$). Further micro analysis of the magnitude of change from the sitting to supine position between groups rejected the hypothesis. The ANCOVA established statistical significance between the mean supine SpO_2 values (adjusted for baseline) across both groups between the immediate supine measurements and the 1 minute supine value ($p = .001$). Continued significance was displayed when analyzing the difference of the SpO_2 values between the mean 1 minute supine and 2nd minute supine measurement ($p = .041$). The paired t-test determined which group had the significant change between measurements. It was demonstrated that the obese group had a statistical significant change in SpO_2 values between the sitting and immediate supine ($p = .007$), immediate supine and 1 minute ($p = .034$), and 1 minute supine and 2nd minute supine ($p = .013$) measurements. The nonobese group had a statistical significant change in SpO_2 value only from the sitting to the immediate supine measurement ($p = .005$).

Chapter One

Introduction

Obese patients present to the anesthesia provider for a variety of surgical procedures. Obesity alone increases an individual's mortality. When weight is increased 20% above average, mortality rises by 20% for men and 10% for women (Bray, 1987).

The supine position is one of the most often used positions for anesthetic delivery. In normal weight subjects, research has shown that the functional residual capacity (FRC) is reduced in the supine position when compared to the upright position (Craig, Wahba, Don, Couture, & Becklake, 1971). A decrease in the FRC below the closing volume provides a setting for venous admixture and hypoxemia.

Obese patients in the supine position are at an increased risk of developing hypoxemia compared to normal weight patients. The excess weight on the obese chest cage contributes to a greater reduction in lung volumes when the obese patient lies supine. A considerable additional reduction in the FRC is found while the obese patient lies

supine. This larger decrease in FRC found in the supine obese patient places this population at a greater risk for the development of hypoxemia (Sharp, Druz, & Kondragunta, 1986).

Early studies of pulmonary gas exchange in obese subjects include analysis of arterial blood gases. Arterial hypoxemia is evident in supine obese subjects (Said, 1960; Sharp, Barrocas, & Chokroverty, 1980; Vaughan & Wise, 1976;). Arterial hypoxemia is of particular significance since it may delay recovery, intensify organ dysfunction, and contribute to morbidity and mortality. The pulse oximeter uses optical plethysmography and spectrophotometry to determine the arterial oxygen saturation values. Recognizing the effects of the supine position on arterial oxygenation can assist anesthesia providers in optimizing ventilatory conditions of the obese patient population preoperatively, intraoperatively, and postoperatively.

Currently the literature lacks data relating the effects of body position changes in the obese patient on arterial saturation measured noninvasively by pulse oximetry. The purpose of this study was to determine whether the decreases in lung volumes predicted in the supine obese patient can be demonstrated by a decrease in oxygen saturation as measured by pulse oximetry.

Statement of Purpose

The purpose of this study is to evaluate the effect of position change from sitting to supine on oxygen saturation values as measured by pulse oximetry in obese subjects.

Statement of the Problem

Is there a significant difference between oxygen saturation values during position change from sitting to supine in obese subjects as compared to subjects within 20% of ideal body weight?

Hypothesis

There is no difference between oxygen saturation values measured by pulse oximetry with position change from sitting to supine in obese subjects as compared to subjects within 20% of ideal body weight.

Variables

Independent. The independent variables include (a) the seated position, and (b) supine position.

Dependent. The dependent variable is arterial oxygen saturation values as measured by pulse oximetry (SpO_2).

Definition of Terms

The operational definitions for the purpose of this study are listed below:

ASA physical status classes I and II. Physical status classificiation of a patient's physical condition independent of a planned surgical procedure as developed by the American Society of Anesthesiologist (ASA).

ASA I is used to identify patients without organic, physiologic, biochemical, or psychiatric disturbance. ASA II describes patients with mild to moderate systemic disturbance that may or may not be related to the reason for surgery (Stoelting & Miller, 1989).

Morbidly obese. Morbidly obese refers to those individuals greater than twice their ideal body weight (200%) as determined by the modified Brocca Index (Klotz, 1986).

Obese. Obese refers to individuals with body weight greater than 20% of ideal body weight as determined by the modified Brocca Index (Klotz, 1986).

Oxygen saturation. Percent of total hemoglobin that is oxyhemoglobin defines oxygen saturation.

Pulse oximetry. Pulse oximetry is the measurement of **percent** oxyhemoglobin by the use of spectrophotometry.

Lung volumes. The amount of air the lungs contain at various points in the ventilatory cycle can be measured. These lung measurements are classified into volumes and capacities as describe above. Volumes are primary measurements and include inspiratory reserve volume (IRV), tidal volume (TV), expiratory reserve volume (ERV), residual volume (RV), vital capacity (VC), and residual volume (RV). Capacities are combinations of the volumes and include inspiratory capacity (IC), functional residual capacity (FRC), and total lung capacity (TLC). Figure 1 describes graphically the relationship between the volumes and capacities (see Figure 1).

Total Lung Capacity (TLC). The total volume of air that can be contained in the lungs.

Vital Capacity (VC). Vital capacity describes the maximum amount of air expired after maximal inspiration.

Residual Volume (RV). Residual volume is the amount of air in the lungs after the end of maximal expiration.

Functional Residual Capacity (FRC). Function residual capacity is the volume of air in the lungs at end-expiration of a normal breath.

Inspiratory Capacity (IC). Inspiratory capacity defines the maximum amount of air inspired from the FRC.

Tidal Volume (TV). Tidal volume describes the amount of air that moves in and out of the lungs with each normal breath.

Inspiratory Reserve Volume (IRV). Inspiratory reserve volume describes the maximum amount of air inhaled in excess of normal tidal volume.

Expiratory Reserve Volume (ERV). Expiratory reserve volume defines the maximum amount of air expired beyond the FRC.

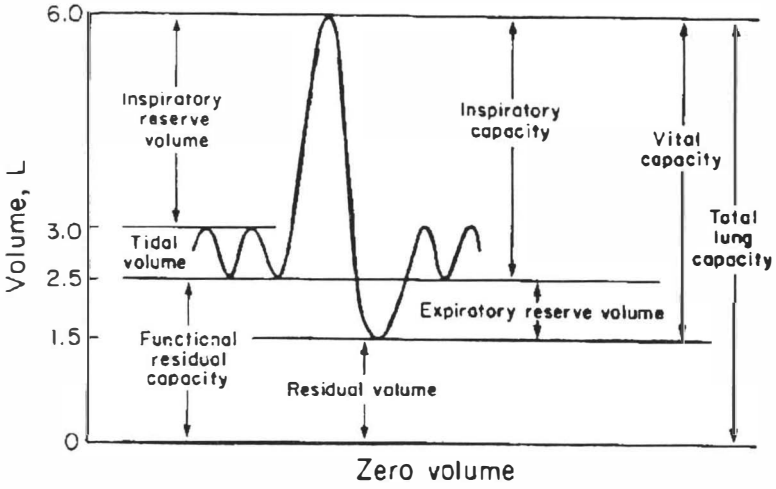

Figure 1. Schematic representation of lung volumes and capacities.

Note. From Anesthesiology: Problem-Oriented Patient Management, (p. 7), by F. F. Yao and J. F. Artusio, Jr., 1988, Philadelphia: J. B. Lippincott.

Sitting position. The sitting position refers to when the subject's body is supported upon the buttocks and thighs, with the torso vertical, and legs dangling over the side of a stretcher or bed.

Supine position. The supine position describes the subject when lying recumbent on his back with the head of bed at zero degrees elevation.

Position change. Position change defines the adjustment of body position from sitting upright to lying supine on a stretcher or bed.

Assumptions

The following assumptions are acknowledged by the investigator of this study:

1. The Nelcor N-100® pulse oximeter is not affected by skin pigmentation, tissue thickness, venous blood, light intensity and ambient light; these considerations lack pulsatile flow and thus are not accounted for by the oximeter.

2. The quality of vascular pulsations is without reduction during the period of data collection.

3. The quantity of dysfunctional hemoglobins (carboxyhemoglobin, methemoglobin) is insignificant.

4. Variance between disposable digit sensors applied among patients does not exist.

5. Pulsatile blood flow to all of the subject's digits is equal in quality.

6. The subjects's hemoglobin level in the light path of the oximeter is adequate to detect pulsatile changes in vascular beds.

7. The Nelcor N-100® oximeter has reliability and validity for saturation values: between 70% - 100% ± 2%, and between 50% - 70% ± 3%.

Limitations

The limitations created by the methodology of the study include:

1. The selection of oxygen saturation level measured by pulse oximetry limits the investigator's ability to detect changes in oxygenation within the flat portion of the oxy-hemoglobin dissociation curve.

2. The inability to differentiate oxygen saturation changes due to position changes from the effect of other variables is not accounted for: alterations in temperature, carbon dioxide measurement, pH, 2,3-diphosphoglycerate ion, dysfunctional hemoglobins, inadequate perfusion, hypovolemia, or undiagnosed respiratory, cardiovascular, or metabolic conditions. However, a sitting baseline SpO_2 was determined for each subject.

Delimitations

The delimitations imposed by the investigator include:

1. The sample is one of convenience: a non-probability sample from preoperative anesthetic evaluations at a single 1,052 bed mid-Atlantic teaching hospital.

2. Only ASA I and II patients are accepted into the study population.

3. Exclusion criteria exists which limits patient eligibility for the study: history of smoking, restrictive pulmonary disease, or cardiovascular disease.

Conceptual Framework

Before analyzing the effects of position changes on oxygen saturation in obese individuals, it is necessary to understand normal respiratory function. The focus of this section is to present the concepts of normal ventilation in the upright and supine position so that obesity oxygenation literature can be understood.

Muscle activity of breathing. The actual process of ventilation involves the movement of gases into and out of the lung by the muscles of respiration. The visceral pleura covers the outer surface of the lungs. It is in contact with the parietal pleura which lines the interior surface of the thoracic cage. The lungs and thorax are held firmly together by intermolecular forces between the two pleural surfaces. These forces are represented by intrapleural

pressure measurements. This intrapleural pressure depicts the tendency for the lungs and chest wall to pull apart. It reflects the strength of the collapsing elastic lung tissue or the strength of the expanding chest wall force.

Contraction of the diaphragm and intercostal muscles produces inspiration and causes the chest to increase its anterior-posterior, superior-inferior, and lateral dimensions. With this increased capacity of the thoracic cavity, the pleural pressure becomes more subatmospheric. This pressure change is transmitted to the alveoli. A concentration gradient between the atmosphere and the alveoli of the lungs permits atmospheric air to enter the pulmonary system. Incoming atmospheric air ceases to flow when the intra-alveolar pressure equilibrates with atmospheric pressure.

Expiration begins when the elastic recoil force of the lung equals the opposing force of the chest wall and inspiratory muscles. The pressure within the lungs increases above atmospheric. Collectively, this pressure gradient and increased elastic recoil force of the lungs decreases the air volume in the lung. Exhaled air is released through the upper airway passages (Berne & Levy, 1988).

Elastic forces of the lung. The lungs innately tend to collapse due to forces contributed by the elastin/collagen

fibers, air-surface forces, and surfactant. The physiochemical properties of the lung tissues in the alveolar wall allow for the expansion and contraction of the alveoli. Elastin fibers allow for expansion; whereas, collagen fibers resist stretch and favor recoil. The thin liquid film lining the alveoli (surfactant) contributes to an air-liquid interface in the alveoli. These two surfaces act together to comprise the elastic recoil of the lung.

The transpulmonary pressure measures the strength of these elastic forces acting on the lung. This pressure is defined as the difference between the alveolar pressure and the pleural pressure. Measurement of these values throughout different lung volumes can be performed through the use of an intra-esophageal catheter. A volume-pressure graph is then created (see Figure 2).

Figure 2 illustrates that a greater change in transpulmonary pressure is needed when the lung volume increases. High lung volumes create a need for higher transpulmonary pressures. Higher pulmonary pressures cause the lung to resist expansion and favor recoil, thus diminishing the amount of air in the lung (Berne & Levy, 1988).

Elastic forces of the chest wall. The inherent elastic property of the chest wall is dependent upon the pressure gradient between the pleural pressure and the body surface. The pleural pressure is affected by the alveolar pressure.

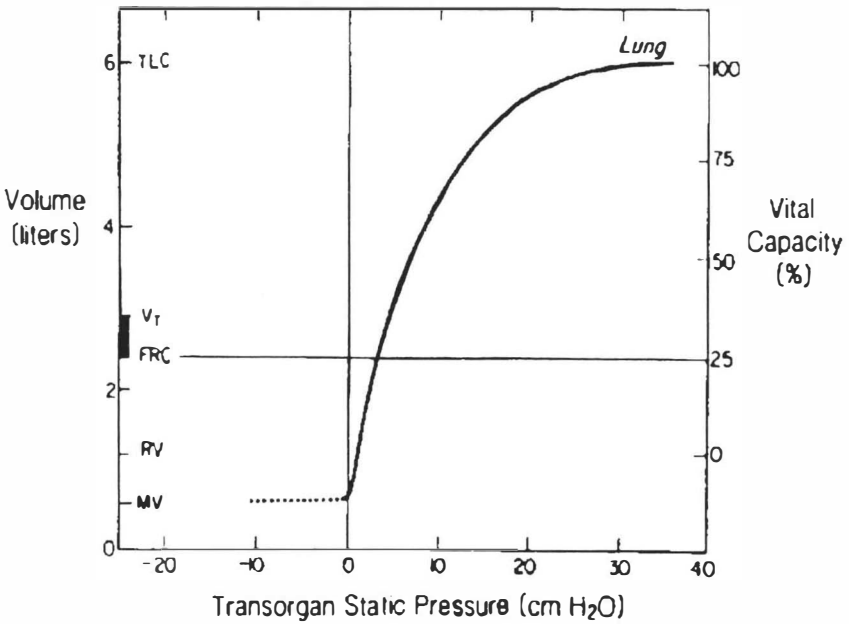

Figure 2. Relationship between lung volume and transpulmonary pressure.

Note. From Basic Respiratory Physiology (p. 64) by N. C. Staub, 1991, New York: Churchill Livingstone.

Thus, the chest wall forces are opposed by the recoil forces of the lung tissues. At resting states (lung volume = FRC), the pressure gradient between these two surfaces is directed outward. This phenomenon assists inspiratory efforts.

The chest cavity pressure at different lung volumes can be measured and plotted to visualize its effect on lung capacity (see Figure 3). As the graph depicts, the chest cavity can expand to about 70% of TLC at zero chest wall pressure when the gradient is absent between the pleura and the body surface. This is the equilibrium point of the chest wall. At 40% of TLC (FRC), the graph shows a negative pressure value. At this point, the chest wall seeks equilibrium to zero and tends to expand. As the volume of the chest cavity increases above the zero pressure point, the chest wall tends to collapse and seeks pressure equilibrium. When the volume of the chest cavity drops below the zero equilibrium point, the chest wall tends to expand (Berne & Levy, 1988).

Elastic properties of the chest-lung system/FRC. The contraction of the respiratory muscles provides the needed force to overcome inherent elastic recoil properties of the lung and chest wall. The FRC is the lung volume during the respiratory cycle where the tendency for the lungs to collapse is balanced by the tendency for the chest wall to expand. This is considered the resting volume of the chest wall/lung system (40% of total lung capacity).

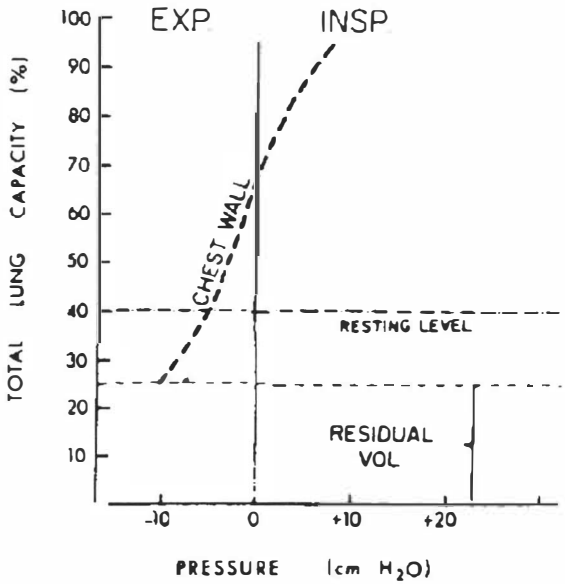

Figure 3. Elastic forces of the chest wall.

Note. Adapted from Respiration in Health and Disease (p. 17), by R. M. Cherniack and L. Cherniack, 1983, Philadelphia: W. B. Saunders.

Elastic forces of the chest wall/lung system. The graphs of transpulmonary pressure and the elastic forces of the chest wall can be combined to obtain the volume-pressure relationship of the chest-wall system; the relaxation curve (see Figure 4).

Figure 4 reveals that at volumes below FRC, the expanding force of the chest wall is stronger than the collapsing force of the lungs; the chest wall/lung system tends to expand toward FRC. At volumes above the FRC, the collapsing force of the lungs is stronger than the expanding force of the chest wall; the system tends to collapse towards FRC (Berne & Levy, 1988).

Compliance. Compliance is described as the distensibility of a structure, the ease with which it can expand. The mathematical expression for compliance can be expressed as the change in volume (dV) per unit change in pressure (dP):

$$\text{Compliance} = \frac{dV}{dP}$$

The concept of compliance is depicted from the slope of a curve. The mathematical derivative of a horizontal line is zero. Compliance values are lower at a point on a curve where the slope of the line is flatter. Conversely, a larger derivative value is obtained when the slope of a curve approaches a more vertical appearance. Compliance of a structure is greater at more upright positions on a curve.

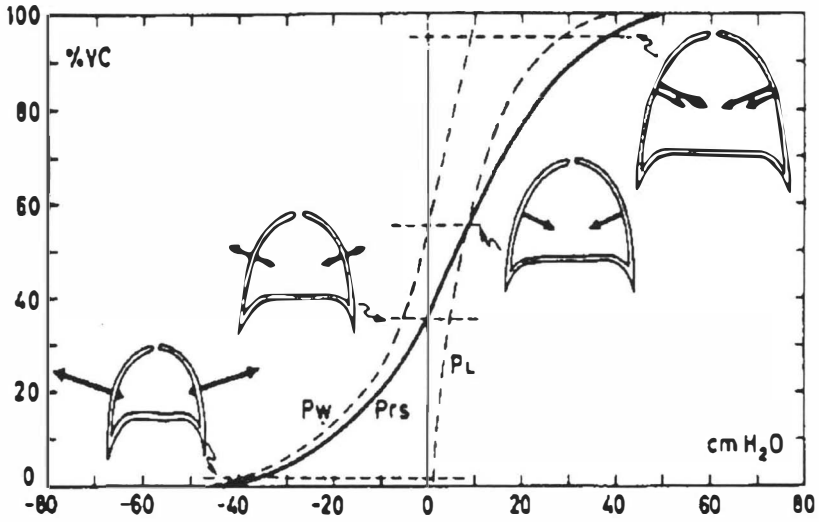

Figure 4. Relaxation curve: Volume-pressure curves of the lung (PL), chest wall (Pw), and total respiratory system (Prs).

Note. From Respiratory Function in Disease (p. 30) by D. V. Bates, 1989, Philadelphia: W. B. Saunders.

A compliance curve of the intact respiratory system is generated by the compliance of the lung tissues together with the compliance of the chest wall. Both lung and chest wall compliance change at different lung volumes and in the presence of pulmonary disease.

As seen by the lung volume-pressure curve, the lung functions with greater compliance (more vertical slope) at lower lung volumes than higher lung volumes (more horizontal slope). Different graphs of lung volume-pressure relationship can be plotted for various disease states and conditions (see Figure 5). The shape curve of the graph changes along with its location on the x-y axis.

Figure 5 describes that certain conditions increase lung compliance by exhibiting a more vertical appearance, have a larger derivative value, and produce a greater volume in the lungs. Situations that decrease lung compliance reveal a more horizontal curve, display a smaller derivative value, and produce a lesser volume of air in the lungs (Berne & Levy, 1988).

The compliance of the chest wall can be observed from the chest wall volume-pressure graph. At low and high volumes on the curve, the compliance is low. This reflects the small slope of the curve found at these volumes. When compliance of the chest wall is low, the thoracic cage is stiffer. At points on the curve where the slope is more vertical in appearance, the chest wall is more

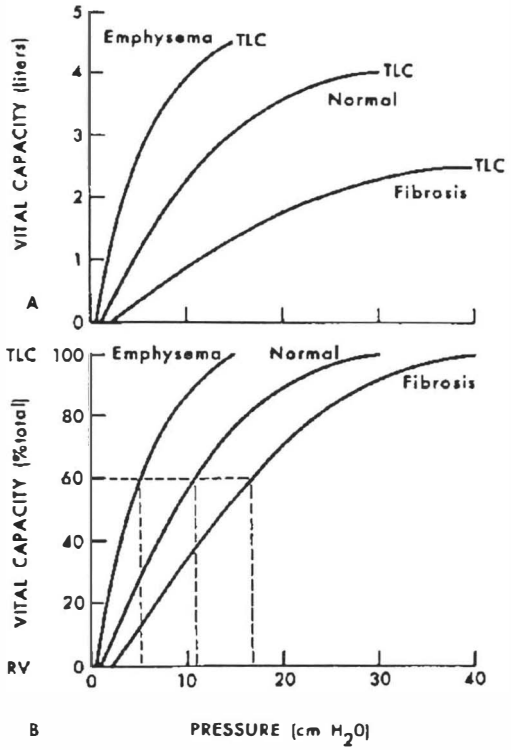

Figure 5. Lung-volume pressure relationships in different lung conditions.

Note. From The Normal Lung: The Basis for Diagnosis and Treatment of Pulmonary Diseases (p. 87) J. F. Murray, 1986, Philadelphia: W. B. Saunders.

compliant and a greater change in volume per unit of pressure occurs. Various physical changes decrease chest wall compliance and cause the slope to flatten (Berne & Levy, 1988).

Postural effects on lung volumes. The effects of posture on respiratory function have been extensively reviewed in the literature. One of the early lung volume and posture studies was conducted by McMichael and McGibbon (1939). Lung volume determinations were carried out in twenty-five subjects. The average postural changes in lung volumes from sitting to supine included: (1) a decrease in FRC of 780 milliliters (ml) of air, (2) decrease in total lung volume of 340 ml, (3) a slight decrease in residual volume of 150 ml, and (4) a decrease in vital capacity of 190 ml. They concluded that the shift in respiratory volumes was due almost entirely by the pressure of the abdominal viscera on the diaphragm. Svanberg (1957) also found a decrease in FRC with change of position from sitting to supine.

Ventilation distribution in the erect position. Radioactive isotopes are utilized in order to assess the distribution of ventilation in various lung regions. A radioactive isotope is inhaled by the subject. When the isotope is absorbed by the pulmonary tissues, a flash of light is generated. Scintillation counters or gamma cameras are placed over the chest as a means of measuring the

topographic distribution of ventilation. The results of multiple studies on the vertical gradient of ventilation in the erect position reveals that inspiration is consistently distributed to lower dependent lung regions (Ball, Stewart, Newsham, & Bates, 1962; Bryan, et al., 1964; Dollery, Hugh-Jones, & Matthews, 1967; Ewan, Jones, Nosil, Obdrzalek, & Hughes, 1978; Rosenzweig, Hughes, & Jones, 1969; West & Dollery, 1960). The results of the studies revealed a mean superior/inferior zone ratio of 0.72 (see Table 1). During normal regional distribution of ventilation, when a healthy individual inspires from the FRC, a greater proportion of inspired gas goes to the alveoli in the dependent lung regions.

Gravity causes an increasing pleural pressure difference from the apex to the base of the lung. The effects of gravity results in a pressure gradient of 0.25 cm H_2O/cm lung height. Figure 6 plots the transpulmonary pleural pressure gradient at FRC from apical to basilar alveoli. From Figure 6 it is seen at FRC the basilar alveoli rest at about 40% of TLC. The position of basilar alveoli on a steeper portion of the curve allows for a greater increase in volume for a given change in pleural pressure. Apical alveoli rest at 70% of TLC at FRC and have a smaller incremental change in volume for a change in pleural pressure (Bates, 1989).

Table 1

Vertical ventilation gradient in the erect position

Reference	Radio-nuclide	Superior/inferior ratio
West and Dollery (1960)	$C^{15}O_2$	0.72
Ball et al. (1962)	^{133}Xe	0.64/0.72
Dollery et al. (1962)	^{133}Xe	0.82
Bryan et al. (1964)	^{133}Xe	0.62/ 0.69/0.58
Rosenzweig et al. (1969)	^{13}N	0.80
Ewan et al. (1978)	^{13}N	0.73

Note. Pooled data of vertical gradient of ventilation (superior/inferior lung zone ratio) in the erect position when inspiring from FRC.

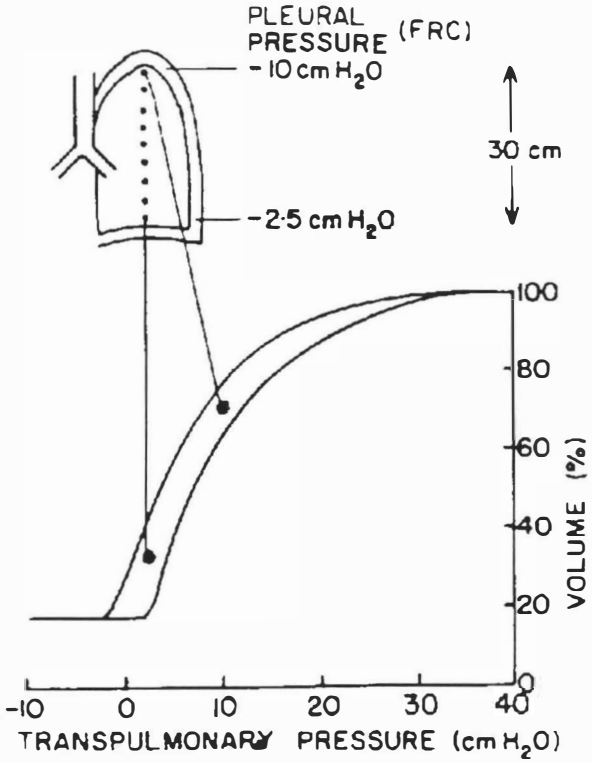

Figure 6. Transpulmonary pressure at FRC.

Note. From Respiratory Function in Disease (p. 36) by D. V. Bates, 1989, Philadelphia: W. B. Saunders.

Ventilation distribution in the supine position. Ventilation distribution in the lung differs as body position changes. The FRC decreases in the supine position. The supine position also produces a vertical ventilation gradient between upper airways (ventral) and lower airways (dorsal). Researchers have studied this ratio with the use of radioisotope breathing and measurement of gamma ray emission (Amis, Jones, & Hughes, 1984; Bynum, Wilson, & Pieerce, 1976; Valind, Rhodes, Clarke, Burke, & Hughes, 1983) (see Table 2). A larger upper/lower gradient of ventilation exists while lying supine.

Table 2.

Vertical ventilation gradient in the supine position

Reference	Radio-nuclide	Superior/Inferior ratio
Bynum et al. (1976)	^{133}Xe	0.35
Valind et al. (1983)	^{19}Ne	0.37
Amis et al. (1984)	$^{81}Kr/^{85}Kr$ ^{85}Kr	0.40 0.50

Note. Pooled data of vertical gradient ventilation (superior/inferior lung zone ratio) in the supine lung in normal subjects inspiring from FRC.

Ventilation along the horizontal axis (craniocaudal) while lying supine initially, was thought to be uniformly distributed (Kaneko, Milic-Emili, Dolovich, Dawson, & Bates, 1966). Further studies revealed that craniocaudal gradients existed. A 1976 study by Bynum, et al. utilized ^{133}Xe to investigate regional lung ventilation. Accumulation of inhaled radioactive isotopes in the lung tissues was measured by four scintillation counters placed over each lung posteriorly from the apex to the base and two counters placed laterally over each lung. Ventilation was reduced significantly ($p < .001$) in basilar lung regions from data obtained by the posterior counters. Lateral counters demonstrated an expected decrease in gradients from vertical superior/inferior measurements but were not shown to be significant. The results suggest the lung bases are the site of ventilatory changes in the supine position (Bynum et al., 1976).

After inhalation of Krypton-81m ($^{81}Kr^{m}$) and Krypton-85m ($^{85}Kr^{m}$), researchers in London placed subjects in front of a gamma camera to measure the effects of posture on ventilation distribution (Amis et al., 1984). The gamma camera generated regional counts of gamma ray concentration present in the lung tissues. From the ratio of the gamma ray counts between the two isotopes inhaled, a regional ventilation per unit alveolar volume (V/Va) measurement was calculated.

Regional ventilation per unit alveolar volume (V/Va) during vertical distribution (ventral/dorsal) in the supine position showed a significant increase of 151%. Lung height decreased when changing position from sitting to supine. With decreasing lung height, the effects of gravity was postulated as less contributory to lung ventilation.

Cranial-caudal horizontal distribution was significantly decreased by 43% in superior lung zones. Inferior lung zones revealed a gradual increase from cranial-caudal distribution to a 3/4 point horizontally, where a decreasing pattern then developed as the diaphragm approached. The study further elaborated on ventilation alterations in the supine position. A decrease in lung expansion was identified in the inferior alveoli. The inferior craniocaudal distribution was not as uniform as once thought. The ventilation per unit of lung volume horizontally increased along the cranial-caudal axis except for a sudden decrease near the diaphragm.

Ventilation/perfusion relationship. During normal states, the matching of ventilation to perfusion results in adequate oxygen delivery to the cardiovascular system. Gravity also effects pulmonary perfusion and produces a greater change in blood flow than in ventilation, even though both ventilation and perfusion are better at the base of the lung than the apex. This effect results in apical

alveoli receiving more ventilation in relation to perfusion, whereas basilar alveoli are better perfused than ventilated.

The conventional relationship between ventilation and perfusion is expressed in terms of the ventilation/perfusion ratio (V/Q). Both quantities are measured in liters per minute. Ventilation is determined by tidal volume per minute and perfusion by blood flow per minute. Total alveolar ventilation is approximately 4 L/min, while pulmonary blood flow is 5 L/min. Thus, the overall normal V/Q ratio is 0.8. Any situation disturbing the ventilation and perfusion match can cause conditions that generate some degree of hypoxemia (Nunn, 1987).

Airway closure/closing capacity. Small airways without structural rigidity rely on volume to assure their patency. As the lung volume begins to decrease towards residual volume, there is a point at which dependent small airways begin to close. The lung volume at which this happens is termed closing capacity. Closing volume is defined as the closing capacity minus the residual volume (Nunn, 1987).

The relationship between closing capacity (CC) and FRC is significant. This association determines a given respiratory unit's role in ventilation. Closing capacity is less than FRC (ERV + RV) in normal healthy adults. This situation ensures opening of airways during normal inspiration. However, when the CC is greater than FRC, air passage into these areas of the lung does not occur during

inspiration; a low V/Q ratio exists. These airways remain closed during inspiration. Closed airways produce atelectatic lung areas and can breed hypoxic conditions (Nunn, 1987). Therefore, if a decrease in the FRC occurs, the closing capacity may be reached. A low V/Q relationship in lung areas can contribute to a hypoxic state.

Effect of supine position on airway closure. The supine position predisposes to conditions that promote hypoxia. Researchers in 1970 examined the effect of body position on airway closure (Leblanc, Ruff, & Milic-Emili, 1970). They employed the helium-dilution method to measure TLC and its subdivisions along with ^{133}Xe to measure closing volume. Their results revealed that closing volume does not change substantially with posture, but there is a decrease in FRC in the supine position. Relating FRC and CV to tidal breathing volumes, the authors showed when FRC exceeded closing volume tidal breathing occurred at low volumes. With low volume breathing, ventilation to affected airways are impaired, causing the potential for a V/Q mismatch and promotion of arterial hypoxemia.

Don, Craig, Wahba, and Couture (1971) examined the effects of posture on the amount of gas trapped in lungs at FRC. Closing volume (CV), FRC, and the volume of trapped gas (VTG) was measured in thirty subjects with the use of single-breath nitrogen and closed circuit helium techniques. The VTG was correlated between the relationship of FRC to

CV. When CV was less than FRC, no gas was trapped; when CV was greater than FRC gas trapping occurred behind closed airways.

Further studies described the relationship between craniocaudal ventilation patterns and various lung volumes. Engel and Prefaut (1981) correlated craniocaudal lung ratios in the supine position at various lung volumes. Measurements were collected by five pairs of scintillation counters with subjects inhaling ^{133}Xe. It was seen when the CC was greater than FRC a large craniocaudal gradient (> 1.5) existed. When lung volumes increased to 40% of vital capacity, a more uniform distribution resulted; while at 60% of vital capacity, the cranial zones became better ventilated than caudal long zones. The researchers suggested that airway closure is implicated as the cause for this ventilation distribution in the supine posture.

Work and oxygen cost of breathing. Work must be executed by the respiratory muscles to overcome the restriction to breathing that is inherent in the mechanical properties of the lung and chest wall. Generally, only a small amount of the maximum force-generating capacity of the respiratory muscles is necessary to overcome the elasticity and resistance of the chest bellows. Oxygen is the required energy source for the respiratory muscles to perform mechanical work. The oxygen cost of breathing is the energy expenditure during breathing.

Work of breathing and oxygen consumption are interrelated. When greater force is needed to overcome increased resistance or elasticity of the chest wall, increased oxygen consumption by respiratory muscles occurs. This added oxygen need might in turn limit the maximum level of ventilation and exercise capacity (Berne & Levy, 1988).

The chemistry of hemoglobin. The hemoglobin molecule has a porphyrin molecular structure found in erythrocytes. A porphyrin results when four pyrrole rings are linked together by methylene bridges (see Figure 7). The porphyrin ring has available four possible binding sites on its nitrogen molecules. Metals often are attracted to these nitrogen binding sites. The metal ferrous ion has six valence bond available for bonding. When the ferrous ion (Fe^{++}) binds to a porphyrin ring, each iron atom is attached by covalent bonds to the four nitrogens on the pyrrole groups. The substance is now referred to as a heme moiety (see Figure 7). Long chains of amino acids link together to form polypeptide chains known as protein molecules. Two specific types of chains are know as alpha and beta chains. The alpha chain contains 141 amino acids and the beta chain has 146 amino acids. When two alpha and two beta protein units combine by salt bridges, a protein globulin results (see Figure 8).

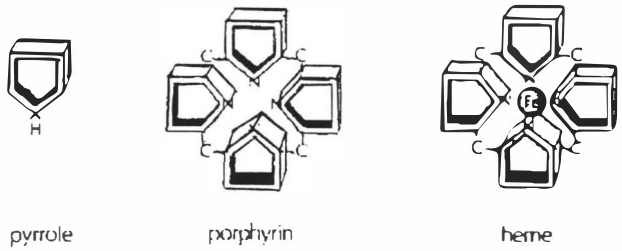

Figure 7. Chemical structure of heme

Note. From Clinical Application of Blood Gases (p. 20) by B. A. Shapiro, R. A. Harrison, and J. R. Walton, 1982, Chicago: Year Book Medical.

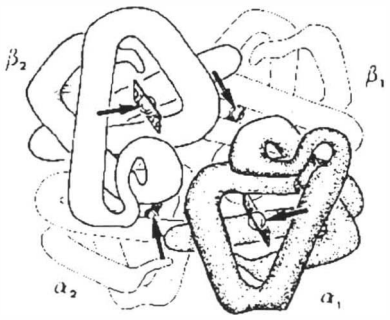

Figure 8. Globular protein structure of hemoglobin.

Note. From The Pathway for Oxygen: Structure and Function in the Mammalian Respiratory system (p. 139), by E. R. Weibel, 1984, Cambridge: Harvard University Press.

The heme molecule attaches itself to a globulin protein molecule by a fifth valence bond between the ferrous ion and a nitrogen atom of a histidine amino acid portion of the protein molecule (see Figure 9). Thus, four of the six available ferrous valence bonds are attached to the nitrogen atoms of the four pyrroles that constitute the porphyrin ring; the fifth is attached to the globin molecule. This leaves available the sixth covalent bond of iron for reversible combination with oxygen. Each molecule of hemoglobin can carry four molecules of oxygen, one on each heme unit (Shapiro, Harrison, & Walton, 1982).

Oxygen transport in the blood. Two different methods of oxygen transport exist in arterial blood: dissolved in plasma and bound to hemoglobin. Oxygen carried in plasma represents approximately 1-2% of all oxygen carried in the blood. The partial pressure of oxygen in the blood represents the pressure exerted by these oxygen molecules in the blood. Nearly 0.003 ml of O_2 is dissolved in 100 ml of blood at an oxygen partial pressure of 1 millimeter of mercury (mmHg).

Oxygen bound to hemoglobin carries 98-99% of the oxygen in the blood. Each gram of hemoglobin when saturated with oxygen can carry 1.34 ml of oxygen (Nelcor, 1988).

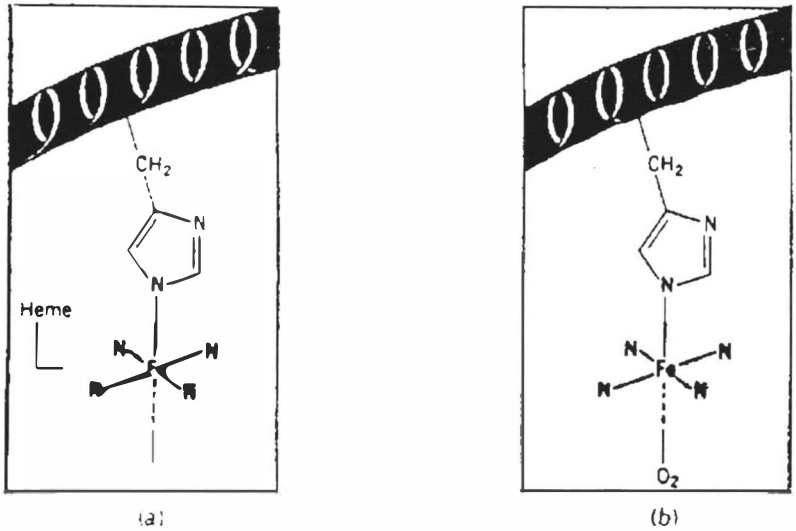

Figure 9. Attachment of heme group to hemoglobin protein. (a) Sixth bonding site of Fe^{++} vacant; (b) O_2 bound to Fe^{++} of heme at sixth bonding site.

Note. From General Chemistry: Principles and Structure (p. 699), by J. E. Brady and G. E. Humiston, 1978, New York: John Wiley & Sons.

Oxygen capacity. The amount of oxygen that hemoglobin carries in arterial blood can be calculated. As stated earlier, the amount of oxygen carried by each gram of saturated hemoglobin is 1.34 ml of oxygen. An adult has a normal hemoglobin value of 15 grams per deciliter. Multiplying these two values together one obtains the maximum volume of oxygen that can be carried by each deciliter of saturated blood:

$$1.34 \text{ ml } O_2/\text{gm Hb} \times 15 \text{ gm Hb/dl} = 20.1 \text{ ml } O_2/\text{dl}$$

This maximum value is expressed as the oxygen carrying capacity of hemoglobin in arterial blood: 20.1 ml O_2/dl. (Nelcor, 1988).

Oxygen content. The blood oxygen content is the sum of the amount of oxygen bound by hemoglobin and the amount of oxygen dissolved in plasma. This can be represented by the following formula:

$$CaO_2 = (Hb \times 1.34 \times SaO_2) + (PaO_2 \times 0.003)$$

Abbreviations include: CaO_2 = oxygen content, Hb = hemoglobin, SaO_2 = Saturation percent of oxygen, PaO_2 = partial pressure of oxygen (Mumme, 1991). The actual amount of oxygen combined with hemoglobin at any time depends on the PaO_2 and the hemoglobin value.

Oxygen saturation. Henry's law states that the amount of gas that can be dissolved in a liquid is proportional to the partial pressure of the gas to which the liquid is exposed (Shapiro, et al., 1982). The amount of O_2 dissolved in plasma is linearly related to the partial pressure. This does not hold true for the amount of O_2 that is combined with hemoglobin (Bates, 1989).

The saturation of hemoglobin is the ratio of the number of hemoglobin molecules already occupied with oxygen, oxyhemoglobin, to the number of hemoglobin molecules available. This expression incorporates the previous two concepts and is depicted by the following:

$$\text{Saturation \%} = \frac{O_2 \text{ content of Hb}}{O_2 \text{ carrying capacity of Hb}}$$

Oxyhemoglobin dissociation curve. A graph can be plotted to depict the hemoglobin oxygen saturation (SO_2) and the partial pressure of O_2 (PO_2) in the blood. The graph results in an S-shaped curve that is referred as the oxyhemoglobin (oxy-hem) dissociation curve (see Figure 10). This graph represents the percentage of available hemoglobin combined with oxygen (% saturation) and its corresponding relationship to blood PO_2 (Bates, 1989).

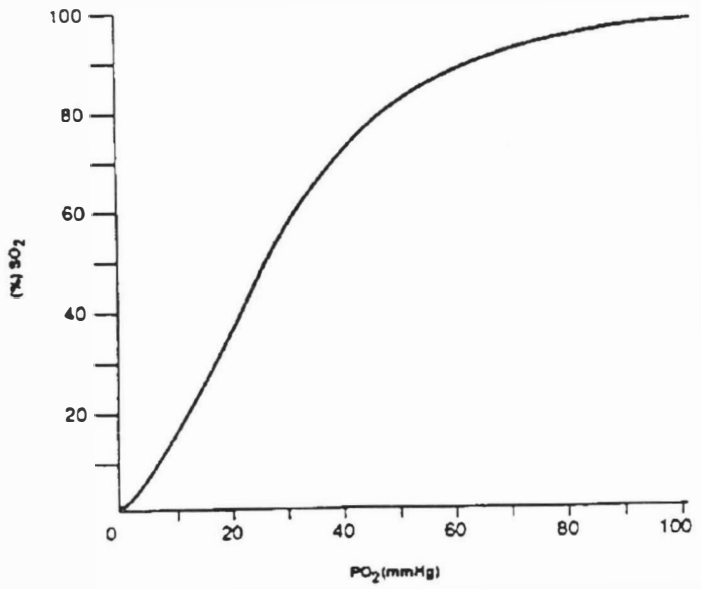

Figure 10. Oxyhemoglobin Dissociation Curve

Note. From Principles of Pulse Oximetry (p. 6) by anonymous, 1988, Nelcor Incorporated.

The binding of oxygen to hemoglobin. Oxygen is drawn into red blood cells by a reaction with hemoglobin molecules. The oxygen affinity of the hemoglobin molecule increases as the partial pressure of oxygen increases. Figure 11 schematically depicts the binding of oxygen to the heme units on the hemoglobin molecule.

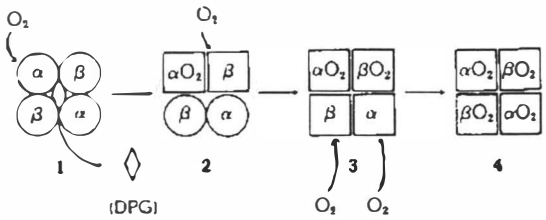

Figure 11. Oxygen binding to heme groups.

Note. From Fundamentals of General, Organic, and Biological Chemistry (p. 570) by J. R. Holum, 1982, New York: John Wiley & Sons.

Structure 1 of Figure 11 depicts a deoxygenated hemoglobin molecule. Each circle is a polypeptide subunit with a heme molecule, but without O_2. The diamond centered within the structure represents an organic ion, 2,3-diphosphoglycerate (DPG). The DPG ion is a normal product

of red blood cell metabolism and plays an important role in regulating the affinity between hemoglobin and oxygen. The DPG molecule is nestled in a cavity between the four subunits. When the first O_2 molecule binds (structure 1 → 2) to the ferrous ion of one of the subunits, the shape of the subunit then changes. This change in shape starts to ease out the DPG molecule. The "lid" is off the hemoglobin molecule now, and another subunit undergoes a change in shape. The absence of DPG increases the affinity of the hemoglobin for oxygen.

The second O_2 molecule can now approach more easily than the first (structure 2→3). When the second molecule attaches to the second heme, the remaining two subunits undergo changes in their framework that allow the last two O_2 molecules to bond more easily (structure 3→4). The first molecule of O_2 breaks open a small molecular "dam" that allows the second molecule to approach more easily and widen the "break." The last two O_2 molecules then "flood" in. This chain of reactions thus ensures almost 100% of the hemoglobin molecules quickly become fully saturated with oxygen as they move through the lungs (Holum, 1982).

Significance of the oxyhemoglobin dissociation curve. The sigmoid shape of the oxyhemoglobin dissociation curve is of significance. At normal PO_2 tensions of 75-100 mm Hg, the saturation of hemoglobin is about 95%. When oxygen tensions are high in the blood, additional loading of oxygen

does not significantly increase since most of the hemoglobin molecules are already saturated. The steeper lower portion of the curve portrays the value of PO_2 at the cellular level. Small changes in decreasing oxygen tension produces greater amount of oxygen release from the hemoglobin molecules.

Technology of pulse oximetry. The pulse oximeter merges the principles of optical plethysmography and spectrophotometry to determine the arterial oxygen saturation values. A spectrophotometer generates light at a known intensity going into a solution and measures the intensity of light leaving the solution. The concentration of a solute in a solution can be measured if the light source has wavelengths that are in agreement with the amount of light the solute absorbs. The principle of this measurement is based on Beer's law:

$$A = DC\epsilon$$

where A = absorption of the light; D = distance the light is transmitted through the solution (path length); C = concentration of the solute (hemoglobin); and ϵ = the extinction coefficient of the solute (a constant for a given solute at a specific wavelength) (Tremper & Barker, 1989).

The Nelcor N-100® pulse oximeter uses two light emitting diodes (LEDs) of red light (660nm) and infrared light (920nm) and a photodetector placed between an arterial vascular bed to measure the intensity of transmitted light across the vascular bed (see Figure 12).

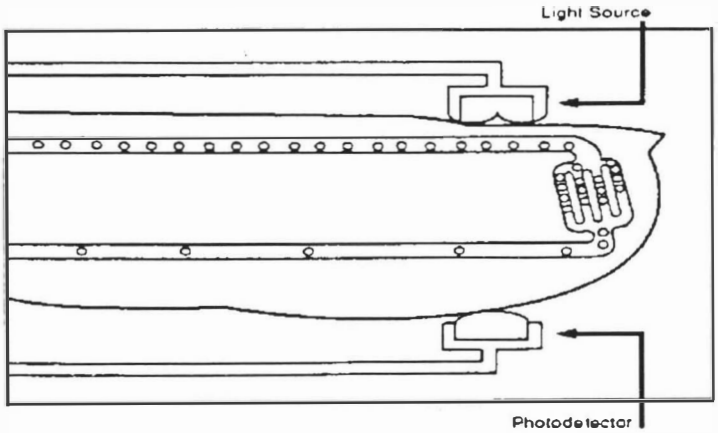

Figure 12. Pulse oximetry digital sensor.

Note. From Principles of Pulse Oximetry (p. 10) by anonymous, 1988, Nelcor Incorporated.

Optical plethysmography uses light absorbance technology to measure the absorbed light in pulsatile blood flow. This technology enables the pulse oximeter to eliminate the effects of nonpulsatile structures, such as tissue and venous blood light absorbance, from measurement. A photodetector, placed opposite the light source measures the intensity of the light transmitted. The pulsatile vascular bed expands and contracts in correspondence with systole and diastole. This movement creates a change in the light path length that is depicted by Beer's law.

Hemoglobin absorbs light at different wavelengths depending upon whether it is bound to oxygen (oxyhemoglobin) or not (reduced hemoglobin). Figure 13 demonstrates the changes in absorption of oxyhemoglobin (O_2Hb) and reduced hemoglobin (RHb) that are observed at the red light (660 NM) and infrared light (920 nm) wavelengths.

The plethysmograph measures the amount of light absorbtion that is recognized by the photodetector. When more absorption occurs, the light wave is transmitted through the medium and a larger amplitude is produced on the plethysmograph.

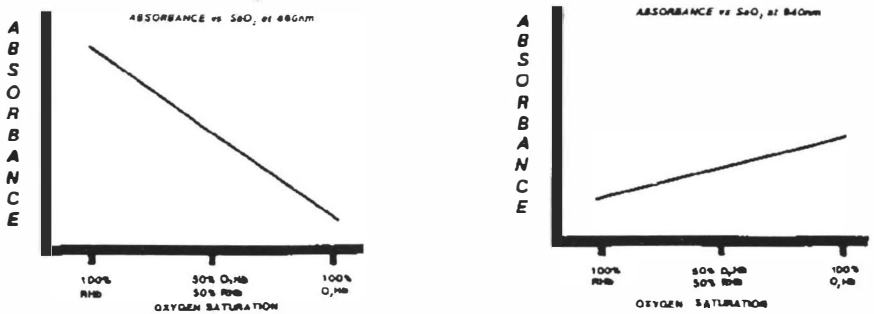

Figure 13. Changes in absorption of oxygenated (O_2HB) and reduced hemoglobin (RHb) as a function of arterial oxygen saturation at (a) 660 nm and (b) 920 nm.

Note. From Pulse oximetry: Technical aspects of machine design (p.141) by J. A. Pologe. In K. K. Tremper and S. J. Barker (Eds.), International Anesthesia Clinics: Advances in Oxygen Monitoring, 1987, Boston: Little Brown.

Figure 14 displays the plethysmographs generated when red light and infrared light are passed through oxyhemoglobin and reduced hemoglobin. When red light (660nm) is passed through reduced hemoglobin (0% saturation), more light is absorbed and a large plethysmograph is produced. When red light is passed through oxyhemoglobin (100% saturation), little light is absorbed and a small amplitude wave is produced. Conversely, infrared light is passed through oxyhemoglobin more than reduced hemoglobin.

The incident light that is transmitted from the LEDs is absorbed by pulsatile (alternating current = AC) and nonpulsatile components (direct current = DC) between the light source and the photodetector. The pulsatile compartment consists of light absorbed by the arterial vascular bed. The nonpulsatile compartment consists of all nonpulsatile absorbers in the tissue; nonpulsatile arterial blood, venous and capillary blood, and all other tissue. Figure 15 schematically illustrates the light absorption through living tissue. Oxygen saturation determination is concerned with the amount of light absorbed by the pulsatile component.

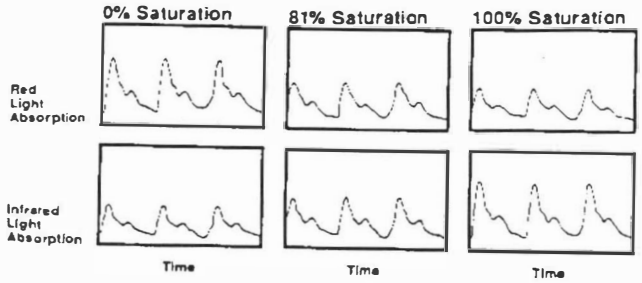

Figure 14. Plethysmograph waves for oxyhemoglobin and deoxyhemoglobin for red light and infrared light absorption. Note. From Principles of Pulse Oximetry (p. 19) by anonymous, 1988, Nelcor Incorporated.

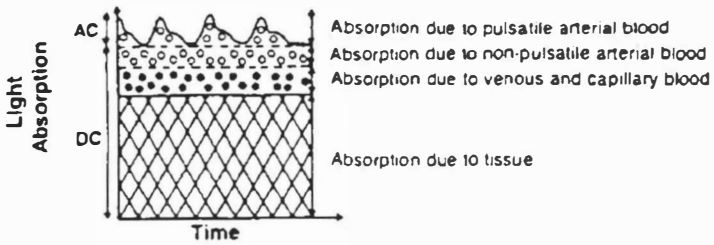

Figure 15. Light absorption through living tissue. Note. From Pulse Oximetry (p. 1010 by K. K. Tremper and S. J. Barker, 1989, Anesthesiology.

A ratio (R) of light transmission between the AC and DC factors of the pulsatile wave provides a mathematical formula that corrects for the DC light measured by the photodetector:

$$R = \frac{AC_{660}/DC_{660}}{AC_{920}/DC_{920}}$$

This ratio provides a corrected value of the light measured by the plethysmograph wave with no interference from light absorbed by nonpulsatile compartments. The ratio obtained is related by an algorithm to SaO_2 measured by a laboratory spectrophotometer in volunteers from arterial blood samples (Nelcor, 1988; Pologe, 1987; Tremper & Barker, 1989).

Summary. The conceptual framework proposes normal arterial oxygenation involves the physiologic concepts of oxygen delivery to the pulmonary system and the binding of oxygen molecules to hemoglobin. Oxygen delivery relies on the interaction of the lungs, chest wall, and respiratory muscles to provide normal distribution of ventilation.

Hypoxemia results when there is a deficiency of oxygen in the blood. Oxygen saturation can be measured noninvasively by the principles of pulse oximetry. Alterations in oxygenation saturation values are evident when variations from normal oxygen delivery or oxygen consumption occur. Hypoxemia may occur in supine obese

patients when decreased lung volumes lead to ventilation perfusion mismatch.

Chapter Two

Review of Literature

It has been shown that the most significant hypoxemia in obese subjects occurs in the supine position (Sharp, et al., 1986). An extensive literature search reveals that obesity contributes to changes in lung volumes, compliance, work of breathing, oxygen cost of breathing, and oxygenation when compared to individuals of normal weight. An understanding of the pulmonary alterations when the obese patient lies supine provides rationale for the findings of reduced saturation values in obesity.

Effect of obesity on lung volumes

A low expiratory reserve volume (ERV) was a consistent feature of studies relating obesity and lung volumes while RV remained unchanged. The ERV was further decreased when the obese patient was supine. Bedell, Wilson, and Seebohm (1958) sought to determine the frequency of pulmonary abnormalities in the obese population. Vital capacity, inspiratory capacity, and expiratory reserve volume were measured by spirometry. Nitrogen washout studies assessed FRC. Arterial blood gases were also analyzed in the

semirecumbent position. Only descriptive analyses of the mean measurements were reported.

The patients were classified into two groups based on arterial oxygen saturation (SaO_2): normal and hypoxic. Oxygen saturation above 93.2% was considered normal. Hypoxia defined a SaO_2 of less than 93.2%. Thirteen patients revealed normal SaO_2 measurements and 15 indiviuals produced hypoxic values. Various cardiopulmonary disorders were clinically diagnosised in 12 of the 15 patients with hypoxic saturation values. Cardiopulmonary disorders might have contributed to the lower saturation values found in these patients.

The researchers found that the ERV was decreased whether the SaO_2 was normal (> 93.2%) or hypoxic (< 93.2%). The mean ERV was only 75% of predicted values (based on height) in the 13 patients with normal saturation values. Pulmonary function studies were further evaluated in six of the 13 patients with normal saturations before and after weight loss. A mean weight loss of 52 pounds was achieved. An increase in ERV from 63% to 102% after weight loss was demonstrated. A substantially lower ERV from predictied was evident when evaluating the three patients with hypoxic arterial saturations (< 93.2%) who had no cardiopulmonary diseases. A mean ERV of 42% of predicted volumes was observed. These findings suggested when a low arterial

saturation was present a larger decrease in ERV from predicted values could be found.

Cullem and Formel (1962) studied 15 male subjects aged 29-73 years with weights between 142%-240% of ideal body weight (IBW). Respiratory defects in subjects were investigated. Oxygen and carbon dioxide washout methods measured the total lung volume and its subdivisions while the patients were supine with the head of the bed elevated 30 degrees.

The average ERV of all subjects was found to be 0.44 L. Less reduction in inspiratory capacity was shown. Residual volume measurements were normal except in three subjects who were considered to have pulmonary emphysema. Cullem and Formel (1962) commented that the low ERV observed was due to the "increased elastic recoil of the lung, a chest wall defect favoring a less expanded resting position for the thorax or elevation of the diaphragm due to abdominal fat" (p. 529).

Craig et al. (1971) studied closing volume and its relationship to gas exchange in seated and supine positions. The researchers studied 22 subjects, aged 21-78 years, who were clinically free of cardiorespiratory disease. Eight of the 22 subjects were greater than 110% above ideal bodyweight. Respiratory volume measurements of closing volume, FRC, ERV, and IC were obtained by the closed helium technique. Pulmonary measurements were obtained in a

straight-backed chair and supine on an operating-room table. Ten minutes were allowed to elapsed between posture change and measurement. Analysis of the data was evaluated by the Student's *t*-test for paired differences. Of the eight subjects who were greater than 110% above IBW, the mean sitting FRC (2.54 ± .29 L) was greater than the mean supine FRC (2.03 ± .10 L). Using the Student's *t*-test for paired differences, a $p < .05$ was determined. In these same eight subjects, the mean sitting and supine closing volume (sitting = 2.90 ± .26 L; supine = 2.67 ± .24 L) was found to be greater than the FRC in both the sitting and supine position. No significance reference to this finding was determined. The research showed that some percent of small airways do close during normal tidal breathing.

Studying four obese subjects aged 29-43 and 192-248% above IBW, Emirgil and Sobol (1973) sought to demonstrate the effect of obesity on pulmonary function before and after weight loss by therapeutic starvation. Pulmonary function testing included spirometry, single breath carbon monoxide diffusing capacity, body plethysmograph measurements, and single breath nitrogen washout.

Weight loss of this very small sample size ranged from 62-150 pounds. The ERV increased in all subjects after weight loss (an average of 0.7 L - 1.775 L). When comparing residual volume to percent of TLC, the findings revealed that the residual volume was essentially unchanged. This

data from Emirigil and Sobol indicated that the lower FRC before weight loss was the effect of excessive weight on the chest wall decreasing the ERV.

Farebrother, McHardy, and Munro (1974) found a statistically significant decrease in ERV while supine in eight severely obese subjects. Research participants included four men and four women, ages 24-56 years, and weight ranging from 147% - 236% of IBW. Spirometric tracings obtained in the sitting and supine position measured VC and ERV. Utilizing the paired t-test, the mean sitting ERV was reported as 0.72 L ± 0.11 while a mean supine ERV of 0.37 L ± 0.07 was seen. A significance of $p < .025$ was determined. Estimation of each subject's closing volume (CV) was determined by inhalation of argon boluses and measured radiographically. The difference between ERV and CV was expressed as a percentage of VC by the following formula:

$$\left[\frac{ERV - CV}{VC}\right] \%$$

Underventilation of dependent lung regions became more probable when a greater negative value (representing more airway closure) was obtained from this calculation. These values have been summarized in Table 3. Mean sitting and supine indexes before weight loss were negative in all but two cases sitting. A significance using the paired t-test

($p < .001$) was found between sitting and supine indexes before weight loss. After weight loss, a greater value for the sitting index was obtained. Significance between sitting and supine after weight loss was reduced to $p < .025$. When evaluating the sitting values before and after weight loss a $p < .025$ was obtained. The difference between supine values before and after weight loss was not significant.

Table 3.

Mean values of (ERV-CV)/VC% in eight obese patients before and after weight loss

	$\frac{(ERV - CV)}{VC}$ %		
	Before	After	p
Sitting	- 6.0 ± 2.9%	+ 3.4 ± 4.9%	< .025
Supine	- 15.8 ± 2.2%	- 9.5 ± 4.3%	> .05
p	< .001	< .025	

Note. Adapted from "Relation between Pulmonary Gas Exchange and Closing Volume before and after Substantial Weight Loss in Obese Subjects" by J. B. Farebrother, G. J. R. McHardy, and J. F. Munro, 1974, British Medical Journal, 3, p. 392.

Vaughn, Cork, and Hollander (1981) used sitting pulmonary function testing and arterial blood gas analysis in the evaluation of the lung function of 37 morbidly obese patients. These subjects were studied before and after weight loss following elective jejunoileal small bowel bypass. Subjects were followed 5-20 months post-operatively. Using the Student's *t*-test for data analysis, a significant mean weight loss of 52 kg was observed ($p < .001$). A significant ($p < .001$) increase in ERV after weight loss (54.6 ± 4.1 % to 93.2 ± 5.7 %) from predicted values was demonstrated. Arterial blood gas values were not statistically significant before or after weight loss. However, a significant improvement in oxygenation was detected when employing linear regression analysis among changes in arterial oxygen tension (PaO_2) and alveolar-arterial oxygen tension difference ($PAO_2 - PaO_2$) and between several morphological factors of the subjects. Improvement in oxygenation by correlating the $PAO_2 - PaO_2$ differences with weight loss ($p < .003$), change in body mass index ($p < .001$), per cent change in ideal weight ($p < .001$), change in body surface area ($p < .005$), change in abdominal girth ($p < .027$), and change in ERV ($p < .005$) was significant. The oxygenation improvement found with weight loss resulted from an increase in FRC due to a greater ERV.

Compliance in obesity

According to the findings of Naimark and Cherniack (1960), total respiratory compliance in obesity was decreased. A total of 36 subjects were studied: 24 normal weight and 12 obese. Obese subjects were defined as weighing greater than 25% above their ideal weight. Negative histories of pulmonary, cardiac, or neuromuscular diseases were obtained. A latex balloon positioned in the esophagus measured transpulmonary pressure by esophageal-mouth pressure differential. To avoid air flow recordings from nasal airway breathing, a tightly fitted nose clip was positioned. A pneumotachometer registered air flows. Patients then entered an "airtight body plethysmograph and breathed through a tube into and out of an oxygen-filled spirometer, containing a CO_2 absorber, situated outside the plethysmograph" (Naimark & Cherniack, 1960, p. 378). Pressure was exerted upon the pulmonary system by increasing the pressure inside of the body plethysmograph. A motor blower provided the source for the application of negative pressure around the subjects body. Pressure applied varied from -25 cm H_2O to +20 cm H_2O. After allowing the subjects to achieve a steady breathing pattern at each pressure level, changes in lung volumes and end-expiratory transpulmonary pressure were measured. Compliance (dV/dP) across the lung, chest wall, and total respiratory system were also plotted for all subjects at each pressure level.

Results of the investigation revealed that the total respiratory compliance in obesity (0.052 L/cm) was reduced to only one-third that of normal weight subjects (0.119 L/cm). When graphing pressure-volume curves of the total respiratory system, flatter curves were noted for obese subjects. The compliance of the chest wall was significantly lower ($p < .01$) than the normal weight individual when differentiating between chest wall compliance and lung compliance. Mean chest wall compliance in normal weight subjects equalled 0.224 L/cm; whereas, obese subjects values decreased to 0.077 L/cm. Lung compliance was shown not to differ among the two subject groups. The authors concluded the significant decrease in chest wall compliance was a major contributing factor for the decrease in total respiratory compliance found.

Naimark and Cherniack (1960) also correlated compliance and posture between 18 of the normal weight subjects and 12 of the obese subjects. Compliance values of the lung, chest wall, and total respiratory system in the supine position were essentially unchanged in normal weight subjects when compared to values obtained in the seated position. A decrease in total respiratory system and chest wall compliance was evident in seven of the eight obese subjects while supine. A significance of $p < .05$ was calculated for the observed chest wall compliance reduction.

A decrease in overall obesity chest compliance was also demonstrated by Sharp, Henry, Sweany, Meadows, and Pietras (1964a). The researchers placed 30 lb. and 60 lb. buckshot bags on the thorax of 14 normal weight subjects (mean weight 175 lbs.) and compared total respiratory volume-pressure curves to 15 extensively obese individuals (mean weight 304 lbs.). Six of the normal weight subjects were further studied while anesthetized and paralyzed.

Subject age ranges were as follows: conscious normal weight subjects; 29-45 years; unconscious normal weight subjects 38-67 years; and obese subjects, 29-51 years. The population sample of the conscious normal weight subjects included male physicians and laboratory personnel. The six unconscious, anesthetized and paralyzed subjects consisted of patients who were undergoing elective surgical procedures. All normal weight subjects were free of respiratory disease and symptoms. Population profile of the obese subjects included 10 subjects with essentially normal respiratory systems, while 5 subjects suffered from obesity hypoventilation syndrome (OHS). The authors grouped the obese subjects in two groups to compensate for the presence of hypoventilation in their data analysis. Information regarding population sampling methods of the obese subjects was lacking in the published study.

The purpose of the study was to assess the effects of loading an excessive mass on the normal weight thorax and to

"determine whether obesity produces similar changes in the thoracic and total respiratory volume-pressure curves and hence whether the externally loaded normal respiratory system is a relevant model to the respiratory system of the obese subject" (Sharp, et al., 1964a, p. 959). Thorax and abdominal mass loading was performed.

Functional residual capacity was measured in all normal weight subjects prior to mass loading by the nitrogen washout method. The authors placed the eight conscious unparalyzed normal weight subjects and the 15 obese subjects supine in a tank respirator to measure static volume-pressure curves of the lungs, thorax, and total respiratory system. The respirator served as a housing in which the investigators applied negative pressures to the thorax. The use of an esophageal balloon catheter measured esophageal pressure. Tidal volume, tank pressure, and esophageal pressure were recorded based on 8-25 volume-pressure points. Thirty and sixty pound buckshot bags were also placed at different intervals to the lower thorax, upper thorax, and abdomen of four normal weight subjects. The data enabled the researchers to plot volume-pressure curves of the thorax, lung, and total respiratory system.

Results of the curves plotted from mass loading of the thorax and abdomen of normal weight subjects generated a parallel displacement of the total respiratory curve to higher pressure levels. The chest wall and lung compliance

curves were isolated from the total respiratory compliance curve. The total chest wall compliance was found to be most reduced when mass loading was applied to the abdomen and lower thorax of normal weight subjects. Changes in the chest wall compliance curve included an increase in the slope in the lower portion of the curve, but little change in the slope of the upper portion of the curve with mass loading of the 30 lbs. bags. Heavy abdominal loading of the 60 lbs bags flattened both the lower and upper portion of the volume-pressure curves. A similar alteration of the chest wall compliance curve was seen when comparing paralyzed to non-paralyzed normal weight subjects. Flattening of the lower portion of volume-pressure curves provides relevance, since this is the portion of the curve that an individual breathes during normal spontaneous respiration. Less compliance at this location on the curve hindered the ability of the lung and chest wall to expand during normal inspiration efforts.

When devising the curves for the ten obese subjects without OHS, similar findings of the mass-loaded curves of normal weight subjects were observed. In the five obese patients who had OHS a more severely flattened slope of the total respiratory volume-pressure curve was seen. The research by Sharp and colleagues (1964a) reported the compliance curves of obesity resembles compliance curves obtained when the thorax and abdomen of normal weight

subjects was mass loaded. Chest wall compliance was more severely altered than lung compliance and contributed to an overall reduction in total respiratory compliance.

Work of breathing in obesity

The added weight of the obese chest required increase work by respiratory muscles during ventilation. Additional work by Sharp, Henry, Sweany, Meadows, and Pietras (1964b) sought to determine the work of breathing in 22 obese subjects. Subject profiles included eight subjects of normal weight in relation to height (136-208 lbs.), and 14 obese subjects (253-370 lbs). Age dispersion of the normal weight group varied between 29-45 years, while the obese group ages ranged from 29-60 years. Obesity hypoventilation syndrome was evident in four of the 14 obese individuals studied.

Subjects were placed in a tank respirator to measure the mechanical work of breathing. After the subjects were placed in the enclosed tank respirator, pressure changes inside the respirator performed the respiratory work for the subjects. A mouth-piece in place measured tidal volume and air flows while also preventing room air breathing by the subjects. The change in pressure across the respiratory system was calculated by the apparatus and correlated with coinciding changes in lung volumes. The investigators plotted volume-pressure curves with the values derived. By

measuring the volume-pressure plots of the tank respirator against the inspired volume the researchers calculated total inspiratory work. The measurements of work was expressed as kilogram-meter(kg-m)/breath at a common respiratory rate of 20 breaths/minute.

Results of the measurements revealed normal weight subjects respiratory work averaged 0.073 kg-m/breath whereas, obese weight subjects averaged 0.095 kg-m/breath. Subject three of the obese normal group had a total respiratory work greater than twice the average of the normal weight subjects (0.148 kg-m/breath). The difference between normal and obese subjects respiratory work was 30 per cent. This increase in the obese respiratory work was significant ($p < .05$).

Oxygen cost of breathing

Oxygen is necessary for respiratory muscles to perform the mechanical work of breathing. Oxygen consumption by the respiratory system can be estimated by determining the difference between the oxygen consumption during restful breathing and that seen during periods of increased ventilation. When increased mechanical work is present the oxygen cost of breathing is increased further.

Fritts, Filler, Fishman, and Cournand (1959) studied seven normal weight subjects (aged 19-56 years; weight ranging from 117.92 lbs - 200.86 lbs) and five obese

subjects (aged 33-61 years; weight ranging from 264 lbs.-310.2 lbs) to determine the efficiency work performed by the respiratory system. The researchers described the respiratory system as a mechanical structure comprised of an engine and a pump. The engine was composed of the chest wall and diaphragm. It transformed metabolic energy into mechanical work. The pump was created by the airways and lungs which translates the mechanical work into the transaction of air between the atmosphere and alveoli. Frits et al. (1959) devised a method to estimate the energy used for breathing (expressed in Kilogram-meter/Liter = Kg-m/L) and the mechanical work performed on the lungs. The measurements were recorded at various respiratory rates from 5-30 breaths per minute. The following formula determined the per cent efficiency of the respiratory system:

$$\% \text{ efficiency} = \frac{\text{mechanical work performed on the lungs}}{\text{total energy used for breathing}} \times 100$$

The energy expenditure (energy cost) for breathing in normal weight individuals varied between 0.8 - 1.9 kg-m/L/minute; values from the obese individuals ranged between 6.6 - 26.2 kg-m/L/minute. The mechanical work performed on the lungs in normal weight subjects was found to be moderately high in only two of the obese subjects. The remaining three obese subjects were found to have normal mechanical work values. The obese subject's efficiency

values of breathing at all ventilation rates were found to be lower than those in normal subjects.

The investigators concluded the abnormally high energy cost in the obese population studied contributed to the lower efficiency values obtained. The authors postulated that the extra energy needed to move the adipose chest overlying the thoracic cage and abdominal wall contributed to the results found in their findings.

Cherniack and Guenter (1961) also studied the efficiency of the respiratory muscles and the mechanical work done to overcome elastic resistance during breathing in normal and obese individuals. Normal weight subjects investigated included seven males, between 18-35 years. Six female and one male obese subjects, ages 34-70, who were at least 25% above their ideal weights, were also studied. Both subject sets had no clinical evidence of cardiac or respiratory disease. Respiratory rate, ventilation (L/min), oxygen cost of breathing, and efficiency of ventilation were measured. As in previous studies, measurements were obtained at various respiratory rates.

Results indicated that the mean oxygen cost of breathing in normal subjects was 0.85 milliliters/liter (ml/L); whereas, obese subjects demonstrated a value of 2.88 ml/L. A lower efficiency of the respiratory muscles was also observed in the obese subjects (3.9%) when compared to the normal subjects (9.7%).

In the same study, Cherniack and Guenter (1961) also sought to determine the mechanism leading to the low efficiency found in obese individuals. The normal subjects were further studied. A pressure vest was applied to the chest wall of normal subjects. The vest was inflated to a pressure of 15-20 cm of water at maximum expiration. Measurements as above were recorded again. The employment of extrapulmonary resistance by the pressure applied caused a fall in chest wall compliance, but no change in lung compliance. The fall in chest wall compliance corresponded with an increase in the oxygen cost of breathing (104-214% of control) and a decrease in the efficiency of the respiratory muscles (34-74% of control). Externally applied pressure also decreased the normal weight subjects FRC.

Cherniack and Guenter (1961) proposed the low efficiency seen in obese patients respiratory muscles was related to the altered chest wall compliance. They suggested a low FRC in the obese population also contributed to the low respiratory muscle efficiency found.

Oxygenation in obesity

Selected literature available on oxygenation values in obesity included studies regarding oxygen tension (PaO_2) and oxygen saturation measurements by blood gas analysis (SaO_2). Bedell et al. as early as 1958 investigated oxygen saturation in obesity. The authors studied semirecumbant

arterial blood gas values in 28 obese patients who were at least 100 pounds over IBW. Thirteen subjects had normal arterial saturation values (SaO_2 > 93.2% as defined by the authors). These subjects averaged 303 pounds, and were from 19-67 years of age. Fifteen of the subjects experienced arterial hypoxemia (SaO_2 < 93.2%). Twelve women and 3 mean with age ranges from 39 to 67 years, and a mean weight of 325 pounds provided the population profile of the hypoxic group. Ten of the hypoxic group subjects displayed clinical or laboratory evidence of pulmonary disease (asthma, emphysema, pulmonary infarcts, pulmonary edema, myxedema). Although three of the patients with arterial hypoxemia (SaO_2 = 90.6%, 91.4%, & 92.4%) were without evidence of lung disease.

Said (1960) also studied pulmonary gas exchange in obesity. Twenty obese subjects without cardiac or pulmonary disease were investigated in the recumbent (supine) position. Physical characteristics of the subjects included a mean age of 48 years and mean weight of 312 pounds. Arterial samples from the brachial or radial artery were evaluated for PaO_2, partial pressure of carbon dioxide (PCO_2), and blood pH. Tabulated gas exchange data from Said's results revealed a mean PaO_2 value of 70 mm Hg. Oxygen tension values ranged from 46-86 mm Hg. The study lacked a control group for comparison of values.

The effect of the sitting and supine position on gas exchange was further assessed by Said in 5 of the 20 subjects. The subjects investigated in both position included two males and three females; ages 36-58 years, and weights between 215-435 pounds. Subjects served as their own control. An average sitting PaO_2 of 78.8 mm Hg was reported, while the average supine PaO_2 dropped to 67.2 mm Hg. Said's study reported that alterations in arterial oxygen were evident in obese patients without known cardiac or respiratory illness. No significance statistics were provided.

Farebrother et al. (1974) evaluated pulmonary gas exchange in eight severely obese subjects before and after weight loss by therapeutic starvation. Subjects ranged from 147% to 236% of ideal body weight and were between 32-56 years of age. Four women and four men were studied. Sitting arterial blood samples were first obtained. A second arterial sample was then drawn after the subjects were supine for at least 15 minutes. Blood samples were analyzed for PaO_2 and PCO_2 values.

Subjects endured therapeutic starvation for 8-44 weeks and achieved a mean weight loss of 73.92 pounds. Using the paired t-test, the data revealed the mean supine PaO_2 (77.1 ± 3.5 mm Hg) was significantly less than the mean sitting PaO_2 (84.6 ± 4.3 mm Hg) before weight loss: $p < .025$. After weight loss, the mean supine PaO_2 (81.6 ± 4.2 mm Hg)

was still significantly less than the sitting mean PaO_2 (03.9 ± 5.0 mm Hg): $p < .05$. There was no significant improvement of arterial oxygenation after weight loss. A significant improvement was seen only when the weight loss placed the subjects within 30% of ideal body weight (p values were not provided for this conclusion). The authors suggested that a "low weight threshhold" might have to be reached before significant improvement in pulmonary exchange occurred.

The effect of the semirecumbant and supine position on blood gas exchange was evaluated by Vaughan and Wise (1975) using arterial blood gas analysis in 22 non-smoking obese female subjects preoperatively and postoperatively. The subjects lacked clinical evidence of any cardiopulmonary disease. After breathing room air for 15 to 20 minutes, radial artery sampling followed. The arterial blood samples were analyzed for PaO_2, PCO_2, pH, SaO_2. The Student's paired t-test was utilized for statistical examination to evaluate the significance of the differences between supine and semirecumbant. The range of semirecumbant SaO_2 equalled 89.9 - 97.8 % (mean of 95.6 ± 0.8); the supine SaO_2 ranged from 92.6 - 97.1% (mean of 95.5 ± 0.7). A mean supine PaO_2 of 78.4 ± 2.4 mm Hg along with a mean semirecumbant PaO_2 of 76.4 ± 1.6 mm Hg was observed. No significance was found between the two positions on blood gas exchange; a finding not routinely found in the obesity literature when position

changes were evaluated. The investigators failed to clearly define "semirecumbant" and "supine". It was possible that the degree of head elevation in the semirecumbant position used by the investigators altered their findings from other published literature.

Several examples of studies examining oxygenation in obesity compare before and after weight loss values can be found in the literature. In 1981, investigators Vaughan et al. examined the effect of massive weight loss on oxygenation and pulmonary function testing. A total of 37 patients (34 females and 3 males) were studied with a mean beginning weight of 305.8 ± 8.8 pounds. Mean weight loss was 114.4 pounds. Eleven of the patients had supine arterial blood gas values sampled before and after weight loss. Specific characteristc profiles of these 11 subjects were not available. The arterial blood samples were analyzed for pH, PaO_2, PCO_2, and alveolar-arterial difference. The Student's *t*-test for paired data was employed to analyze the data for significance. A significance was defined as $p < .05$. Of the 11 subjects who had arterial blood gases drawn, the researchers reported a nearly significant ($p = .053$) improvement in PaO_2 with weight loss. Although, when applying linear regression analysis, a significant improvement in PaO_2 was correlated with the duration of weight loss ($p < .05$) and a change in ERV ($p < .01$). An increase in ERV correlated with an

improvement in oxygen tension values. The authors suggested that a significant difference in PaO_2 might have been demonstrated if the number of patients studied had been greater.

Taylor, Kelly, Elliott, Jensen, and Jones (1985) prospectively studied arterial blood gases in 56 patients before and after Roux-en-Y gastrojejunostomy for morbid obesity. The researchers examined the incidence, severity, and the course of arterial hypoxemia in obese patients. They endeavored to predict from presurgical data which patients were at greatest risk for postoperative hypoxemia. Physical traits of the population sample revealed 54 females and two males. Characteristics of the sample population showed a mean weight of 250 lbs. and ages ranging from 17-58 years. Eleven patients were previous smokers, although none of the 56 patients studied had any history of pulmonary disorders. Arterial blood gases were drawn with the patient breathing room air and lying in the supine position with head elevated at 30 degrees. Preoperatively 13% (seven of 56) of the patients had a PaO_2 value of less than 60 mm Hg. Nine percent (five of 56) had an SaO_2 less than 90%.

Summary

A search through the literature found the respiratory complications of obesity included effects on lung volumes, compliance, work of breathing, oxygen cost of breathing, and

oxygenation. The results of these respiratory complications is hypoxemia, especially in the supine position.

No studies have been found in the literature that utilize pulse oximetry measurements to study the effect of posture on oxygen saturation in the obese individual. This study sought to confirm the alterations in respiratory function in supine obese subjects can be detected with the use of a pulse oximeter.

Chapter Three

Methodology

The literature review produced evidence that the supine obese person experiences alterations in the respiratory system. These supine pulmonary changes have been manifested as low arterial PaO_2 and SaO_2 values. The purpose of this study was to relate the effect of body position changes on arterial saturation values measured by pulse oximetry (SpO_2) in the obese individual as compared to the nonobese subject.

Research Design

The study utilized a quasi-experimental pretest-posttest design that can be symbolized as:

$$O_1 \quad X \quad O_2$$

$$O_3 \quad X \quad O_4$$

O_1: observation by pulse oximetry in obese subjects

X: represents change in position from sitting to supine

O_2: observation by pulse oximetry in obese subjects

O_3: observation by pulse oximetry in nonobese subjects

O_4: observation by pulse oximetry in nonobese subjects

Population, Sample, and Setting

The population consisted of obese and nonobese groups of 14 subjects each. All subjects were healthy, nonsmokers, and were without clinical evidence of cardiopulmonary disease. The control sampling frame included volunteer nonobese subjects from a single mid-Atlantic Nurse Anesthesia program; all with an ASA 1 physical status. The study's sampling frame consisted of obese patients who presented to the anesthesia department in a single 1,052 bed mid-Atlantic metropolitan medical center. The entire obese group physical status was ASA II based solely on the magnitude of their obesity. Subjects were collected by nonprobability convenience sampling. The data was obtained in a preoperative holding area where patients present for anesthetic consultation prior to surgical procedures.

Data Collection Procedure

Research approval for the experiment was provided by the medical center's Committee on the Conduct of Human Research. The researcher solicited patients who presented to a preoperative holding area, or volunteer personnel. Explaination of the study's purpose and procedure was given prior to the subject's consent. After agreeing to participate, the subject was asked to sit on the edge of a stretcher. Torso position was 90 degrees to lower extremities and legs dangled over the side of the stretcher.

A pulse oximeter monitoring probe was placed on the subjects index finger once seated. Under spontaneous room air ventilation, the pretest arterial oxygen saturation was measured by the pulse oximeter (SpO_2) and was recorded (see Appendix A). After measurement of the sitting SpO_2 value, the subjects were placed supine (zero degrees head elevation) on a stretcher for 10 minutes. Pulse oximetry values were recorded immediately following position change and then serially for a total of 10 minutes.

Instrumentation

Arterial oxygen saturation measurements were obtained from a single Nellcor® N-100 Pulse Oximeter, serial number: 100-146607-D-C. Disposal pulse oximeter sensor probes (D-25) were used for each subject. The pulse oximeter used was manufactured by Nellcor, Incorporated (Inc.), 2595 Whitesell Street, Hayward, California, 94545. Accuracy values (% SpO_2 ± one SD) of the instrument as stated by Nelcor were: 70-100% ± 2% and 50-70% ± 3%. Nellcor reported the disposable sensor probe was accurate within a temperature range of 28-42 degrees Celsius, and the pulse oximeter monitor was accurate within a temperature range of zero - 40 degrees Celsius (Nellcor, Inc., 1985)

Data Analysis

The data were analyzed by repeated measures analysis of covariance (ANCOVA) using the sitting SpO_2 as the covariant. The paired t-test employed further analysis. The data was reviewed to determine if position changes in the obese subject from sitting to supine produced a statistical significant alteration in SpO_2 measurement as detected by pulse oximetry.

Chapter Four

Results

This study compared the effect of body position changes on arterial oxygen saturation in obesity as measured noninvasively by pulse oximetry. Fourteen obese ASA II subjects and fourteen nonobese ASA I subjects were included in the sample population. Thirteen of the obese subjects were further classified as morbidly obese since their ideal body weight (IBW) exceeded twice the calculated IBW. All subjects were nonsmokers and clinically absent of any cardiopulmonary disorders. A significance of $\alpha = .05$ was used for data analysis.

Sample characteristics of the two groups height (centimeters = Cm), weight (kilograms = Kg), IBW (Kg), percent of IBW (% of IBW), age (years), and gender were collected (see Appendixes B & C). The Student's *t*-test was performed to analyze the means of the percent (%) of IBW, age, and gender between the obese and non obese group (see Table 4). By the study design, a large and statistically significant difference ($p < .0001$) in % IBW was shown. No significance in age or gender was evident.

Table 4

Student t-test summary for profile characteristics between the obese and nonobese group

Variable	Mean Difference	t-ratio	df	p
Percent IBW	135.535	11.174	26	.0001
Age	4.643	1.792	26	.085
Gender	0.143	1.080*	--	.280

Note. *z- ratio, binomial approximation to the normal curve

Oxygen saturation values were noninvasively measured by optical plethysmography and spectrophotometry by the Nellcor® N-100 Pulse Oximeter. Baseline sitting SpO_2 values were first determined. The subjects were then placed supine on a stretcher. Measurement of the SpO_2 value was collected immediately supine and for 1 minute intervals up to 10 minutes. The raw data obtained from the data collection was tabulated (see Appendix D).

The mean SpO_2 values across groups at each measurment were determined. The paired t-test examined the difference between the means of each measurement by group (see Table 5). No significance difference was found between the mean group values at each measurment interval at $\alpha = .05$.

Table 5

Paired t-test results of mean measurements across groups

Measurement Number*	Mean Difference	t-ratio	df	p
1	-0.214	-0.508	26	.616
2	-1.143	-1.460	26	.156
3	-0.786	-1.461	26	.156
4	-0.143	-0.262	26	.796
5	-0.215	-0.410	26	.685
6	-0.643	-1.205	26	.239
7	-0.715	-1.193	26	.244
8	-0.643	-1.101	26	.281
9	-0.928	-1.760	26	.090
10	-0.857	-1.578	26	.127
11	-0.763	-1.205	26	.239
12	-0.571	-1.096	26	.283

Note. Measurement number codes:

1 = sitting SpO_2
2 = SpO_2 immediately after position change to supine
3 = SpO_2 1 minute after position change
4 = SpO_2 2 minutes after position change
5 = SpO_2 3 minutes after position change
6 = SpO_2 4 minutes after position change
7 = SpO_2 5 minutes after position change
8 = SpO_2 6 minutes after position change
9 = SpO_2 7 minutes after position change
10 = SpO_2 8 minutes after position change
11 = SpO_2 9 minutes after position change
12 = SpO_2 10 minutes after position change

The evaluation of the data by repeated measures analysis of covariance (ANCOVA) was reported (see Table 6). The sitting SpO_2 value was used as the covariate. The Between Group effect tested the hypothesis that the two group means across all times were equal. This difference was found not to be significant at $p < .05$.

The Within SpO_2/Time effect (see Table 6) measured the extent to which the means across both groups (adjusted for baseline) differed from each other. This analysis tested the hypothesis that the means (μ) of the population across groups at each time measured were equal: $\mu_1 = \mu_2 = \ldots\mu_{12}$. The Within SpO_2/Time effect approached significance: $p = .089$.

Seeing that the Within SpO_2/Time effect approached significance, the Within SpO_2 X Group interaction effect analyzed the pattern of change over time between the two groups. No significance ($p = .574$) was found in the pattern of change between the obese and non obese group.

The ANCOVA further explored the difference between the mean supine SpO_2 values (adjusted for baseline) across both groups between adjacent time measurements (see Table 6). A strongly statistical significant ($p = .001$) difference from the mean immediate supine SpO_2 value (μ-2) and the mean 1 minute supine SpO_2 value (μ-3) was observed. Significance continued when analyzing the difference between the mean 1 minute supine SpO_2 value (μ-3) and the mean 2nd minute

Table 6

ANCOVA Summary of SpO_2 Values

Source	Sum of Sqs	df	Mean Sq	F-ratio	p
Between					
Group	19.296	1	19.296	1.674	.208
Error	288.108	25	11.524		
Within					
SpO_2/Time	10.863	9	1.207	1.708	.089
SpO_2 X Grp	5.387	9	0.599	0.847	.574
Error	159.123	225	0.707		
$\mu 2 - \mu 3^{\bullet}$	23.250	3	7.750	6.982	.001
Error	27.750	25	1.110		
$\mu 3 - \mu 4$	4.149	3	1.383	3.186	.041
Error	10.851	25	0.434		
$\mu 4 - \mu 5$	0.391	3	0.130	0.144	.933
Error	22.609	25	0.904		
$\mu 5 - \mu 6^{\bullet\bullet}$	1.439	3	0.480	0.954	.430
Error	12.561	25	0.502		

Note. $^{\bullet}$ Measurement number codes:
2 = SpO_2 immediately after position change to supine
3 = SpO_2 1 minute after position change
4 = SpO_2 2 minutes after position change
5 = SpO_2 3 minutes after position change
6 = SpO_2 4 minutes after position change

$^{\bullet\bullet}$ Remaining contrast measurements were found not to be significant

supine SpO_2 value (μ-4): $p = .041$. Further analysis between other adjacent time measurements was found not to be significant.

The paired t-test was executed to further analyze the magnitude of change between the mean measurements across the obese and nonobese group (see Table 7). Analysis revealed that both groups had a statistically significant drop in oxygen saturation from the mean sitting (μ-1) to the mean immediate supine (μ-2) values: obese, $p = .007$, nonobese, $p = .005$. The magnitude of the drop was larger for the obese group.

The obese group continued to have a statistically significant change for the next two mean time measurements: (1) from immediate supine to the 1 minute measurement ($\mu 2$-$\mu 3$, $p = .034$), and (2) from the 1 minute to the 2 minute measurement ($\mu 3$-$\mu 4$, $p = .013$). The obese group had no further statistically significant changes. The nonobese SpO_2 value change between measurements was statistically significant from sitting to immediately supine ($\mu 1$-$\mu 2$, $p = .005$).

Table 7

Paired t-test results of mean differences across time by group

Group	Measurement Number*	Mean Difference	t-ratio	df	p
Obese	$\mu 1-\mu 2$	1.786	3.209	13	.007
Nonobese	$\mu 1-\mu 2$	0.857	3.379	13	.005
Obese	$\mu 2-\mu 3$	-0.857	-2.375	13	.034
Nonobese	$\mu 2-\mu 3$	-0.500	-1.836	13	.089
Obese	$\mu 3-\mu 4$	-0.500	-2.876	13	.013
Nonobese	$\mu 3-\mu 4$	0.143	0.806	13	.435
Obese	$\mu 4-\mu 5$	0.000	0.000	13	1.000
Nonobese	$\mu 4-\mu 5$	-0.071	-0.322	13	.752
Obese	$\mu 5-\mu 6$	0.286	1.749	13	.104
Nonobese	$\mu 5-\mu 6$	-0.143	-0.694	13	.500
Obese	$\mu 6-\mu 7$	0.071	0.434	13	.671
Nonobese	$\mu 6-\mu 7$	0.000	0.000	13	1.000
Obese	$\mu 7-\mu 8$	0.000	0.000	13	1.000
Nonobese	$\mu 7-\mu 8$	0.071	0.366	13	.720
Obese	$\mu 8-\mu 9$	0.071	0.201	13	.844
Nonobese	$\mu 8-\mu 9$	-0.214	-1.385	13	.189
Obese	$\mu 9-\mu 10$	0.143	0.354	13	.729
Nonobese	$\mu 9-\mu 10$	0.214	1.147	13	.272
Obese	$\mu 10-\mu 11$	-0.286	-1.295	13	.218
Nonobese	$\mu 10-\mu 11$	-0.071	-0.366	13	.720
Obese	$\mu 11-\mu 12$	-0.143	-0.618	13	.547
Nonobese	$\mu 11-\mu 12$	-0.071	-0.434	13	.671

* Same measurement codes as Table 5

The graphic trends of the mean SpO_2 values of both groups are plotted (see Figure 16). Despite the illusion of a widespread difference in the mean group values at measurements one, two, nine, and ten the paired $\underline{t}$-test revealed no significant different between the groups measurements. From the graph, there is a notable drop in SpO_2 values in both groups from $\mu 1$-$\mu 2$. The obese group appeared to have had a larger more dramatic drop (larger negative slope) from the sitting to supine than the nonobese group. The difference from $\mu 1$-$\mu 2$ was found significant for both groups by the paired $\underline{t}$-test of mean differences across time by group: obese, $\underline{p} = .007$; nonobese $\underline{p} = .005$. The obese groups magnitude of change was found significant between $\mu 1$-$\mu 2$, $\mu 2$-$\mu 3$, and $\mu 3$-$\mu 4$. The obese trend recovered slow, and then dropped again gradually for several minutes. The nonobese group had a smaller initial drop in SpO_2 and generally trended slowly upward. The nonobese group experienced a significant change only from $\mu 1$-$\mu 2$. The appearance of a notable increase in the mean obese value changes from $\mu 10$ - $\mu 11$ was found not significant ($\underline{p} = .218$).

From the paired $\underline{t}$-test, it was shown the most significant difference in obese oxygen saturation occurred at mean SpO_2 measurements $\mu 1$-$\mu 2$, $\mu 2$-$\mu 3$, and $\mu 3$-$\mu 4$. The nonobese group had a significant difference in oxygen saturation between $\mu 1$-$\mu 2$. The Pearson Product-Moment correlation coefficients were computed (across both groups)

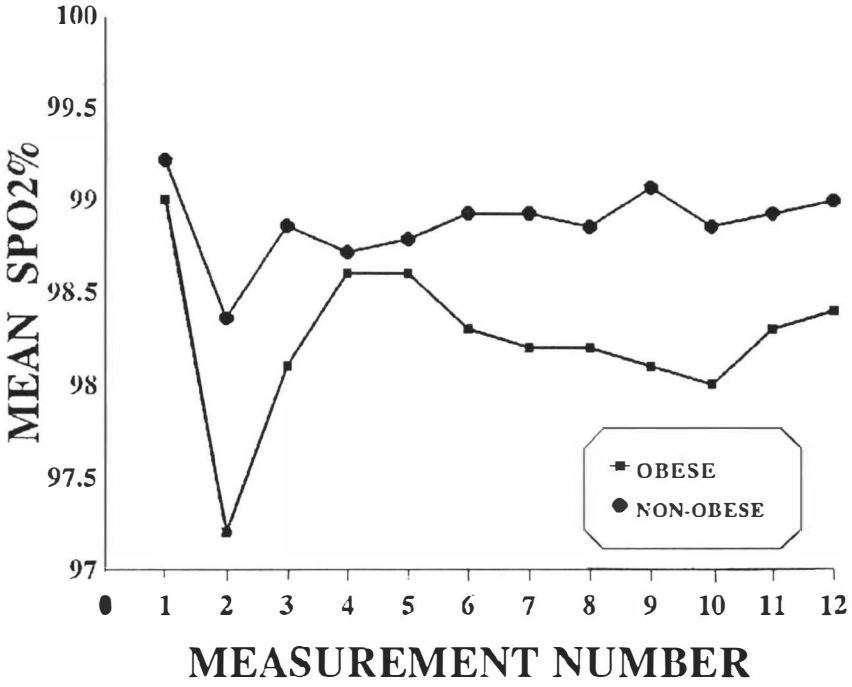

Figure 16. Mean SpO_2% vs. measurement number

between percent IBW vs. SpO_2 values at mean measurement one, two, three, and the amount of change from measurement 1-2, and 2-3 (see Table 8). The null hypothesis there was no correlation between IBW and oxygen saturation was tested.

Table 8

Pearson correlation coefficients between per cent of IBW and oxygen saturation measurements across both groups

Per cent of IBW vs.	r	p
μ Measurement 1 (sitting)	-0.067	.735
μ Measurement 2 (immediate supine)	-0.317	.100
μ Measurement 3 (1 minute supine)	-0.368	.054
μ Measurement 1-2 (amt of change)	0.428	.023
μ Measurement 2-3 (amt of change)	-0.200	.307

Note. Measurement number codes:
1 = sitting SpO_2
2 = SpO_2 immediate after positon change
3 = SpO_2 1 minute after position change

The correlation of IBW and oxygen saturation in the sitting position was virtually zero ($r = -0.067$). Immediately changing to the supine position, the correlation was -0.317, which approached ($p = .100$) but did not reach significance at $\alpha = .05$. At mean Measurement 3 (1 minute supine) there was a moderate negative correlation ($r = -0.368$) which was very close to statistical significance ($p = .054$). As IBW increased, the SpO_2 value at 1 minute supine was lower.

However, the amount of change in the SpO_2 value from mean Measurement 1-2 had a positive correlation of $r = 0.428$, which was significant ($p = .023$). As the per cent of IBW increased the amount of the change of the SpO_2 value from sitting to supine was larger. There was no significance found in the change from mean Measurement 2 (immediate supine) to mean Measurement 3 (1 minute supine).

Chapter Five

Discussion

The purpose of this study was to compare the effect of position changes on arterial oxygen saturation in obesity as measured noninvasively by pulse oximetry. The hypothesis stated there would be no difference between oxygen saturation values measured by pulse oximetry with position change from sitting to supine in obese subjects as compared to subjects within 20% of ideal body weight. By study design, a statistical significance between groups in per cent of ideal body weight was apparent ($p = .001$).

Macro analysis of the means across all measurements between both groups failed to reject the hypothesis. The ANCOVA revealed that the Between Group effect was not significant ($p = .208$), while the Within SpO_2/Time effect across both groups approached significance ($p = .089$).

Further micro analysis of the magnitude of change from the sitting to supine position between groups rejected the hypothesis. The ANCOVA established statistical significance between the mean (μ) supine SpO_2 values (adjusted for baseline) across both groups between the mean immediate supine measurement and the mean 1 minute supine measurement

($\mu 2-\mu 3$, $p = .001$) and also between the mean 1 minute supine measurement and the mean 2 minute supine measurement ($\mu 3-\mu 4$, $p = .041$).

The paired t-test determined which group had the significant change between measurements. It was shown that the obese group had a statistical significant change in SpO_2 values between the mean sitting SpO_2 and the mean immediately supine measurement ($\mu 1-\mu 2$), as well as between the $\mu 2-\mu 3$ and $\mu 3-\mu 4$ measurements. The nonobese group had a statistical significant change in SpO_2 value only from $\mu 1-\mu 2$.

Comparison of results to previous studies

An extensive review of the obesity literature was performed. No previous studies relating the effects of position change in obesity on arterial oxygen saturation as measured noninvasively by pulse oximetry was found for comparison to this study.

Limitations with the study

Several difficulties were associated with the study:

1. Initially during data collection subjects were interrupted by other health care personnel. Later, subjects were excluded from personnel interruption.

2. Anxiety produced by the normal milieu of the pre-op holding area (conversation of other personnel and patients;

presence of equipment), fear of instrumentation, and pre-op apprehension could have increased the subjects respiratory rate above normal volumes.

3. The obese subjects were further involved in data collection that included arterial blood gas sampling. Anxiety related to anticipation of blood sampling may have increased their respiratory rate above normal volumes.

4. Control subjects were obtained by a sample of convenience from nurse anesthesia students. The control group possessed knowledge of the sampling instrumentation and lacked instrumentation fear. These two circumstances could have altered the control groups respiratory status as compared to the study group.

Recommendations for further study

The recommendations for future study are:

1. Replicate the study with better corresponding study and control groups (either all volunteer subjects, or all pre-op subjects).

2. Replicate the study in a location where the milieu is consistent and lacks interruption.

3. Repeat the study using different positions at various degrees of head elevations: sitting, supine, trendelenburg, reverse trendelenburg, prone, jack-knife, or lateral.

4. Replicate the study with a larger sample population.

Conclusion

This study attempted to show statistical significance in supine arterial saturation values measured by pulse oximetry between 14 obese subjects and 14 nonobese subjects during position change from sitting to supine. The hypothesis stated there would be no difference between oxygen saturation values. Results indicated that both groups had a statistical change in the sitting SpO_2 measurement and the immediate supine SpO_2 measurement. The obese group continued to have statistical changes in the SpO_2 values up to three minutes supine. Therefore, the results from this study rejected the hypothesis.

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Appendix A

Appendix A

Name:_____
(print)

Record #_____

Data Collection Tool
Pulse oximetry/Position changes

Anna M. Klosterman RRNA-II
Department of Nurse Anesthesia
Virginia Commonwealth University

AGE: _____ yrs. SEX: F or M ASA: I or II

HEIGHT: Ft_____ Ins_____
Inches_____
Cm_____

WEIGHT:_____lbs
_____kgs

IDEAL BODY WEIGHT _____

% IDEAL BODY WEIGHT _____

GROUP PLACEMENT: Study group: Subject # _____
Control group: Subject # _____

SpO_2 MEASUREMENTS:

1. Sitting upright on stretcher: _____
2. Immediately after position change: _____
3. 1 minute after position change: _____
4. 2 minutes after position change: _____
5. 3 minutes after position change: _____
6. 4 minutes after position change: _____
7. 5 minutes after position change: _____
8. 6 minutes after position change: _____
9. 7 minutes after position change: _____
10. 8 minutes after position change: _____
11. 9 minutes after position change: _____
12. 10 minutes after position change: _____

Appendix B

Appendix B

Sample characteristics of obese group

n	Height (Cm)	Weight (Kg)	IBW (Kg)	% of IBW	Age	Gender
1	162.50	117.72	57.50	204.740	43	F
2	160.00	139.09	55.00	252.890	23	F
3	156.20	115.90	48.75	204.000	42	F
4	165.00	124.00	60.00	206.000	42	F
5	167.50	136.36	67.50	222.220	32	M
6	160.00	191.00	55.00	347.930	49	F
7	168.75	153.18	63.75	240.290	30	F
8	150.00	121.36	45.00	269.700	52	F
9	176.25	145.45	71.25	204.150	29	F
10	163.75	150.00	61.25	255.320	28	F
11	158.75	96.36	52.50	183.750	36	F
12	167.50	142.72	62.50	228.360	37	F
13	166.25	124.09	61.25	202.590	26	F
14	168.75	188.18	63.75	295.190	37	F
Minimum				183.750	23	
Maximum				347.930	52	
Mean				236.938	36.143	
SD				44.585	8.690	

Appendix C

Appendix C

Sample characteristics of nonobese group

n	Height (Cm)	Weight (Kg)	IBW (Kg)	% of IBW	Age	Gender
15	167.50	56.82	62.50	90.910	39	F
16	155.00	48.64	50.00	97.270	31	F
17	165.00	63.64	65.00	97.900	28	M
18	170.00	68.18	70.00	97.400	31	M
19	165.00	54.55	60.00	90.210	26	F
20	160.00	59.09	55.00	107.440	32	F
21	160.00	52.27	55.00	95.040	29	F
22	160.00	65.91	55.00	119.830	36	F
23	172.50	67.27	67.50	99.660	39	F
24	162.50	60.91	58.75	105.930	31	F
25	180.00	79.55	80.00	106.060	30	M
26	160.00	52.27	55.00	95.840	28	F
27	155.00	56.82	50.00	113.640	26	F
28	160.00	56.82	55.00	103.350	35	F
Minimum				90.210	26	
Maximum				119.830	39	
Mean				101.403	31.500	
SD				8.487	4.292	

Appendix D

Appendix D

Raw data of SpO_2 measurements

	Measurements											
n	1	2	3	4	5	6	7	8	9	10	11	12
1	98	98	99	99	98	98	97	98	98	98	97	97
2	100	99	97	97	96	96	96	95	97	96	96	95
3	100	97	98	99	99	99	99	99	99	99	100	99
4	98	99	100	100	100	100	100	100	98	99	100	100
5	99	99	99	98	98	97	96	99	96	98	98	97
6	98	92	94	95	97	95	94	94	95	94	95	96
7	99	96	98	99	98	98	98	98	99	98	98	99
8	100	99	99	100	99	99	100	99	98	100	99	99
9	100	100	100	100	100	100	100	99	100	96	98	100
10	100	100	99	100	100	100	100	100	100	100	100	100
11	99	94	96	97	98	98	99	98	97	98	98	98
12	97	94	97	98	97	97	97	96	97	97	97	98
13	98	95	97	98	100	99	99	100	100	99	100	100
14	100	99	100	100	100	100	100	100	100	100	100	100
15	98	98	99	99	98	98	98	98	98	97	97	98
16	100	97	99	99	100	100	100	100	100	100	99	100
17	100	99	98	98	99	99	98	98	99	99	99	98
18	97	96	97	96	95	96	96	97	97	98	97	97
19	100	99	100	99	99	99	99	98	98	98	99	99
20	100	99	100	100	100	100	100	99	100	100	100	100
21	100	100	99	100	99	99	99	98	97	98	99	98
22	98	96	97	96	97	97	98	98	99	98	97	98
23	97	97	98	98	99	99	99	99	99	98	99	99
24	100	99	99	100	100	100	99	100	100	99	100	100
25	100	100	99	99	98	100	99	100	100	100	100	100
26	100	100	100	100	100	99	100	100	100	100	100	100
27	99	99	99	98	99	100	100	99	100	99	99	99
28	100	98	100	100	100	99	100	100	100	100	100	100

Measurement number codes:

1 = sitting SpO_2
2 = SpO_2 immediately after position change to supine
3 = SpO_2 1 minute after position change
4 = SpO_2 2 minutes after position change
5 = SpO_2 3 minutes after position change
6 = SpO_2 4 minutes after position change
7 = SpO_2 5 minutes after position change
8 = SpO_2 6 minutes after position change
9 = SpO_2 7 minutes after position change
10 = SpO_2 8 minutes after position change
11 = SpO_2 9 minutes after position change
12 = SpO_2 10 minutes after position change

Vita