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Clinical Anatomy and Physiology of Ventilation

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The respiratory system plays two major roles in the body, for the introduction of oxygen and the removal of carbon dioxide. This paper will give an overview of respiratory anatomy and physiology, with an emphasis on information clinically relevant to acute airway control and ventilatory support.

CLINICAL ANATOMY OF THE AIRWAY

Air enters the body through the nares and mouth. Through the nares, air passes into the nasal cavity, which is separated vertically into right and left halves by the nasal septum. In each nasal cavity, there are shelf-like bony projections called turbinates. They add surface area to the mucous membranes that line the airway and create turbulence in the airflow entering the nose. The mucous membranes are covered with tiny hairs, called cilia, which slowly and continuously move a blanket of mucous from the lower airway up to the nares and mouth. As air is inhaled, it impacts the various mucous membrane covered surfaces of the airway. This allows dirt, germs and other foreign materials to be trapped in the mucous blanket and expelled. The large surface area of the upper and lower airways readily exchange heat and moisture with the inspired air. By the time air reaches the alveoli, the air is at body temperature, 100% humidified, and is essentially sterile.

After air passes through the nasal cavity, it enters a somewhat vertical chamber called the pharynx, which extends from the back of the nasal cavities down to the larynx. The upper part of the pharynx that joins the nasal cavities is called the nasopharynx. The section which joins the oral cavity is the oropharynx. The lower section extending down towards the larynx is the hypopharynx (Figure 1).

The anatomy of the hypopharynx is important in consideration of esophageal and endotracheal intubation methods. Notice that the larynx and the trachea are on the midline and anterior in the neck. The esophagus and its opening in the hypopharynx are also in the midline, but posterior to the trachea and larynx. Therefore, when an esophageal airway or nasogastric tube is inserted, it should be kept toward the posterior surface of the airway. Inserting the esophageal tube and letting it slide against the posterior hypopharyngeal wall on the midline will bring it to the esophageal opening. In contrast, the endotracheal tube needs to be kept towards the anterior of the neck. However, entry of the endotracheal tube in the opening of the trachea, called the glottis, requires passage around the epiglottis. The epiglottis acts as a door to the glottis, which is closed during swallowing or gagging to prevent entry of food or foreign materials into the lower airway. If an endotracheal tube or tracheal suction catheter is inserted too anteriorly, they may lodge in the vallecula (Figure 1). If esophageal or tracheal devices stray from the midline, they can be caught on either side in the pyriform sinuses. Forceful efforts can perforate these tissues, potentially leading to serious bleeding and/or subcutaneous emphysema.

The larynx houses the vocal apparatus, with the various structures labeled in Figure 2. It is important to remember that the glottic opening between the vocal cords is the narrowest point along the upper airway. Pieces of food or foreign material that can obstruct the airway often become lodged here. Gag and other airway protection reflexes can tighten up the airway to prevent material from falling deeper. The vocal cords can tighten to close off the airway completely.

When an upper airway obstruction cannot be removed by simple means (i.e. back blows, abdominal thrusts, or laryngoscopy with forceps extraction), it may be necessary to make a surgical opening below the vocal cords to allow ventilation. This is usually performed in the field at the level of the cricothyroid membrane (Figure 1).

The cricoid cartilage is a rigid and complete ring, unlike the tracheal cartilage rings that are open on their posterior surfaces. Applying posteriorly directed pressure with the fingers on the the cricoid cartilage, the airway remains open and is displaced posteriorly. This has two advantages. It can sometimes make visualization of the vocal cords easier during laryngoscopy. It can also pinch the esophagus closed, as it becomes compressed between the cricoid cartilage and the spine. This can prevent regurgitation and prevent the entry of air into the stomach during ventilation without an endotracheal or esophageal airway adjunct (1-3).

The trachea runs from the larynx to the carina - the point where the trachea bifurcates into the right and left mainstem bronchi. The right divides off the trachea without as much angulation as does the left. Consequently, if an endotracheal tube is inserted too deep, it usually ends up in the right mainstem bronchus and only the right lung may be ventilated. Each of the mainstem bronchi divide into major bronchi that serve anatomically distinct lobes of the lung. Good technique in lung auscultation requires listening to each of these lobes.

As the bronchi in each lobe divide into smaller and smaller airways, there are lesser proportions of cartilage in their walls and more smooth muscle. Thus, bronchoconstric-

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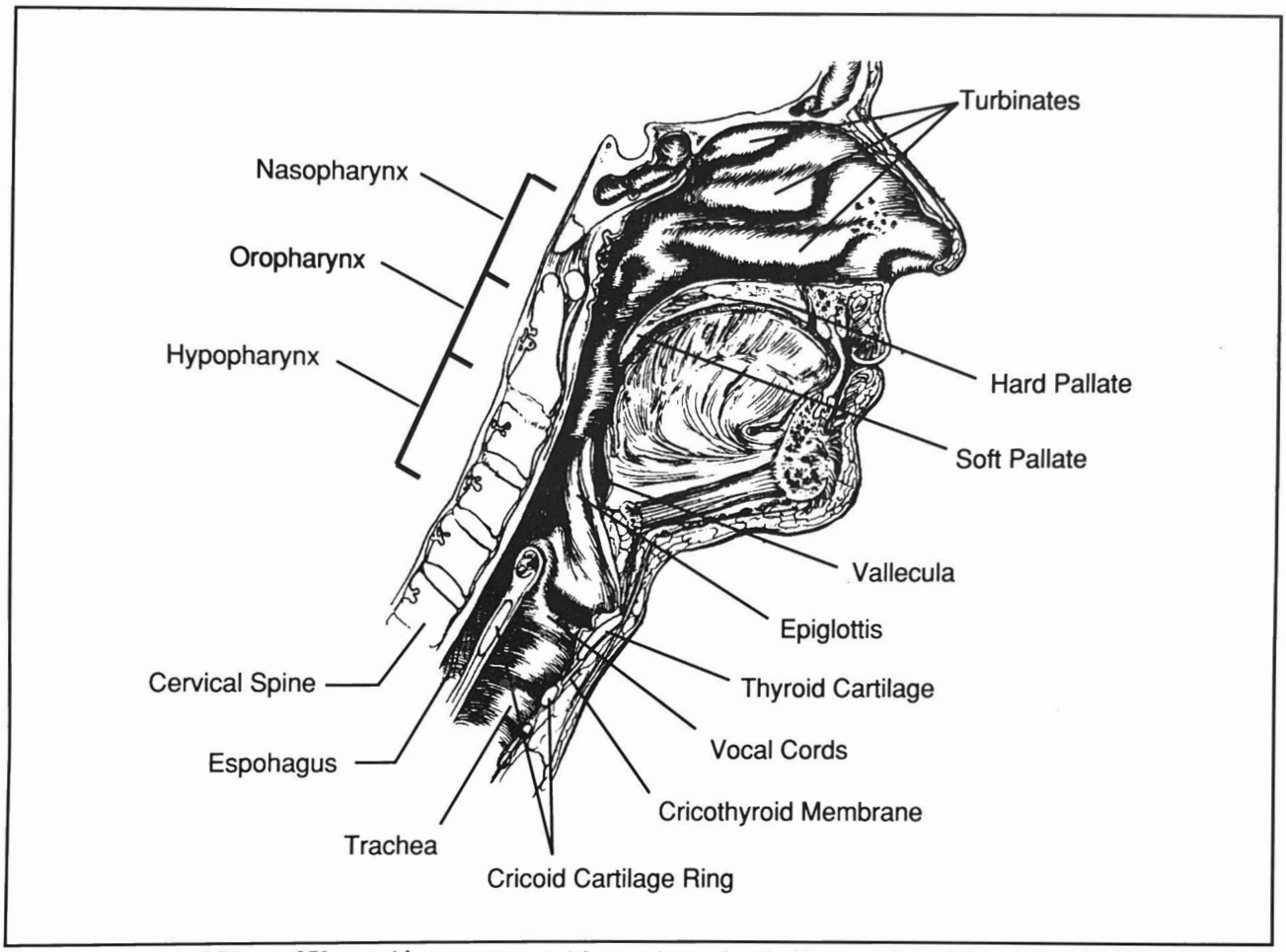

Figure 1 - Saggital View of Upper Airway - (Adapted from and reproduced with permission - Schlossberg L, Zuidema GD: *The Johns Hopkins Atlas of Human Functional Anatomy.* Johns Hopkins University Press, Baltimore 1977.)

tion has its greater effects on the smaller bronchial passages. Like the stems in a cluster of grapes, the terminal bronchiole enters a cluster of alveoli. The sac-like alveoli are thin membrane structures surrounded by a dense network of pulmonary capillaries (Figure 3). This is where the business of oxygen and carbon dioxide exchange between the air and blood takes place.

MECHANICS OF BREATHING

Inhalation

Inhalation is an active muscular process. The primary muscle of inhalation is the diaphragm. It is a dome-shaped muscle stretched across the lower end of the thorax. When the diaphragm contracts, it pulls downwards towards the abdomen. This enlarges the vertical dimensions of the chest cavity, creating a potential vacuum that can draw air into the lungs. However, the vacuum must be inside the lung, not just the chest cavity. Therefore, the outer surface of the lung must cling to the inner surface of the chest cavity, including the diaphragm. Thus, when the chest walls expand, the lungs expand with it to create the vacuum (negative pressure) that brings in the air.

The clinging of the outer surface of the lung to the inner surface of the chest cavity is facilitated by the pleural membranes. There are two pleural membranes in the chest, for the right and left sides, respectively. The point where a mainstem bronchus penetrates its respective pleural membrane is called the hilum.

Each pleural membrane covers an entire lung and then folds on itself at the hilum to line the respective hemisphere of inner chest wall. The portion of the pleural membrane that covers the lung is called the visceral pleura. The part that lines the chest wall is the parietal pleura. The parietal and visceral portions of the pleural membrane normally cling to each other because of high surface tension in the very small volume of lubricating pleural fluid between their surfaces. Thus, as the chest wall expands, so does the lung. If a wound

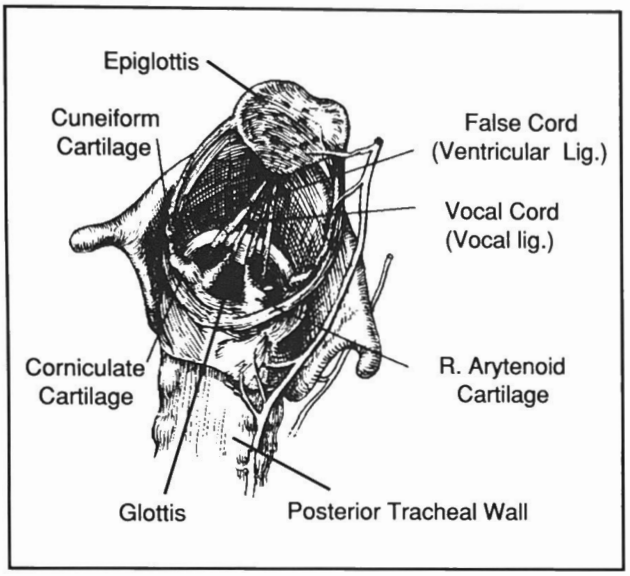

Figure 2 - Vocal Apparatus - (Adapted from and reproduced with permission - Schlossberg L, Zuidema GD: *The Johns Hopkins Atlas of Human Functional Anatomy.* Johns Hopkins University Press, Baltimore 1977.)

on the pleural membrane allows air to get in the potential space between the parietal and visceral layers, called the pleural space, this is called a pneumothorax.

Besides the vertical expansion of the chest cavity caused by the diaphragm, the chest can expand in its anterior-posterior dimension. This is facilitated by the ribs. Each pair of ribs hinge on the spine and join together anteriorly on the sternum or costal margins. This is analogous to a series of bucket handles, all joined in the middle. At rest, the ribs are angled slightly inferior. With inspiration, they swing out anteriorly and superiorly to enlarge the chest cavity. This normally occurs as a passive consequence of lung expansion pushing out against the ribs. However, with the more forceful inhalation that occurs with respiratory distress or exercise, the so-called accessory muscles of inhalation lift the ribs as an active muscular effort. The intercostal muscles can pull the ribs together, raising the angle and extending the chest wall outwards. The sternocleidomastoid muscles on the sides of the neck attach to the sternum, pulling it and the attached ribs with it up and out during its contraction. Observing effort by the intercostal muscles or the sternocleidomastoid muscles during inspiration is a helpful clinical indicator of respiratory distress.

Exhalation

Exhalation is normally a passive activity. The elastic recoil of the lung tissues and muscles of the chest wall forces out air in an amount generally equal to the preceding inhaled volume. In respiratory distress or exercise, exhalation can become an active process.

The primary muscles for exhalation are the abdominal muscles. By contracting them, the contents of the abdomen are pushed up against the diaphragm to force air from the lung. Additional effort in exhalation may be provided by intercostal muscles, separate from those used for inhalation. Just as utilization of the accessory muscles of inhalation indicates respiratory distress, so does activity with the accessory muscles of exhalation.

VENTILATORY MEASUREMENTS

Airway Resistance

When air is drawn into or out of the lungs, there is friction between the air and the walls of the airway that is referred to as airway resistance. Airway resistance depends on three factors - airway radius, airway length, and airflow velocity[1].

The airway radius, assuming a cylindrical airway, is inversely proportional to resistance. The larger the airway radius, the lesser the resistance[2]. This is why the largest possible size endotracheal tube should be used during intuba-

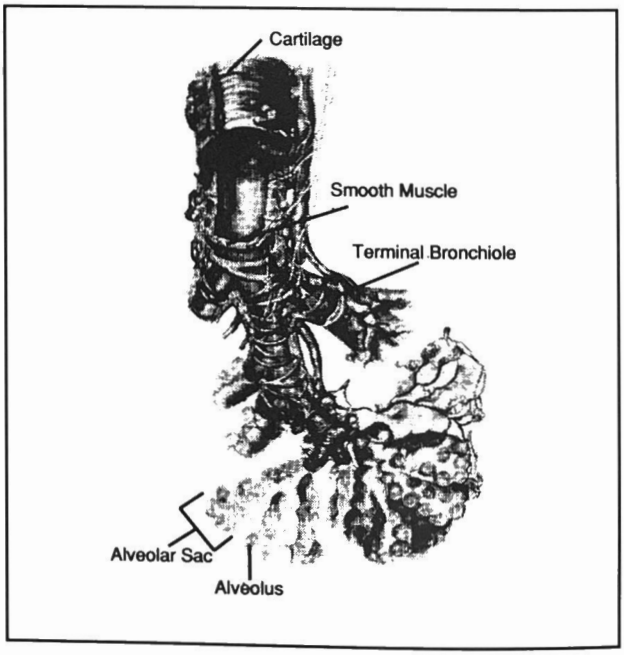

Figure 3 - Terminal Airway Structures - (Adapted from and reproduced with permission - Schlossberg L, Zuidema GD: *The Johns Hopkins Atlas of Human Functional Anatomy.* Johns Hopkins University Press, Baltimore 1977.)

1 The mathematical relationship between these factors applies not only to air, but to any gas or liquid, as shown in the following equation: Resistance =8 x viscosity x length/(π x radius^4).

2 Firefighters have an excellent appreciation for this in selection of hose sizes. Even though a 1 1/2" hose is only 1/4" smaller than a 1 3/4" hose, firefighters know the 1 3/4" hose has much less friction loss and has a significantly greater water flow capacity (gallons per minute).

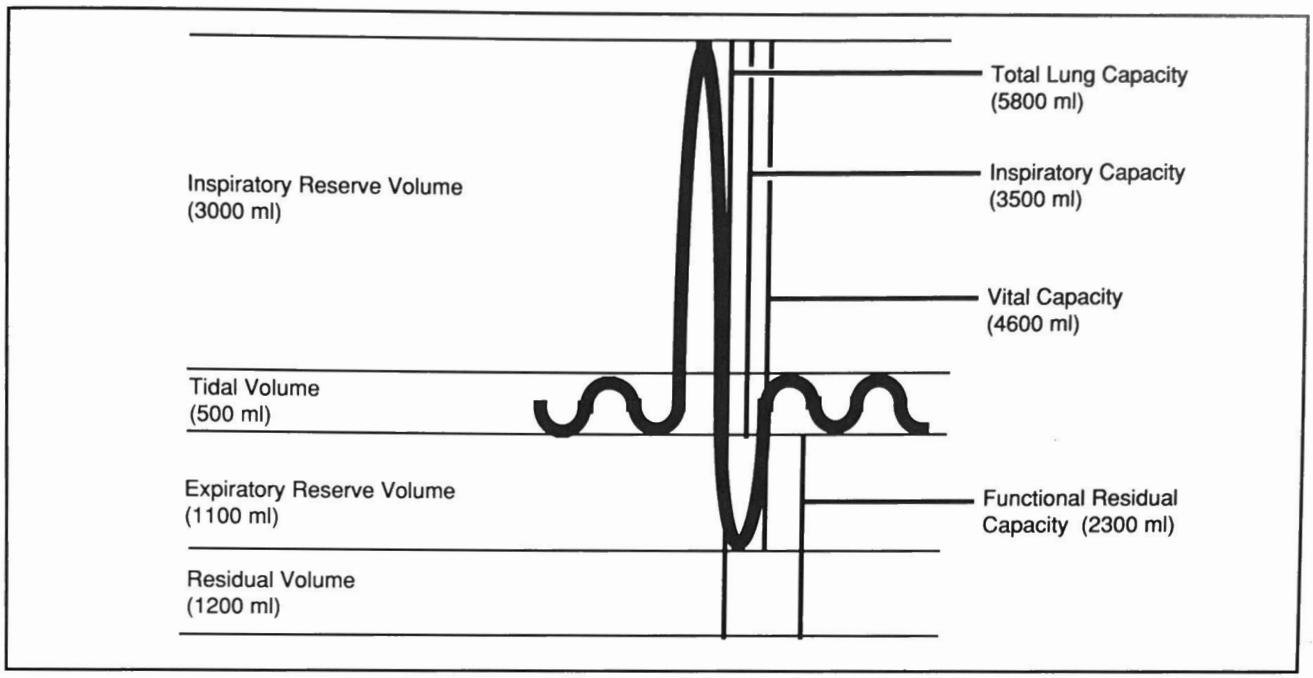

Figure 4 - Lung Volumes and Capacities - The figure shows a graphic recording of air volumes during normal breathing and maximum inspiratory effort followed by a maximum expiratory effort. These figures are for a healthy young adult male. Data taken from Guyton A: *Textbook of Medical Physiology*. 5th Ed. WB Saunders. Philadelphia, 1976. Pg 521.

tion. A smaller tube might be a bit easier to insert, but ventilation, spontaneous or artificial, will require more effort. Even though the airway is not exactly cylindrical, the concept still applies.

The length of the airway is also an important factor. Resistance is proportional to length - a longer airway has a higher resistance. However, even a short segment of airway with a small radius can act as a nozzle, dramatically increasing airway resistance. Bronchial smooth muscle spasm, bronchial swelling or accumulated secretions are frequent causes for narrowed bronchial diameter with increased airway resistance.

The speed at which air is moved in or out of the airway, the airflow velocity, is directly proportional to airway resistance. A fast breath, artificial or spontaneous, will meet more resistance than a slower breath.

Pulmonary Compliance

The lungs and chest are elastic structures. Inflation with air must also overcome this elastic resistance. The value for this total resistance, combining the resistance of the airway and elastic resistance lung and chest wall, is called compliance. Compliance is expressed as the amount of lung expansion that occurs with incremental increases in the intra-alveolar pressure. Normally, this value is 0.13 liters per centimeter of water, such that an intra-alveolar pressure increase by one centimeter of water will expand the air volume in the lungs by 130 ml (4). Decreased compliance, like airway resistance, makes spontaneous and artificial ventilation more difficult.

There are many conditions that can cause decreased compliance, such as obesity, muscle tightness (i.e. seizures) or pleurisy. A patient whose chest has restricted expansion due to a tightly bound extrication vest, tight stretcher straps, or has their chest pinned by a car or dirt in a collapsed trench, may have little or no compliance. The compliance may also be reduced when airway resistance alone increases (i.e. during airway constriction with asthma or anaphylaxsis). Changes in the lung itself can reduce compliance, such as with accumulation of interstitial and/or alveolar fluid with pulmonary edema or with COPD, by loss of overall lung elasticity.

Lung Volumes and Capacities

The total capacity of air in the lung can be divided into four basic volumes (Figure 4). The tidal volume is the amount of air passing through the airway during inhalation or exhalation. The tidal volume is approximately 500 ml in a normal young adult male at rest (Figure 4A). At the peak of normal inhalation, the lung can accommodate still more air with additional inspiratory effort (Figure 4B). That extra maximum inspired volume is the inspiratory reserve volume. It is

normally about 3000 ml. At the peak of normal expiration, an additional maximum expiratory effort (Figure 4C) can push out more air. This extra expired volume is called the expiratory reserve volume, normally about 1100 ml. There is air that must remain in the lung even after a maximum exhalation, or else the lung would collapse. This is the residual volume, normally about 1200 ml (Figure 4D).

Two or more of these ventilatory volumes may be considered together for the sake of ventilatory descriptions. These are called capacities. Adding together the expiratory reserve volume and the residual volume gives the functional residual capacity. The tidal volume and the inspiratory reserve volume equals the inspiratory capacity. Adding together the inspiratory capacity and the expiratory reserve volume gives the vital capacity. The vital capacity plus the functional residual capacity gives the total lung capacity.

Tidal and Minute Volumes

The tidal volume is an important clinical value. In normal quiet breathing for the adult, the tidal volume is about 500 ml. During emergency ventilatory support, there is physical stress with a greater rate of oxygen consumption and carbon dioxide removal. A tidal volume of 10-15 ml/kg of ideal body weight is often used as a starting point for emergency ventilation (5,6). In a 70 kg patient, this would result in a tidal volume of 700 to 1050 ml. The respiratory rate is usually set at 12 per minute. Multiplication of the tidal volume by the respiratory rate gives the minute volume. With a 15 ml/kg tidal volume and a respiratory rate of 12 per minute, the minute volume is 12,600 ml or 12.6 liters per minute. This minute volume is important to remember in the spontaneously breathing patient who has a tight-fitting oxygen mask in place. An oxygen flow less than a patient's minute volume can cause them to try to forcefully pull in more air through leaks in the mask seal or side vents, giving the patient a sensation of suffocation and dramatically increasing their respiratory work (7).

ALVEOLAR GAS EXCHANGE

Dead Space and Alveolar Ventilation

Anatomic Dead Space - Not all inspired air actually reaches the alveoli. Some inspired air remains in the airway passages leading to the alveoli - including the mouth, pharynx, trachea, bronchi and bronchioles. This air does not participate in gas exchange due to its anatomic separation from the alveoli. It is therefore called the anatomical dead space. In a normal young adult male, this is about 150 ml.

Apparatus Dead Space - Artificial airways and ventilation devices can add apparatus dead space. It is the additional volume of air that fills the adjunct(s) in addition to the anatomic dead space. Before adding apparatus dead space, the figure used for anatomical dead space may need to be reduced. An endotracheal tube bypasses all anatomic dead space from its distal tip to the mouth and nose.

Alveolar Dead Space - Not all alveolar air necessarily participates in pulmonary gas exchange. Some alveoli may have little or no blood perfusion. Some alveolar ducts may be blocked or collapsed. This is considered alveolar dead space or partial alveolar dead space for physiological reasons.

Physiological Dead Space and Alveolar Ventilation - The physiological dead space is the arithmetic sum of the anatomic, apparatus and alveolar dead space volumes. In the normal individual, the anatomic is very close to the physiological dead space. In acute emergencies, the physiological

Gas Concentrations in the Atmosphere and Airway

	Atmosphere	*Alveolar Air*	*Expired Air*
Nitrogen (N_2)	597.0 (78.62%)	569.0 (74.9%)	566.0 (74.5%)
Oxygen (O2)	159.0 (20.84%)	104.0 (13.6%)	120.0 (15.7%)
Carbon Dioxide (CO2)	0.3 (0.04%)	40.0 (5.3%)	27.0 (3.6%)
Water Vapor (H2O)	3.7 (0.50%)	47.0 (6.2%)	47.0 (6.2%)
Total	760.0 (100%)	760.0 (100%)	760.0 (100%)

Figure 5 - Gas Concentrations on a Typical Cool Clear Day - Data taken from Guyton A: *Textbook of Medical Physiology*. 5th Ed. WB Saunders. Philadelphia, 1976. Pg 535.

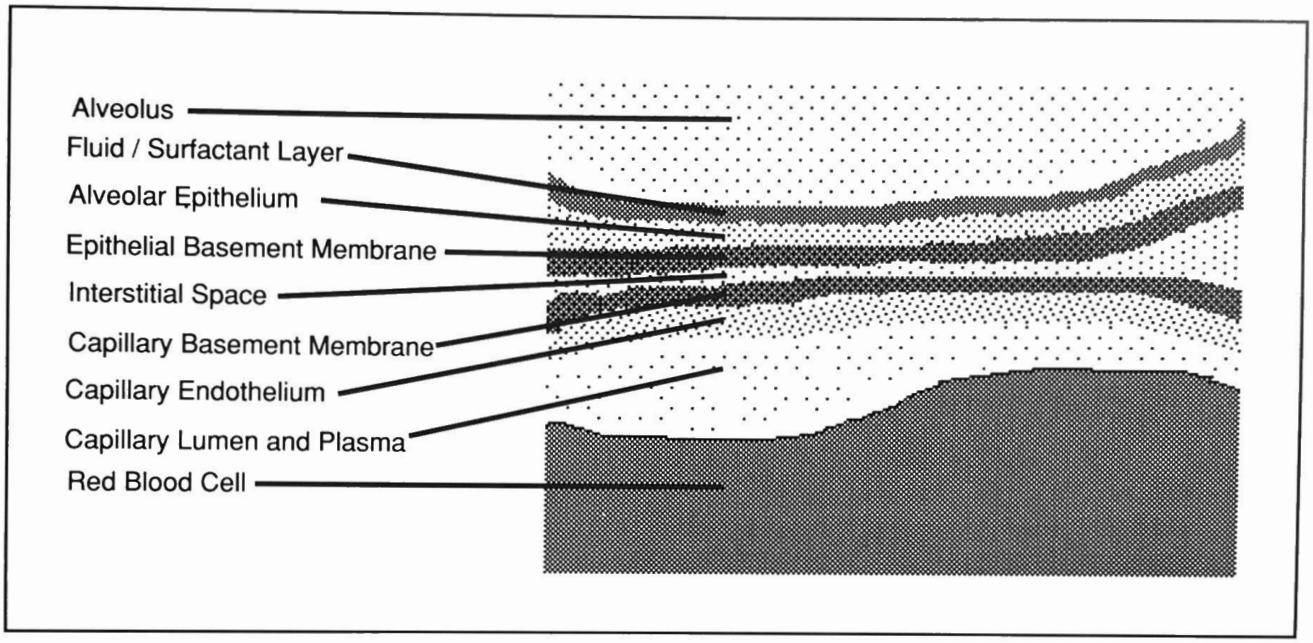

Figure 6 - Barriers to Alveolar-Capillary Gas Diffusion - There are several physical barriers through which gas molecules must pass in order to achieve alveolar - capillary blood gas exchange. In the above diagram, the normal situation is portrayed. In disease or injury, many of these barriers may enlarge with impediment to the gas exchange process.

dead space can be 1000 ml or more. This introduces the idea of alveolar ventilation. The tidal volume less the physiologic dead space is the amount of air that actually participates in effective alveolar ventilation. Multiplying this by the respiratory rate gives the alveolar minute volume This value reflects what all the other mechanical factors in ventilation actually accomplish in terms of moving air in and out of the functional alveoli.

Physical Principals

The freshly inspired air in the alveoli must impart oxygen to the blood and absorb carbon dioxide excreted by the blood.

Without active processes or barriers working the contrary, all particles tend to spread out or diffuse from an area of greater concentration to one of lesser concentration for each particular type of particle. This is also expressed as movement down a concentration gradient. This diffusion continues until the concentration of particles is evenly distributed - the concentration gradient is reduced to a flat line. This is called dynamic equilibrium. Movement of particles still occurs but just as many are coming as they are going.

This principle holds true for dissolution of gasses in water or other solutions. If gas over a solution has more of a particular particle, that particle will diffuse into the solution until the concentration is equal in the air and the solution. Thus, equilibrium will tend to occur between gases in the alveoli and gases dissolved in the pulmonary capillary blood.

Partial Pressure - The atmosphere contains almost 21% oxygen. That is, of all the gasses in the air, 21% of the total volume is oxygen. Instead of expressing this as a percentage, science and medicine often uses terms of partial pressure. Of all the pressure in the air (the current barometric pressure), partial pressure expresses how much of it is due to the presence of a particular gas. The average barometric pressure at sea level is 760 mm Hg. If 21% of that pressure is due to oxygen, oxygen has a partial pressure in the atmosphere of .21 x 760 mm Hg or 160 mm Hg. The atmosphere has almost 79% nitrogen or a partial pressure of 600 mm Hg at sea level.

Alveolar Air Composition - When atmospheric air is inhaled, it is diluted in the air that remained in the lung at the end of exhalation - the expiratory reserve and residual volumes. Additionally, the gasses are humidified to 100%, adding more water vapor. The turbulence and other physical factors mix this fresh and stale air that ultimately composes the alveolar air. On an average cool and clear day, the inspired air contains the concentrations of gasses shown in Figure 5. Also shown are gas concentrations in the resulting alveolar air. Notice that oxygen starts at 159 mm Hg in the atmosphere and ends up at 104 mm Hg in the alveoli.

Oxygen Exchange

The unoxygenated blood coming into pulmonary capillaries has a partial pressure of oxygen of 40 mm Hg, represented as P_v0_2 (P is for partial pressure, $_v$ for venous, 0_2 for oxygen). The alveolar air has a P_A0_2 (P for partial pressure, $_A$

for alveolar, 0_2 for oxygen) of 104 mm Hg. This sets up a gradient that will propel diffusion of oxygen from the alveoli into the blood.

Despite the diffusion gradient, there are factors which impede this gas exchange. A given red blood cell passes through the pulmonary capillary bed in about one second. All gas exchange must occur during this time. With a total pulmonary capillary blood volume of 100 ml, this might seem impossible were it not for the fact that this 100 ml of blood is in contact with approximately 300 million alveoli having a surface area of approximately 750 square feet - about the size of a basketball court.

There are other barriers. Oxygen must cross the alveolar membrane and fluid/surfactant layer, pass through the pulmonary interstitial space, cross the pulmonary capillary membranes, diffuse through the plasma, cross the red cell membrane and reach the hemoglobin. Under normal conditions, the red blood cell is literally squeezing through the pulmonary capillary, making the plasma diffusion factor negligible. The pulmonary interstitial space is also negligible under normal conditions. The capillary and alveolar walls are extremely thin, minimizing their diffusion barrier effect (Figure 6). However, all these barriers can enlarge with disease or injury, making gas exchange much more difficult.

After blood is effectively oxygenated in the pulmonary capillaries, it will mix with blood that has not been oxygenated, due to bypassing of the pulmonary capillary beds or perfusion through poorly ventilated alveoli. This poorly oxygenated blood is called the venous admixture. This normally takes the oxygenated blood coming out of the pulmonary capillaries from a $P0_2$ of 104 mm Hg down to 95 mm Hg.

Carbon Dioxide Exchange

Blood coming into the pulmonary capillaries is loaded with carbon dioxide wastes from the body. It has a P_vC0_2 of 45 mm Hg and the alveolar air has P_A0_2 of 40 mm Hg. This is a much smaller alveolar-capillary blood concentration gradient than found with oxygen. However, carbon dioxide dissolves almost 200 times more readily (has higher solubility) than oxygen, thereby making up for the lower concentration gradient. With the large alveolar surface area and minimized barriers of diffusion that were discussed under oxygen exchange, the carbon dioxide in arterial blood and alveolar air quickly reaches equilibrium.

CONTROL OF VENTILATION

Ventilation is part of a system for homeostatic control of oxygen and carbon dioxide levels in the blood and tissues. These mechanisms regulate the oxygen and carbon dioxide levels with remarkable accuracy under a wide variety of conditions.

Brain Stem Mechanisms

The medulla and pons contain the respiratory center - a group of neurons that collectively stimulate rhythmic breathing. The respiratory center is subdivided into the medullary rhythmicity, pneumotaxic and apneustic areas. Without all three components, ventilation becomes irregular or incorrectly proportioned between inspiration and expiration. Another central nervous system mechanism, the Hering-Breuer reflex, responds to impulses from stretch receptor cells in the lung to prevent over and under inflation. The vasomotor center can stimulate increased ventilation with falling blood pressure. The hypothalamus can stimulate increased ventilation as a heat dissipation mechanism in hyperthermia.

Carbon Dioxide Control Mechanisms

The most influential homeostatic mechanism for basic ventilatory control in normal individuals is based on levels of carbon dioxide in the blood.

Carbon dioxide levels in the body are regulated minute by minute with ventilation. The amount of carbon dioxide in the blood is directly proportional to the alveolar minute volume. The homeostatic objective is adjustment of alveolar minute volume to correspond to increases or decreases in the rates of carbon dioxide production by tissues - usually a function of metabolic activity. Like all homeostatic control systems, the carbon dioxide effect on ventilation has a measurement component with feedback to a regulatory component.

As metabolic activity increases, more carbon dioxide is excreted as waste into the blood by the tissues. As carbon dioxide levels increase, a chemical process ensues that produces carbonic acid, which lowers the blood and interstitial fluid pH. This stimulates chemoreceptors in the brainstem to increase ventilation, thereby lowering $C0_2$ and pH levels back towards normal, eventually dissipating the stimulus for increased ventilation.

In the opposite direction, a fall in $C0_2$ will decrease ventilatory stimulation, allowing $C0_2$ to build up until normal levels are restored. This will finally lessen the stimulus for the ventilatory inhibition.

Oxygen Control Mechanisms

Oxygen levels in the arterial blood are monitored by chemoreceptors in the aortic arch and carotid bodies in the neck. If P_a0_2 rises, it will inhibit ventilation. If P_a0_2 falls, it will stimulate ventilation.

The oxygen regulatory system is not very influential in control of ventilation in normal individuals. In COPD patients, the carbon dioxide control system fails, making the oxygen control system become primary. COPD patients with

spontaneous breathing must therefore receive supplemental oxygen under careful monitoring of respiratory rate and depth, to avoid undetected inhibition of their spontaneous ventilatory activity by a high P_aO_2. Their chronically lower P_aO_2 maintains their ventilatory stimulus - this is the so-called hypoxic drive.

INSTRUMENTS FOR CLINICAL MEASUREMENT OF VENTILATION

Without adjunctive equipment, the emergency clinician can assess ventilation by observation of chest movement, activity in accessory muscles of breathing, skin color, etc. However, quantification with instruments allows treatment decisions to be made with specific data. Responses to therapy may be quantified and used to regulate intervention as a replication of natural homeostatic measurement-control systems.

Historically, the limiting factor in reaching higher levels of clinical sophistication and precision of intervention is the sophistication and precision of our monitoring of the patient. In practical terms, particularly in the field, any monitoring devices must not be such that they detract from basic care. The magnitude of that risk is a function of logistics, training and medical control.

In the apneic or severely impaired case requiring ventilatory assistance, ventilatory measurement may be used to control the tidal volume. It was mentioned earlier that in emergency artificial ventilation, a 10-15 ml/kg tidal volume is desirable (5,6). However, that figure assumes a limited component of dead space, probably of about 150 ml in an adult. If an airway adjunct adds a significant amount of apparatus dead space, tidal volume may need to be adjusted.

Tidal volume may be measured by devices in-line with the airway (usually an endotracheal tube) or by components within a ventilator. However, tidal volume measurement may be inspiratory or expiratory. Expiratory tidal volume is generally preferred, as it will reflect the net ventilation if there are leaks in the apparatus, such as a torn cuff in an endotracheal tube.

Another artificial ventilation monitoring device is an airway pressure manometer. Barotrauma to the lung is a very real risk in ventilatory support, particularly when pop-off valves are not utilized. Further, notation of the changes in airway pressure required to deliver a specific tidal volume with a specific inspiratory time can track changes in pulmonary compliance.

Quantification of ventilation in a conscious and spontaneously breathing patient cannot be as direct, because complete airway control with intubation is usually not available. Here, ventilatory quantification is used not to regulate tidal volume and alveolar minute volume but to make serial measurements to assess the need and effects of respiratory interventions.

Peak expiratory flow is a simple bedside measurement that reflects pulmonary function. The patient's lips seal around a small hand-held peak expiratory flow meter during a maximum forced expiratory effort. Full patient cooperation is essential for this test. By quantification of ventilatory compromise, peak expiratory flow may allow protocols to be developed that might specify values under which certain interventions, like bronchodilator drug therapy, might be considered and responses to which may be documented for contemplation of follow-up treatment or other interventions if initial measures were unsuccessful.

Another helpful field monitor may be transcutaneous oximetry. These devices monitor oxygen saturation by measurement of the color of blood as it passes through the skin beneath its sensor. Oximetry can be an early warning sign for hypoxia, indicating need for immediate intervention - before the patient becomes obviously cyanotic or "crashes". Technology is also available for transcutaneous measurement of oxygen in carbon dioxide partial pressures. These values correlate closely to arterial blood gas values, but diverge with decreased peripheral perfusion.

There is a significant lack of quantification in assessment and measurement of responses to treatment during emergency care. This is often due to a primary concern of simply keeping the patient alive during acute decompensation where interventions are primarily supportive and straightforward. Initiation of monitoring in these extremely acute situations is often impractical. However, if monitoring is initiated prior to or as soon as possible during crisis, the information might allow for better clinical decisions and improvement in patient outcomes.

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