

How to Use This Book

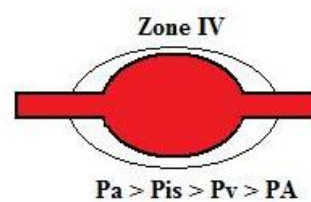
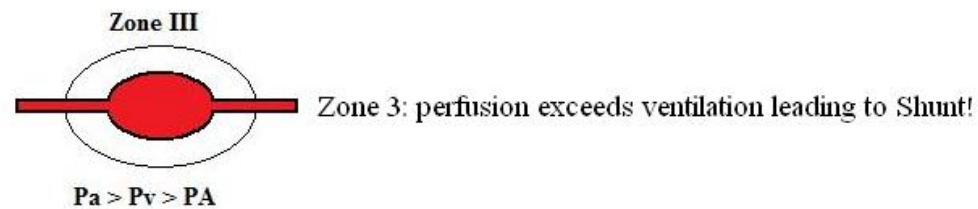
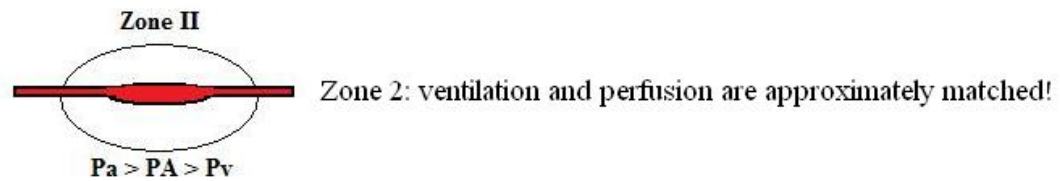
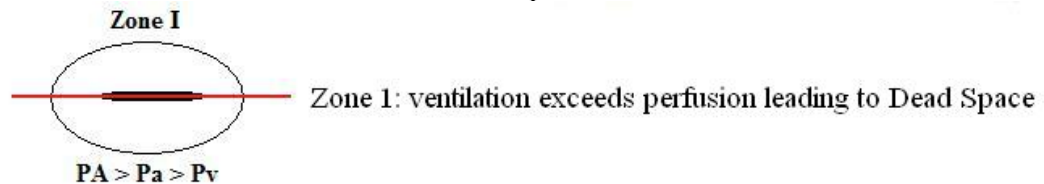
This book was produced after countless hours of studying, note taking, cross-referencing numerous sources, and multiple cups of coffee. This book truly contains all the essential information you *must* know. You may at times ask yourself if you really need to know some of the information contained in this book. The answer to that question is yes, without a doubt! This is not a book you can read once and regurgitate. Repeated exposure and understanding of the study material within these pages is essential. I would not claim that this book contains absolutely everything you will see on the certification exam. But I will promise that if you truly know all the material in this book, you will have the best opportunity to pass the board examination and will become a better physician in the process.

There are 85 chapters in this book. I would recommend that you review each chapter at least three times. Begin each day by reading a chapter and taking notes or making up practice questions based on each section of the chapter. If you resort to highlighting, you may find yourself highlighting entire pages so I would refrain from that specific study technique. At the end of your study session, review your notes or the study questions you've come up with, and then go back and review the contents of the chapter you read the day before. If you study one chapter per day, and review the book in its entirety three times, you will require approximately 255 days (that's about nine months!) so do not procrastinate. True understanding of this material is essential if you wish to pass the board certification examination.

Once you have passed the written examination, utilize the core knowledge in this book to prepare yourself for the oral board examination. If you would like to learn specific techniques in memory enhancement, oral presentation skills, or utilize practical cases designed to assess your knowledge and prepare you for the oral board examination I would recommend using *Preparing for the Anesthesia Oral Board Exam*, which can be obtained at www.BeyondAnesthesia.com. This book will teach you techniques that will help you prepare for and master the oral board examination as administered by the ABA. Now, let us get started...

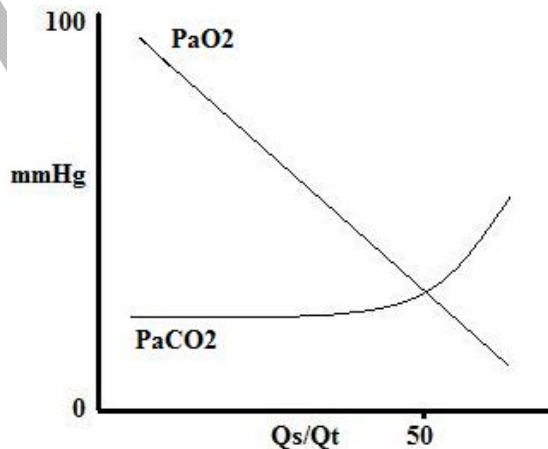
Pulmonary Physiology

1. In order to better understand pulmonary physiology, it is important to understand pulmonary blood flow and how it affects gas exchange. The image below illustrates the three major lung zones and the relative distribution of ventilation to perfusion in each zone:



2. Zone I is the upper most lung zone and it is the collapse zone where alveolar pressure (P_A) is greater than arterial pressure (P_a) and venous pressure (P_v). This is an example of dead space where the lung is ventilated but not adequately perfused. Zone II is the waterfall region where arterial pressure exceeds alveolar pressure, which in turn exceeds venous pressure. Zone III illustrates distention where both arterial and venous pressures exceed the alveolar pressure. Zone IV is the lowest region in the lung and shows arterial pressure to be the greatest, followed by interstitial pressure (P_{is}), then venous pressure, and finally alveolar pressure. This is an example of a “shunt” where the lung is perfused but not adequately oxygenated. In a sitting patient, ventilation is greatest in the upper lung field (zone 1) while perfusion is greatest in the lowest lung field (zone 3). This is very important since zone 1 essentially creates what is called “dead

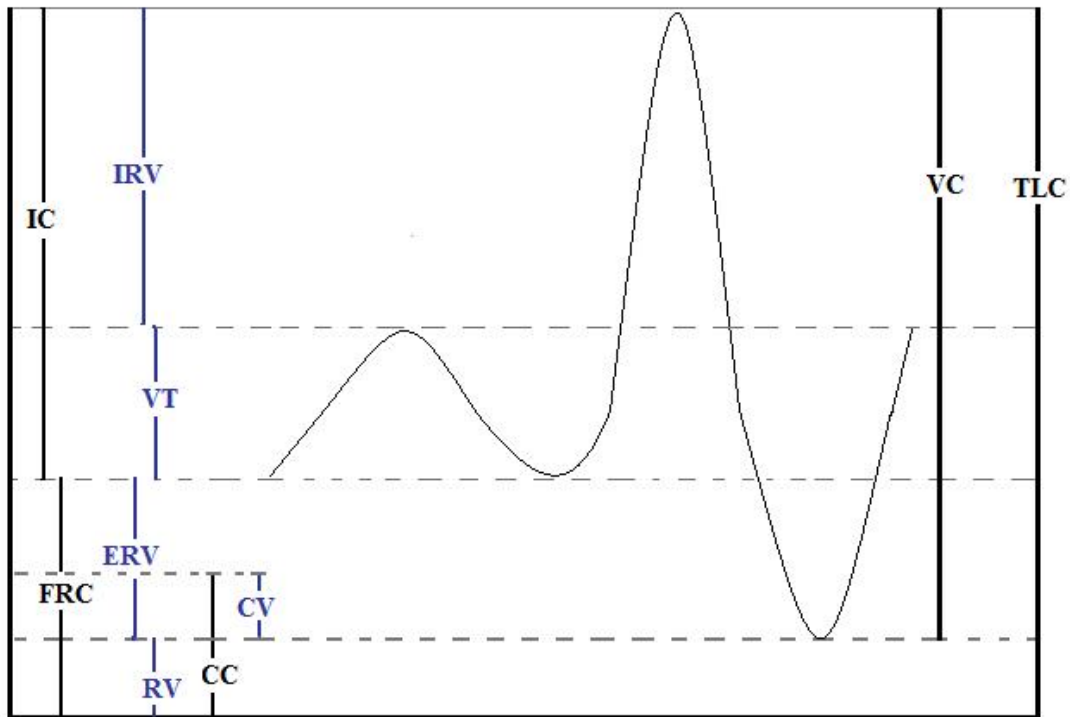
- space”, an area that is ventilated but not perfused. Zone 3 and 4 on the other hand create a “shunt”, an area that is perfused but not well ventilated.
3. Dead space (designated as V_d/V_t) is the fraction of the normal tidal volume that does not participate in gas exchange. Dead space is created when there are areas of the lung that are ventilated but not perfused (think pulmonary embolus). V_d/V_t is characterized by the equation $(PaCO_2 - PETCO_2) / PaCO_2$. Normal dead space is considered to be 0.3 L (300cc).
 - a. In general, any factor that increases pulmonary vascular resistance or increases alveolar pressure will increase pulmonary dead space volume. Some examples include:
 - i. Positive pressure ventilation ($\uparrow P_A$)
 - ii. PEEP/ CPAP ($\uparrow P_A$)
 - iii. Bronchodilation (\uparrow anatomic dead space)
 - iv. Low cardiac output (reduced pulmonary blood flow and increased PVR)
 - v. PE
 - b. Dead space also increases with age, anticholinergics (atropine), upright position, smoking, emphysema, and any factor that may decrease pulmonary perfusion including hypovolemia, hypotension, and hypothermia.
 4. Hypoxic pulmonary vasoconstriction (HPV) is a physiologic response adapted towards decreasing pulmonary shunting. As stated earlier shunting occurs when there is ventilation to perfusion mismatch. Any area that is perfused but not ventilated is a “shunt” whereas areas that are ventilated but not perfused are called “dead space”. An example of a shunt would be a pneumothorax (lung is perfused but not ventilated); while an example of dead space would be a pulmonary embolus (which is an area of the lung that is ventilated but not perfused).
 - a. HPV is reduced in the presence of PEEP, acidosis, alkalosis, hypocapnia, hypothermia, calcium channel blockers, high frequency ventilation, sodium nitroprusside, nitroglycerin, and isoproterenol. HPV is reversed by elements that cause distention of pulmonary vessels including elevated PA pressures, mitral stenosis, volume overload, hypothermia, vasoactive drugs, and pulmonary embolism. HPV is inhibited by volatile anesthetics as well as nitrous oxide.
 - b. HPV is increased if the mixed venous O_2 is equal or less than 30 mmHg.
 5. Shunt fraction (designated as Q_s/Q_t) is within 5-10% under normal circumstances. If Q_s/Q_t increases to above 30%, increases in FiO_2 will no longer increase the PaO_2 .
 - a. Normal pulmonary shunting is approximately 10%; this implies that 90% of the cardiac output participates in gas exchange. Some causes of pulmonary shunting including pneumothorax, bronchospasm, and right to left cardiac shunts.
 - b. As illustrated below, as PaO_2 decreases, the shunt fraction (Q_s/Q_t) increases. The $PaCO_2$ does not begin to increase until the shunt fraction reaches 50%.



- c. PaO_2 increases by 50 mmHg for each 10% increase in FiO_2 . Therefore, a FiO_2 of 0.2 is equivalent to a PaO_2 of 100 mmHg, while a FiO_2 of 1.0 (100% oxygen) is equivalent to a PaO_2 of 500 mmHg. Hence, in the absence of a shunt and with a normal A-a gradient, a patient on 100% oxygen should have a PaO_2 of 500 mmHg.
 - d. Since our lungs are not perfectly efficient there exists a “normal” Alveolar-arterial oxygen gradient (A-a gradient). This gradient is approximately 5-10 mmHg on room air and 20-30 mmHg when on 100% oxygen. Overall, one can approximate a normal A-a gradient to be $\frac{1}{4}$ of a patient’s age.
 - e. The A-a gradient is important when determining the etiology of hypoxemia in a patient. Hypoxemia due to hypoventilation and low FiO_2 do not affect the A-a gradient. On the other hand, ventilation-perfusion mismatch, right to left shunting, and decreased DLCO all cause an increase in the A-a gradient.
 - i. The A-a gradient is also increased in patients undergoing a general anesthetic due to the following reasons:
 - 1. Decreased cardiac output leads to decreased lung perfusion, resulting in greater dead space ventilation and V/Q mismatch.
 - 2. Decreased FRC under general anesthesia leads to decreased pulmonary compliance and ventilation-perfusion mismatch. (The decrease in FRC is greatest 3-5 days post surgery and lasts for 10-14 days postoperatively).
 - 3. Decreased lung and chest wall compliance further aggravates V/Q mismatch.
 - 4. Increased airway resistance contributes to ventilation and perfusion mismatch.
 - 5. Paralysis worsens V/Q mismatch. In a paralyzed supine patient, ventilation is greatest anteriorly in the non-dependent portion of the lung (due to positive pressure ventilation) while perfusion is greatest posteriorly in the dependent portion of the lung (due to gravity). On the other hand an awake spontaneously breathing supine patient has greater ventilation and perfusion in the dependent portion of the lung.
 - ii. Other factors increasing the A-a gradient include: (mnemonic: Smoking Adolescents Avoid Cops)
 - 1. Smoking
 - 2. Advanced age
 - 3. Atelectasis (obesity, pregnancy, hypoventilation)
 - 4. COPD
6. Pulmonary blood flow is dependent on gravity, alveolar pressure, and pulmonary vascular resistance. Perfusion is greatest in the dependent regions of the lung where the gravitational pull is strongest. Hence a supine patient will have a different perfusion/ventilation ratio in the same region of the lung as compared to a patient in the sitting posture. If alveolar pressure (P_A) exceeds arterial pressure (P_a) as occurs in Zone 1, the increase in pulmonary vascular resistance diverts blood flow to areas of lesser alveolar pressure (Zones 2 and 3). Since increased pulmonary vascular resistance results in a reduction in pulmonary blood flow it is important to know some of the physiologic factors that can increase pulmonary vascular resistance: (mnemonic: Hairy Armpits Have Lousy Hygiene):
- i. Hypoxia: leading to hypoxic pulmonary vasoconstriction
 - ii. Acidosis
 - iii. Hypercarbia: leads to reactive vasoconstriction
 - iv. Low cardiac output: low vascular flow increases vascular resistance
 - v. Hypothermia
7. The distribution of pulmonary ventilation is highly dependent on gravity, pulmonary compliance, and airway resistance. As one moves distally in the pulmonary tree, the cross sectional area

increases rapidly and resistance to flow (airway resistance) decreases. Pulmonary compliance describes the change in volume that occurs as a change in pressure is applied to the lungs or the chest wall. Compliance can be affected by factors such as obesity, and pulmonary fibrosis. Gravity is a major determinant of pulmonary ventilation. Irrespective of patient position (supine or lateral decubitus), during spontaneous ventilation the alveoli in the dependent pulmonary region are more compliant (experience a greater change in volume per unit change in pressure) as compared to apical alveoli and hence a greater fraction of the ventilation occurs in the dependent portion of the lung. On the other hand, in an anesthetized but spontaneously breathing patient, atelectasis and hypoventilation will favor the non-dependent portion of the lungs. Similarly, during mechanical ventilation compliance and resistance favor ventilation of the non-dependent regions of the lung.

8. Airway resistance and pulmonary compliance also affect the work of breathing. The work of breathing is increased with increased airway resistance and decreased pulmonary compliance. Hence, patients with obstructive lung disease (increased airway resistance) will increase their work of breathing by rapid shallow breaths, while those with a restrictive lung disease (poorly compliant lung or chest wall) will increase their work of breathing by taking slow deep breaths.
9. Spirometry is a technique used to assess pulmonary function. Spirometric measurements help determine lungs capacities and volumes. The image below indicates the relationship of some of these parameters:



- a. Closing capacity (CC) consists of closing volume (CV) and residual volume (RV). Closing capacity is the volume where small airways begin to close and collapse. Shunting occurs when closing capacity exceeds functional residual capacity (FRC).
- b. Closing capacity increases with chronic bronchitis, left ventricular failure, obesity, smoking, surgery, and extreme age (mnemonic: CLOSE).
- c. FRC is the point at which the elastic recoil of the lung equals the recoil of the chest wall. FRC is increased in the elderly, severe emphysema, application of PEEP, and asthma due

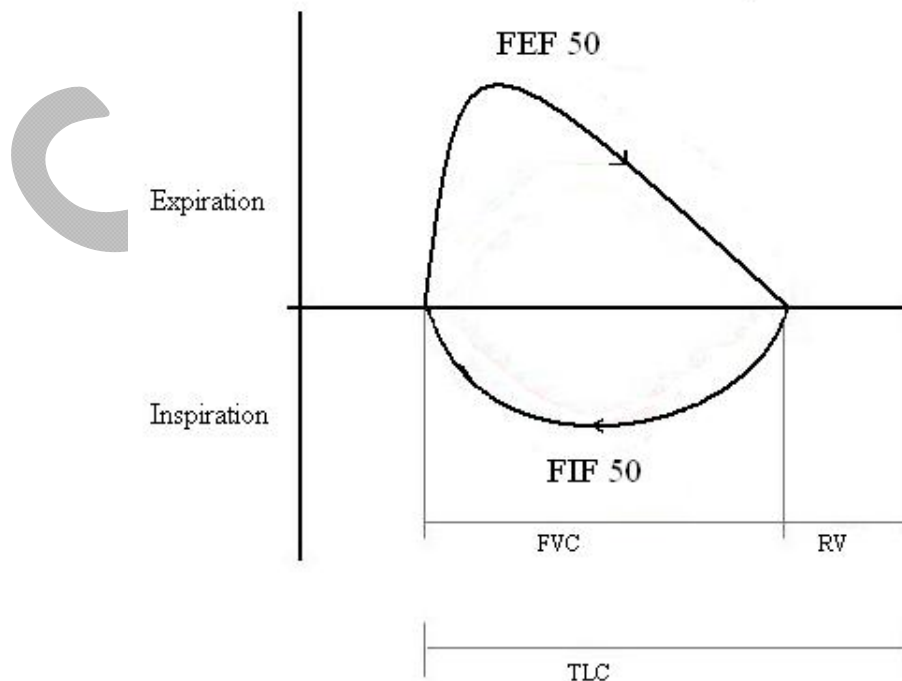
to air trapping. FRC is decreased in neonates, general anesthesia, supine posture, obesity, ascites, and pregnancy (mnemonic: NG SOAP).

- i. Abdominal and thoracic surgery decrease FRC for several days post operatively.
 - ii. Under anesthesia, loss of thoracic muscle tone decreases elastic recoil of the chest wall and results in a decrease in FRC. This reduction is worsened in the supine position as the relaxed diaphragm migrates cephalad.
 - iii. Note that FRC and RV are the two values not measured by spirometry.
- d. A sensory block above the T₆ level can lead to a decrease in expiratory reserve volume (ERV) and hence a reduction in FRC.
 - e. Normal vital capacity is 50cc/kg. Normal pulmonary compliance (change in volume per applied pressure) is 200cc/cm H₂O.
 - f. Obstructive lung diseases (emphysema, asthma, chronic bronchitis) are characterized by decreased flow where FEV₁ is decreased while FVC remains largely normal resulting in a decreased ratio of FEV₁/FVC. Restrictive lung diseases (pulmonary fibrosis, obesity, pregnancy) are characterized by a decrease in volume but flows remain the same hence the ratio of FEV₁/FVC remains normal.
 - g. The table below indicates some normal values associated with each pulmonary parameter:

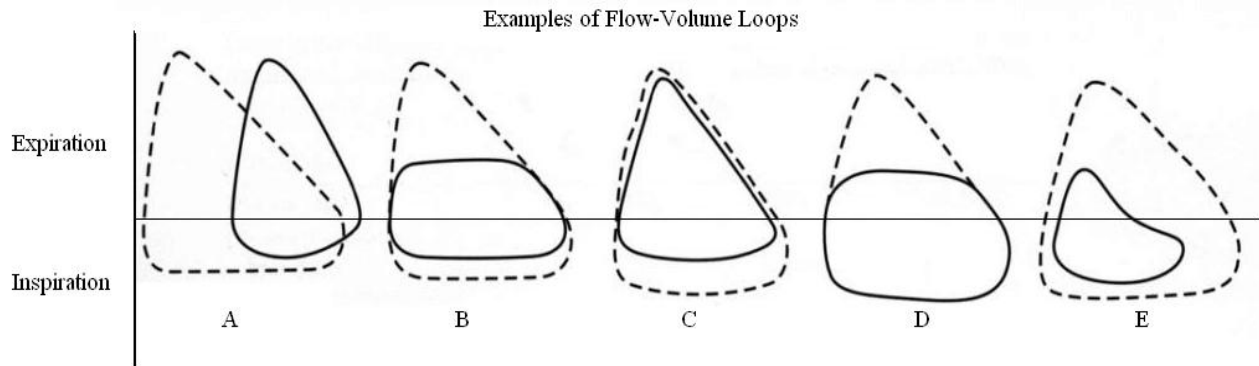
Test	Average "normal" value
TLC	5-6 (L)
VC	4-5 (L)
FEV ₁	3-4 (L)
% FVC ₁	80
FRC	2-4 (L)

10. Flow-volume loops are utilized when one needs to determine the presence and severity of airway obstruction. The image below illustrates a normal flow-volume loop:

Normal Flow-Volume Loop



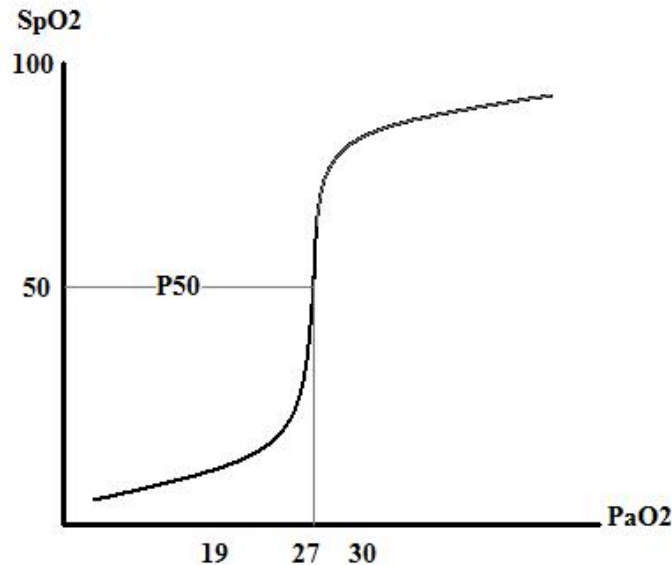
- a. As illustrated by the image, the patient performs a forced vital capacity maneuver in expiration and inspiration which allows the following spiograph to be charted. The y-axis illustrates flow rate in liters/sec while the x-axis illustrates volume in liters. The pleural pressure is negative in the inspiratory phase and positive in the expiratory phase.
- b. The following flow-volume loops illustrate some of the disease processes that can be determined through this method. The dashed lines illustrate a normal loop while the solid lines show the abnormal curve:



- c. Can you determine which disease process is shown in each of the above flow-volume loops?
 - i. Image (A) illustrates a limited inspiratory and expiratory phase with a shift to the right indicating a reduced TLC. This is an example of a restrictive lung disease such as pulmonary fibrosis, scoliosis, or obesity.
 - ii. Image (B) illustrates a reduced inspiratory and expiratory phase without changes to the TLC. This is indicative of a fixed defect such as a tracheal web, stricture, or goiter and is the most common flow-volume loop.
 - iii. Image (C) illustrates a reduced inspiratory phase with a normal expiratory phase. If you consider a “ball-valve” effect where the ball obstructs the opening once pressure is applied to it, then one can deduce that this is an extrathoracic defect such as sleep apnea, or vocal cord palsy.
 - iv. Image (D) illustrates a normal inspiratory phase followed by a reduced expiratory phase. Again, the “ball-valve” is causing obstruction in the expiratory phase hence this is most likely an intrathoracic defect such as a bronchial tumor.
 - v. Image (E) illustrates a severe reduction in both phases of the breathing cycle and a reduction in the FVC. This is indicative of a diffuse obstructive defect such as COPD or asthma.
11. Oxygen delivery to tissues is determined by cardiac output, total oxygen content of blood, and the type of hemoglobin present (ex: fetal hemoglobin levels are increased in sickle cell anemia, and β thalassemia). As you may recall, cardiac output is demonstrated by the formula heart rate multiplied by stroke volume. Oxygen content (CaO_2) is influenced by the number of hemoglobin molecules and their saturation, recall the formula: $(0.003 \times PaO_2) + (1.34 \times Hgb \times SaO_2)$. Although oxygen exists in blood as a dissolved and a bound form, the bound hemoglobin has the greatest contribution to oxygen content. The first portion of the above formula represents the dissolved portion of oxygen in blood. As atmospheric pressure increases, and temperature decreases, more oxygen is forced into solution (dissolved). The second portion of the equation represents the bound portion of oxygen and illustrates that each gram of hemoglobin carries 1.34 ml of oxygen when fully saturated which is why the oxygen saturation expressed as a percentage is included in the formula. Hence, one way to increase oxygen delivery is to increase cardiac output. Another way is to increase the number of hemoglobin molecules and their saturation by supplying 100% oxygen. This concept can be carried forward when attempting to determine the factors which influence mixed venous oxygen saturation. If one thinks critically and understands

the physiology of our cardiovascular system then the following conclusion can be derived: the venous oxygen saturation is determined by the two factors influencing supply which are cardiac output and oxygen content, and the factor influencing demand which is oxygen consumption (which can be affected by a variety of things including metabolic rate).

12. It is essential to understand the behavior of hemoglobin with respect to oxygen carrying capacity as demonstrated by the oxyhemoglobin dissociation curve (OHDC).



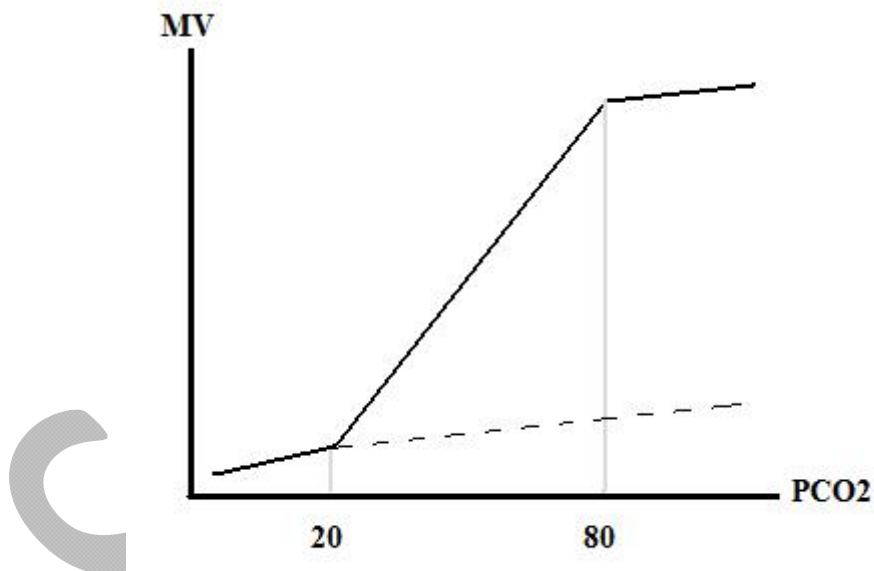
- The above diagram illustrates that at a partial pressure of 27 mmHg, the hemoglobin molecule will be 50% saturated. This is referred to as the “P₅₀”. The P₅₀ for neonates is 19 mmHg indicating a left shift of the OHDC, and the P₅₀ for children is 30 mmHg which indicates a right shift of the OHDC.
- OHDC shifts to the right when there is an increase in PCO₂, H⁺ (acidosis), temperature, 2,3 DPG (seen in anemia, cirrhosis, or increased altitude), and sickle cell hemoglobin. If there is a right shift of the OHDC then at the same partial pressure of oxygen, the hemoglobin molecule will be less saturated (oxygen molecules will have been released from the binding sites). Another way to think about this would be to say that the hemoglobin molecule would have to have a higher PaO₂ in order to maintain the same level of saturation. In short, remember “Right Releases!”
- OHDC shifts to the left with decreases in PCO₂, H⁺, temperature, 2,3 DPG, and in the presence of fetal hemoglobin, methemoglobin, and carboxyhemoglobin. A left shift results in the hemoglobin molecule maintaining its binding to the oxygen molecules and maintaining its saturation despite a lower partial pressure of Oxygen. Hence, “Left Lasts!”
- Chronic anemia can affect oxygen delivery by causing an elevation in 2,3 DPG, P₅₀, cardiac output (due to tachycardia), coronary blood flow (due to decreased viscosity), and oxygen extraction (lowering mixed venous O₂).
- The Bohr effect states that the OHDC shifts with changes in PCO₂.
- The Haldane effect states that deoxygenated hemoglobin can carry more CO₂.
- The factors that affect the OHDC are presented in the table below. Note that a “raise” in the value of these factors creates a right shift and that “lowering” of the factors creates a left shift.

Left shift- "lower"	Right shift- "raise"
↓H ⁺ concentration	↑H ⁺ concentration
↓Temperature	↑Temperature
↓2,3 DPG	↑2,3 DPG
Fetal hemoglobin	Pregnancy, chronic anemia
Carboxyhemoglobin and methemoglobin	Sickle cell hemoglobin

- h. One shortcut to remember when considering the partial pressure of oxygen with respect to hemoglobin oxygen saturation are the numbers "456789" which stand for...

PaO ₂	SpO ₂ %
40	70
50	80
60	90

13. The CO₂ response curve is also important to understand since it too affects the cardiovascular and the respiratory system:



- The CO₂ response curve is linear when the PCO₂ is between 20 to 80 mmHg. As the PCO₂ increases above 40 mmHg, the minute ventilation (MV) also increases until the PCO₂ reaches 100 mmHg. Above 100 mmHg CO₂ acts as a respiratory depressant and actually decreases respiratory drive. Very high levels of narcotics as well as volatile anesthetics depress the response curve such that elevations in PCO₂ create a very small change in MV (illustrated by the dashed line above).
- The CO₂ response curve shifts to the left (implying increased sensitivity to CO₂) in hypoxia, anxiety, elevated intracranial pressure, cirrhosis, and acidemia. The curve shifts to the right (decreased sensitivity to CO₂) in metabolic alkalosis, during normal sleep, when influenced by drugs such as narcotics, catecholamines, salicylates, and aminophylline, and when there is denervation of the peripheral chemoreceptors.

14. Peripheral chemoreceptors adjust the ventilatory response to hypoxemia. When the PaO₂ falls below 60 mmHg (equivalent to an oxygen saturation of 90%) the peripheral chemoreceptors in the carotid and aortic bodies initiate an increase in minute ventilation in order to increase the partial pressure of oxygen in the blood. This is not to be confused with the carotid baroreceptors which respond to changes in aortic blood pressure.
15. Central receptors respond to changes in pH and PaCO₂. Once a change is detected, within a minute the central receptors create a response which includes increases in respiratory rate and tidal volume in order to restore normal physiologic parameters. This response is modulated via the medulla and cranial nerves IX and X.
16. Pulmonary hypertension is defined as pulmonary artery pressure of 30 mmHg or greater. Pulmonary hypertension can occur due to a multitude of etiologies including:
 - a. Increased pulmonary blood flow (ex: left to right shunt)
 - b. Increased pulmonary vascular resistance (PVR). Recall that $PVR = (\text{mean PA} - \text{PCWP}) / \text{CO} \times 80$ with normal values being 100-150 dynes/sec/cm. Several factors can increase the PVR and should be stored to memory. These include hypoxia, hypercarbia, acidosis, and damage or destruction of pulmonary vessels (mnemonic: Hunters Have Active Dogs). Vascular damage can occur secondary to:
 - i. Increased valvular pressures due to mitral stenosis or regurgitation.
 - ii. Pulmonary embolus including fat, air, and amniotic fluid emboli.
17. Treatment for pulmonary hypertension relies on understanding and addressing the underlying etiology. Assuming that hypoxia, and hypercarbia have been addressed, further treatment for pulmonary hypertension includes vasodilators such as inhaled nitric oxide (20-40 ppm), calcium channel blockers, nitroglycerin, and PGE₁ agonists such as alprostadil.
18. Pulmonary hypertension can lead to pulmonary edema. Pulmonary edema also occurs due to cardiogenic and non-cardiogenic causes. Cardiogenic etiologies include factors that increase capillary pressures such as mitral stenosis, congestive heart failure, and fluid retention. Non-cardiogenic etiologies include factors that increase capillary leak including negative pressure pulmonary edema, aspiration, ARDS, DIC, burns, sepsis, neurogenic edema, decreased oncotic pressure, and lymphatic obstruction.
 - a. Cardiogenic pulmonary edema can be diagnosed via elevated pulmonary capillary wedge pressures (PCWP), pink frothy sputum and secretions, and patchy infiltrates on chest x-ray.
 - b. Non-cardiogenic pulmonary edema can be diagnosed via low PCWP, and consideration of underlying etiologies such as sepsis, DIC, smoke inhalation, and massive blood transfusions.
 - c. Treatment includes 100% oxygen, application of PEEP, preload reduction with vasodilators such as nitroglycerin (NTG), diuretics, and enhancement of cardiac output and contractility using inotropes.
19. How does smoking affect general anesthesia?
 - a. Smoking increases airway irritability and secretions; it also raises closing capacity above FRC which results in shunting and hypoxia. Furthermore, nicotine increases sympathetic tone.
 - b. Smoking cessation should take place 8 weeks (2 months) prior to the administration of a general anesthetic for maximal benefits. If stopped one day prior to the operation it will decrease the carboxyhemoglobin levels and begin a correction of the left shift of the OHDC. Cessation one week prior to the anesthetic can aid in diminishing of the pulmonary secretions.
 - c. Carboxyhemoglobin in smokers can be as high as 10-15% (as identified by cooximetry since a dual wave pulse oximeter will interpret carboxyhemoglobin as oxyhemoglobin).
20. Under normal physiologic conditions 250 cc/min of oxygen is absorbed from the alveoli while 200 cc of CO₂ is produced per minute. Apneic oxygenation is a method through which a

continuous inflow of oxygen into the lungs provides for passive exchange of oxygen for carbon dioxide. As the alveolus fills with CO₂, less space is left in the alveolus to be occupied by oxygen. Hence, apneic oxygenation is limited by the starting level of PaCO₂ and the amount of PaCO₂ produced. PaCO₂ increases by 6 mmHg for the first minute and 3 mmHg each subsequent minute. Furthermore, incomplete de-nitrogenation and low FRC can further decrease the available reservoir for oxygen. Eventually, as the PaCO₂ levels rise, active ventilation will need to take place or cardiac arrhythmia ensues.

SAMPLE

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