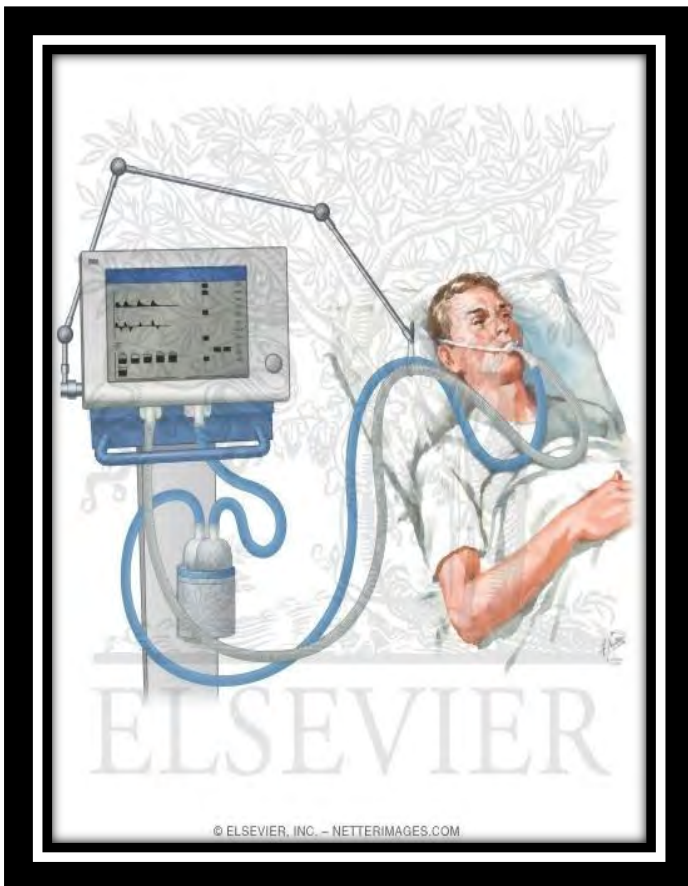




2016

Mechanical Ventilation Learning Package



Sharon-Ann Shunker, CNC, Liverpool ICU

SWSLHD

2/3/2016

Contents List

1.	Respiratory Anatomy & Physiology	Page 3
2.	Pulmonary Ventilation: Pleural pressure	Page 5
3.	Pulmonary Circulation	Page 6
4.	Relationship between Ventilation and Perfusion	Page 8
5.	Lung Volumes & Capacities	Page 10
6.	Dead Space Ventilation	Page 12
7.	Lung Mechanics: Compliance & Resistance	Page 13
8.	Oxygenation	Page 17
9.	Arterial Blood Gases	Page 22
10.	Airway Management	Page 25
	➤ Basic Airway Management	
	➤ Management of the Obstructed Airway	
	➤ Use of Guedel & Nasopharyngeal Airways	
11.	Oxygen Therapy	Page 28
12.	Tracheal Intubation	Page 29
13.	Mechanical Ventilation	Page 37
	➤ Non Invasive Ventilation – CPAP & BiPAP	Page 37
	➤ Invasive Ventilation Principles	Page 43
	➤ Modes of Ventilation	Page 45
	➤ Ventilation Parameters	Page 51
	➤ Alarm Settings	Page 53
	➤ Ventilation Emergency Drill	Page 56
	➤ Troubleshooting Alarms	Page 58
	➤ Humidification	Page 60
	➤ Weaning from Mechanical Ventilation	Page 61
	➤ Nursing Care of the Mechanically Ventilated Patient	Page 65
14.	Legend for Sedation Score	Page 68
15.	Legend for Pain Score	Page 69
16.	Learning Activities	Page 72
17.	Reference List	Page 73

1. INTRODUCTION

The 3 physiological functions that are responsible for adequate tissue and cellular oxygenation are:

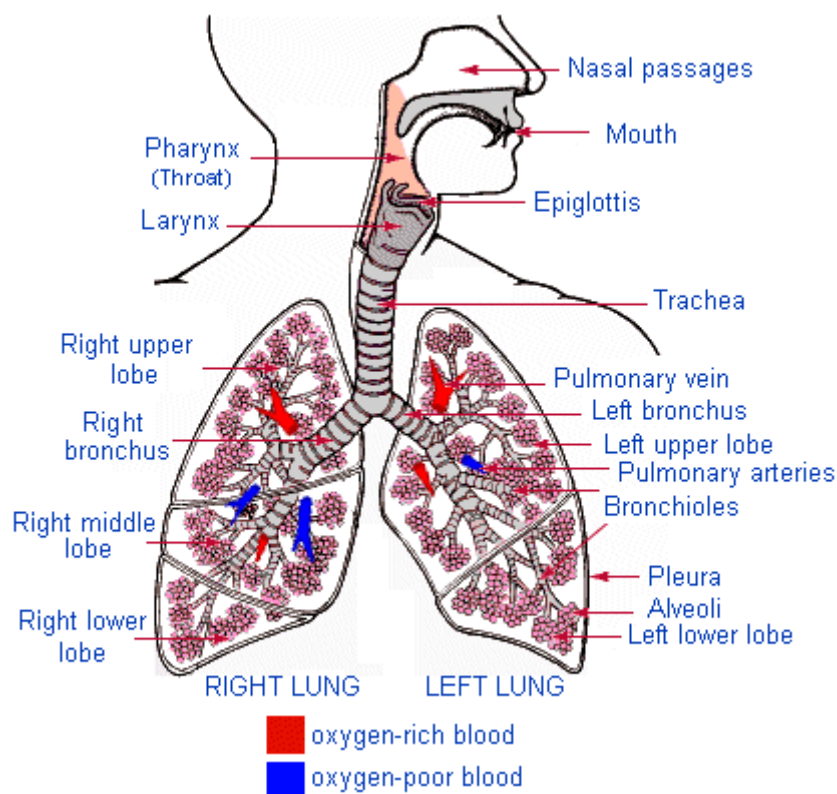
- Pulmonary gas exchange
- Oxygen delivery
- Oxygen Consumption

The respiratory system supports gas exchange through the process of ventilation, diffusion and perfusion for the uptake of oxygen and the removal of carbon dioxide from the body.

Mechanical ventilation can be provided via non-invasive or invasive means and involves the delivery of positive pressure breaths. Gas flow is delivered via a constant or decelerating pattern and the volume is dependent on inspiratory time, gas flow and pressure applied at the airway. Pressure, flow, time and volume are all interrelated. Lung elasticity, chest wall and abdominal characteristics determine compliance (Oh, 1997)

Respiratory Anatomy and Physiology⁴⁵

Functionally the respiratory system consists of the conducting and respiratory zones, namely the upper and lower respiratory tract. The conducting zones consist of the nasal cavities, pharynx, larynx and trachea. The purpose of the conducting zones is to filter, humidify, warm and allow the passage of air to the lower respiratory zones. The respiratory zones are the site of gas exchange, containing the respiratory bronchioles, alveolar ducts and alveoli. (Tortora & Grabowski, 2000).



(Source: <http://webschoolsolutions.com/patts/systems/lungs.htm>)

Functions of the respiratory system

The major function of the respiratory system is to supply oxygen and eliminate carbon dioxide from the body. In addition to the vital function of gas exchange, the respiratory system fulfils the following functions¹³

- Acid base regulation – Through the process of ventilation, the lung removes CO₂ and regulates the pH of the body. Regulation of pH is accomplished by removing volatile acid (ie acid converted into the gaseous state; in this case, carbonic acid converted to CO₂).
- Blood reservoir – The lung receives the venous blood from the right ventricle. Due to the capacity of the pulmonary circulation to receive blood, the lung acts as a reservoir from which the left side of the heart draws blood.
- Filtering mechanism – The lung also constantly filters the air we breathe and removes trapped particles through the mucociliary clearance mechanism and the lymphatic system. The lung also acts as a filtering mechanism for blood by removing particles such as gas bubbles, small fibrin or blood clots, fat cells, aggregates of platelets or WBC, and other pieces of cellular debris.
- Metabolism – The lung produces some very important chemicals that serve physiological regulatory functions such as vascular dilatation, blood clotting, lung structural stability and neurotransmitters. Some chemicals passing through the lungs are converted into their more active form, such as angiotensin I, produced by the kidneys, which is converted to angiotensin II, a potent vasoconstrictor

Control of respiration^{7,13}

Breathing is usually involuntary but voluntary breathing is necessary when the person is doing other activities such as walking, talking, singing, etc. In these cases homeostatic changes in ventilatory rate and volume are adjusted automatically by the nervous system to maintain normal gas exchange.

The lung is innervated by the autonomic nervous system (ANS). Fibres of the sympathetic division in the lung branch from the upper thoracic and cervical ganglia of the spinal cord, while fibres of the parasympathetic division travel in the vagus nerve, which is important in the regulation of ventilation. The respiratory centers in the brain stem control involuntary ventilation by transmitting impulses to the respiratory muscles causing them to contract or relax.

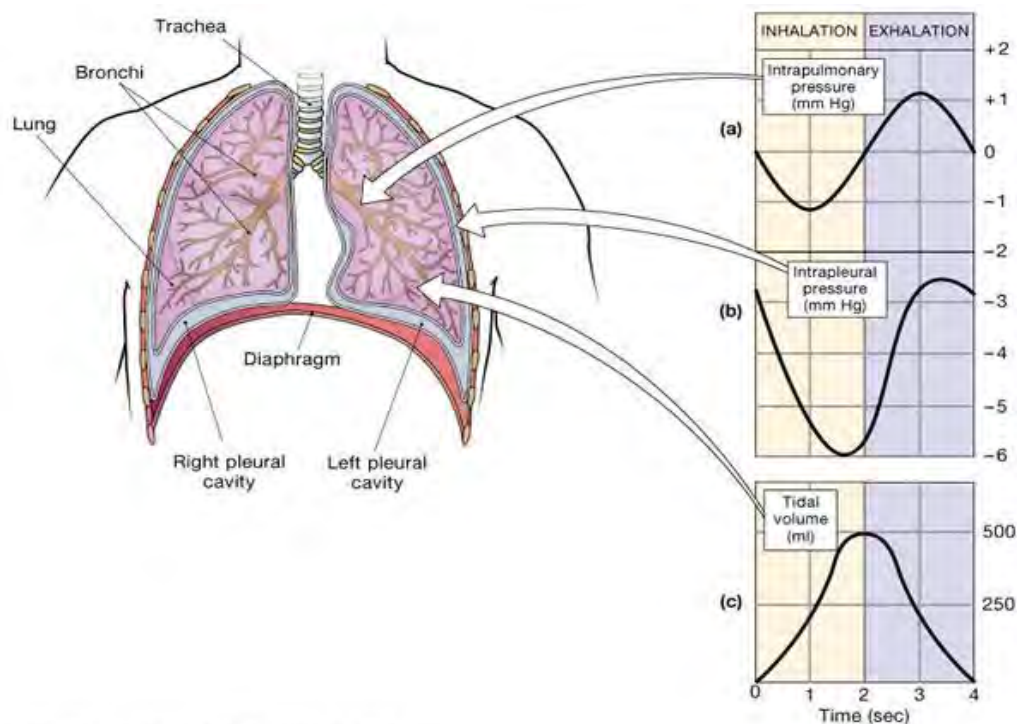
The pneumotaxic center in the upper pons functions to maintain rhythmic respirations. It stimulates the expiratory center, which then sends inhibitory signals to the inspiratory center. Inspiration ends and expiration begins. Strong stimuli from the pneumotaxic center result in shorter inspiration, and mild stimuli results in a longer one. The apneustic center sends stimuli to the inspiratory center and prolongs inspiration. The pneumotaxic center usually overrides the apneustic center. Impulses are transmitted from lung receptors, which are receptors that respond to physical changes in the pulmonary system, and chemoreceptors, which are receptors that respond to changes in oxygen or carbon dioxide concentrations, through the sympathetic and parasympathetic divisions of the ANS and the respiratory centers in the brain stem.²⁵

2. PULMONARY VENTILATION: PLEURAL PRESSURES

The lungs and chest wall are separated by the parietal and visceral pleura. Between the parietal and visceral pleura is the interpleural space. The pressure within the interpleural space is usually negative due to the natural tendency of the lungs to collapse and the chest wall to expand. It is this negative interpleural pressure that keeps the alveoli open and through the interaction of the lungs and chest wall interpleural pressure is altered, enabling the movement of gas into and out of the lungs (ventilation).

In the intact chest, the lungs move as the chest wall and diaphragm moves because of the maintenance of a pressure in the interpleural space that is negative with respect to the alveolar pressure. As the thoracic dimensions increase, pleural pressure is reduced which causes the lungs to increase in volume (inspiration). As the thoracic dimensions decrease, pleural pressure and alveolar pressure is increased, causing gas to flow out of the lungs (expiration).^{45,50}

Interpleural pressure (quiet breathing with normal lungs)



Copyright © 2004 Pearson Education, Inc., publishing as Benjamin Cummings.

3. PULMONARY CIRCULATION

Certain problems with ventilation and perfusion can relate to the distribution of blood flow and hemodynamics that are unique to the pulmonary circulation and important for gas exchange in the lungs¹³

Anatomy of the Circulation

Pulmonary Artery: The pulmonary artery divides into left and right branches which supply blood to the two respective lungs. The walls of the vessels have large diameters and are thin and distensible giving the pulmonary artery system a large compliance of almost 7ml/mmHg.

Bronchial vessels: These carry oxygenated blood to the supporting lung tissues, after it has passed through the tissues it empties into the pulmonary veins and left atrium.

Lymphatics: They extend from the supportive tissue of the lungs into the hilum and empty into the right lymphatic duct. They remove particulate matter from the alveoli and plasma protein leaking from the lung capillaries, thereby helping to prevent pulmonary edema.¹³

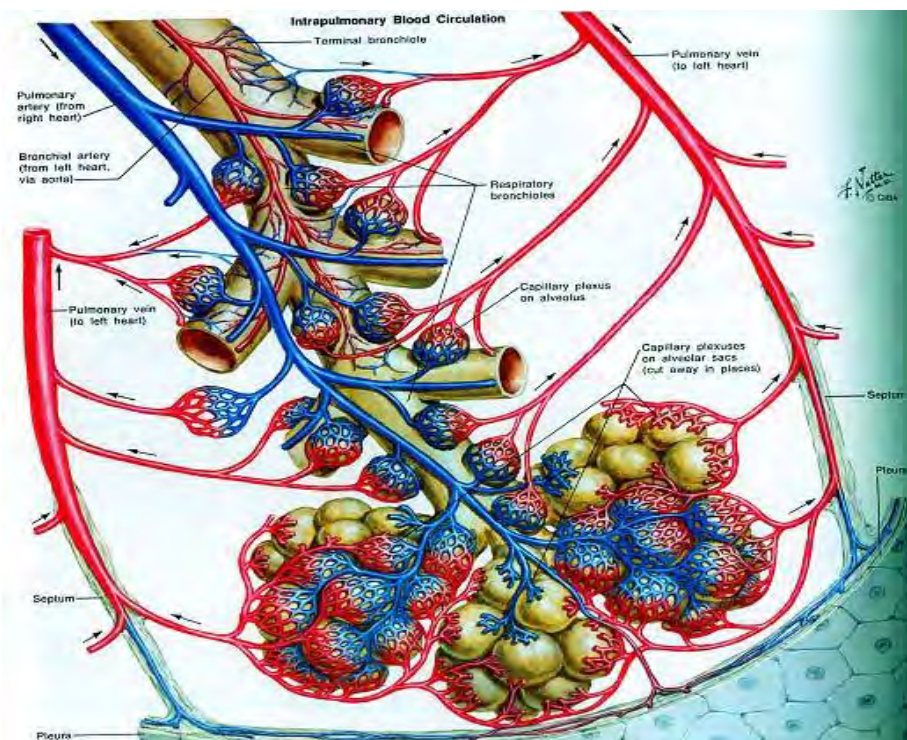
Pressures in the Pulmonary System

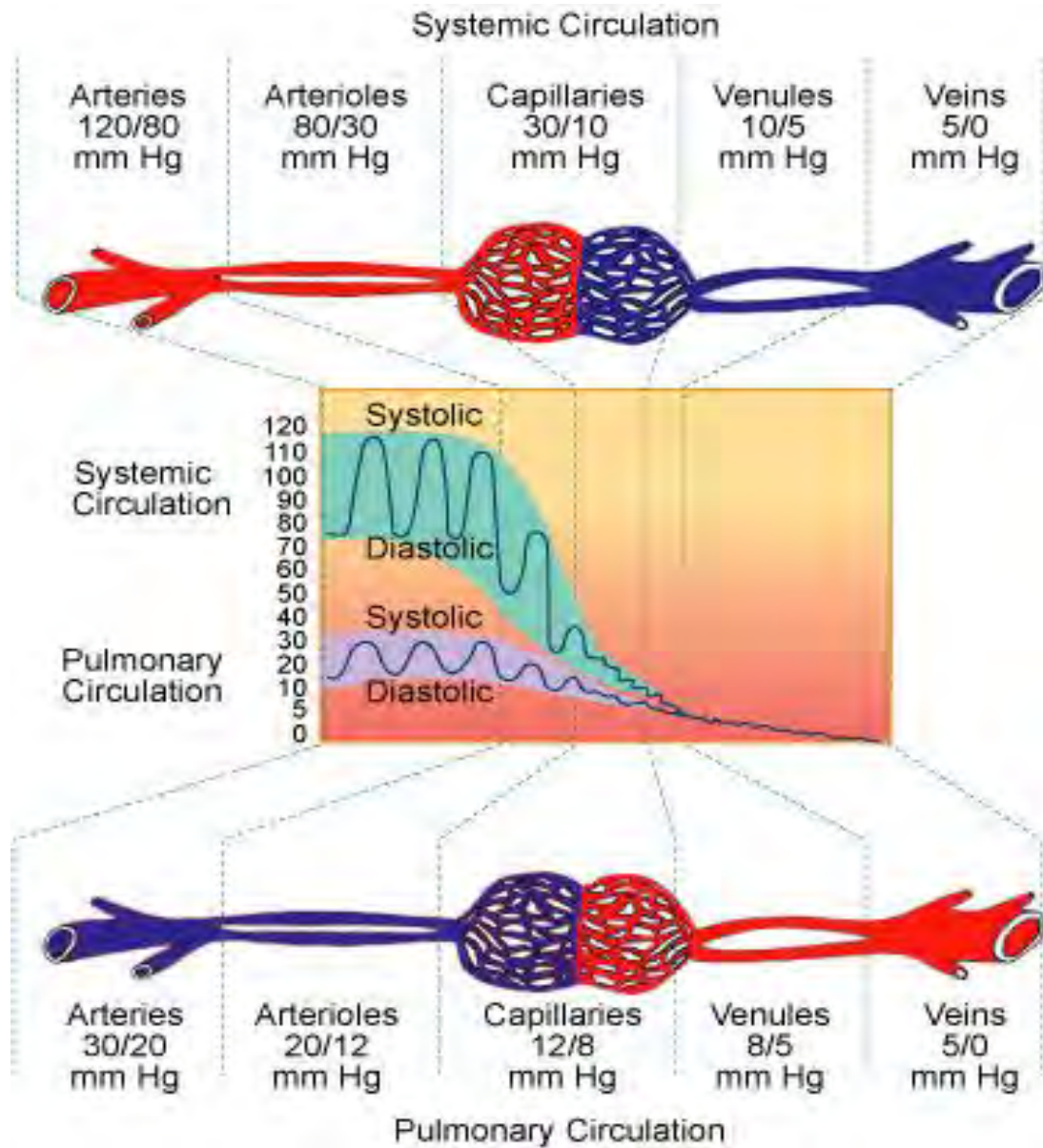
Pressure in the pulmonary artery: During systole the pressure in the pulmonary artery is essentially equal to the pressure in the right ventricle. As the pulmonary valve closes the pressure in the ventricle falls precipitously whereas the pulmonary artery pressure falls more slowly as blood flows through the lung capillaries. Normal systolic PA pressure is 25mmHg, diastolic is 8mmHg and mean is 15mmHg.

Pulmonary capillary pressure: Mean pressure is about 7mmHg.

Left Atrial and Pulmonary Venous Pressure: The mean pressure in the left atrium and major pulmonary veins averages 2mmHg in the recumbent position.¹³

Intrapulmonary Circulation (Percussionaire.com)





(Source: pulmonary circulation, web.cateret.edu)

4. RELATIONSHIP BETWEEN VENTILATION AND PERFUSION

Gas exchange is the key function of the lungs. It is affected by the anatomy of the capillaries and alveoli. Due to a number of physiological factors, ventilation (V) to perfusion (Q) ratio is not matched in a 1:1 relationship. Normal alveolar ventilation is about 4L/min and pulmonary capillary perfusion is about 5L/min, hence normal ventilation to perfusion ratio (V/Q) is 0.8.

Perfusion to the pulmonary circulation, like ventilation, is not evenly distributed and is dependent on hydrostatic pressures.

In the upright position the lung apices receive less perfusion compared with the bases.

In the supine position apical and basal perfusion is almost equivalent, but the posterior portion of the lung receives more perfusion than the anterior lung.

Throughout the lung the bases receive more ventilation per unit volume than the apices.

Blood flow in the pulmonary capillary network (perfusion) is affected by pressure within the surrounding alveoli. The pressure gradient between the arterial and venous end of the pulmonary circulation affects blood flow, but as alveolar pressure can be greater than venous or arterial pressure, this affects blood flow and gas exchange.

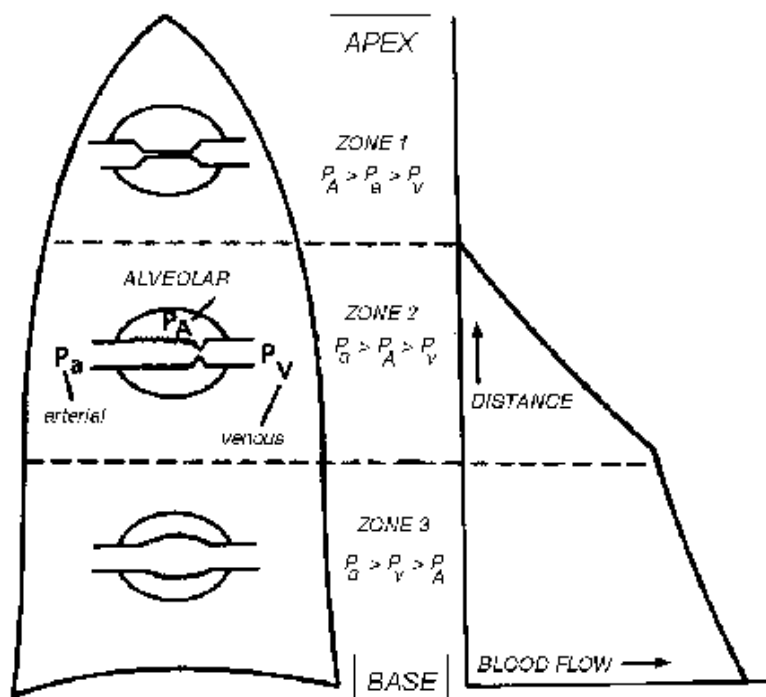
In the upright posture, the three zones are:

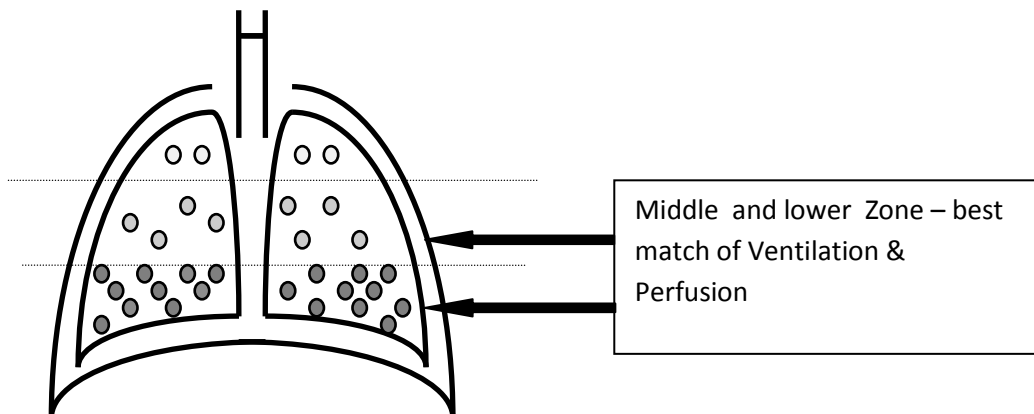
Zone 1: (upper area of lungs) Alveolar air pressure greater than either pulmonary arterial and venous pressures, so blood flow is reduced and there is alveolar deadspace. This is more evident in the patient on positive pressure ventilation.

Zone 2: (middle area of lungs) Alveolar air pressure less than pulmonary arterial pressure but greater than pulmonary venous pressure, there is a normal V/Q ratio.

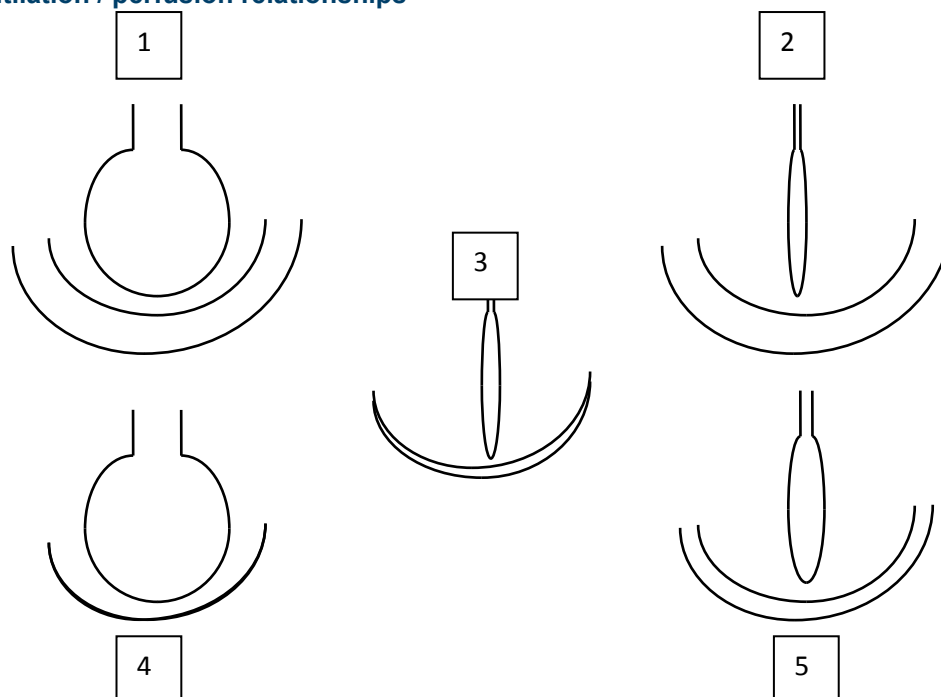
Zone 3: Alveolar air pressure is less than both pulmonary arterial and venous pressures, so ventilation is reduced, leading to shunting. Alveoli are perfused but not adequately ventilated.

Optimal ventilation and perfusion happens in both zone 2 & 3.¹⁰



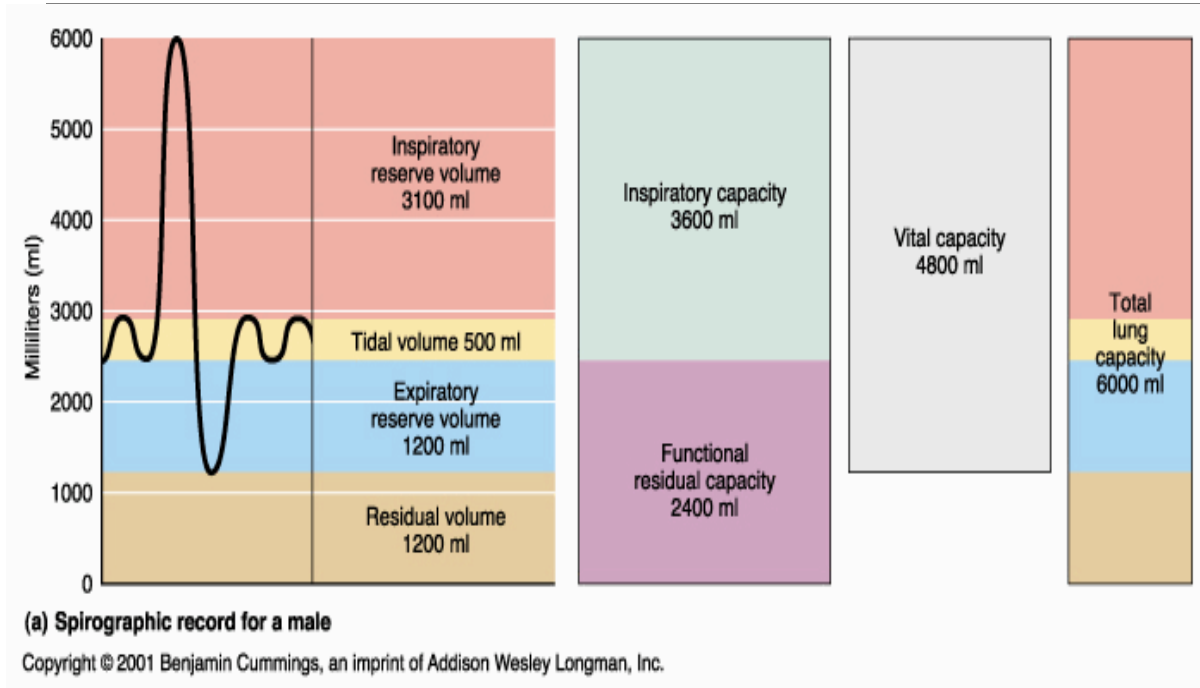
Distribution of ventilation in the normal upright person

It can be seen from this that the best match of ventilation to perfusion occurs in the middle lung zones in the upright person. The goal of many respiratory therapies is to optimise both ventilation and perfusion to the alveoli. The relationship between ventilation and perfusion is optimal when there is a match between ventilation and perfusion, that is, where the alveoli are receiving normal ventilation and perfusion. Disease states can alter this relationship, as depicted in the following diagram (Berghuis, et al, 1992, *Spacelabs Biophysical Measurement Series; Respiration*, page 8).

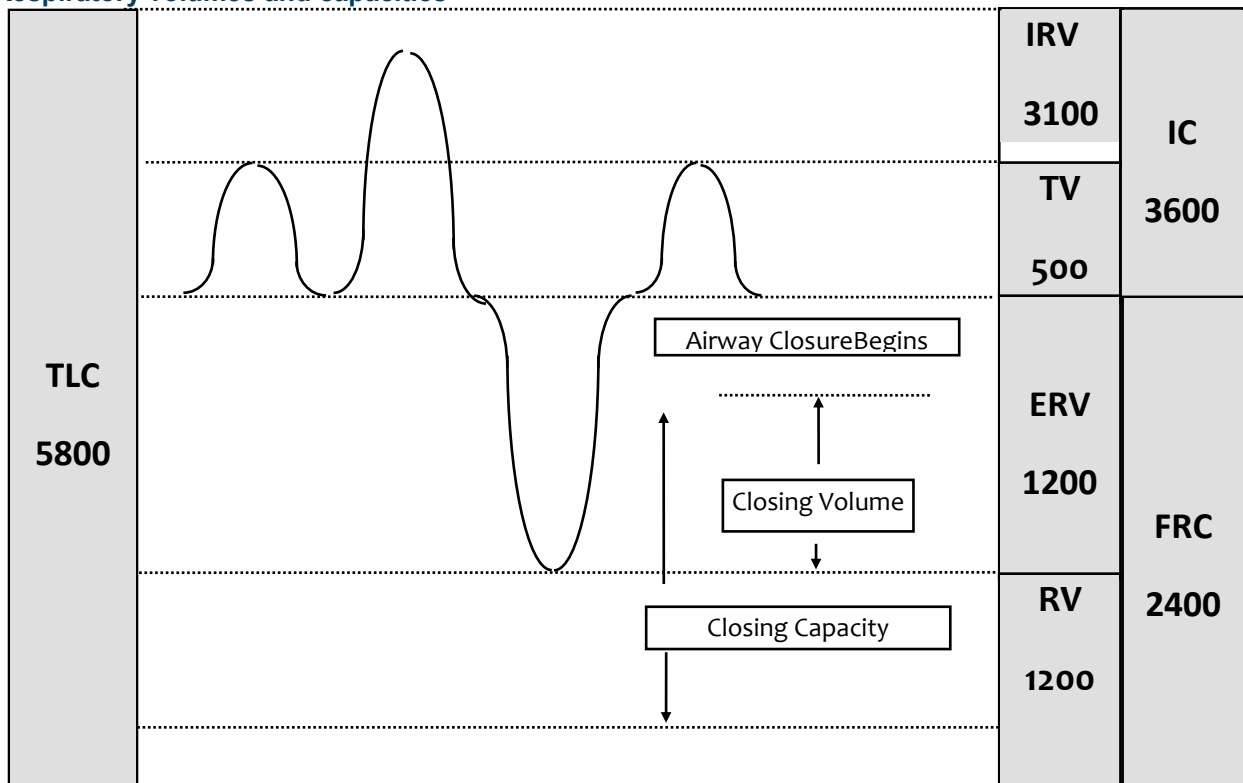
Ventilation / perfusion relationships

1. Normal lung unit, receiving normal ventilation and perfusion
2. Shunt unit, not ventilated but receiving normal perfusion
3. Silent unit, neither ventilated or perfused
4. Deadspace unit, ventilated but not perfused
5. Example of alveoli that is under-ventilated and under-perfused

5. LUNG VOLUMES AND CAPACITIES



Respiratory volumes and capacities



Source: Marieb 1992, pages 742–743

As noted above, interpleural pressures result from the relationship between forces generated by the chest wall and lung. This relationship also determines the resting volume of the lungs (at end of normal expiration). This volume is called the functional residual capacity (FRC). This is the point where chest wall forces and lung forces are in balance. The following concepts are very important to appreciate.

Inspiration and expiration⁵⁰

Inspiration and expiration refer to changes in lung volume. Change in lung volume requires the generation of a pressure difference. The pressure difference (for spontaneous breathing) is generated by the respiratory muscles.

The pressure change required to produce a given change in lung volume (compliance) varies depending upon how full the lungs are. Recording the volume change for particular pressure change produces what is called the lung pressure–volume curve (or the static pressure – volume relationship). This relationship is sigmoidal; that is, the pressure required to produce volume change at low and high lung volumes is much greater than that needed to produce volume change in the middle section. This middle section corresponds to normal tidal breathing in the healthy lung.

Pressure / Volume Curve

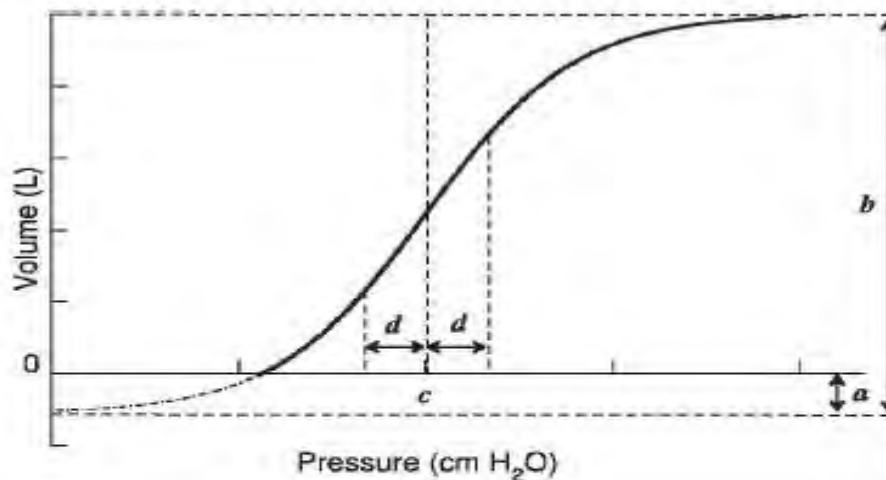


Figure 1 - Parameters for the sigmoidal equation $V = a + b / (1 + e^{-(P-c)/d})$. V is absolute lung volume, P is airway pressure, a is the difference in volume between EELV at ZEEP and residual volume, and b is the volume from a to the upper asymptote approximating vital capacity. The parameter c is the pressure at the mathematical sigmoidal inflection point and represents the pressure at which compliance is maximal. Parameter d is proportional to the pressure range at which most of the volume change occurs and is measured in cm H₂O.

Where tidal breathing occurs in a lung already approaching total lung capacity (eg very small vital capacity, or as a result of hyperinflation) the pressure (negative pressure) that the respiratory muscles must generate will be much higher than that required for normal tidal breathing at normal FRC. Where tidal breathing occurs in a lung with reduced FRC (near residual volume) the same consideration applies (eg obesity/abdominal distension, atelectasis).

This means that at these two extremes (TLC/FRC) the work of breathing will be increased.

Reduction in lung volume⁵⁰

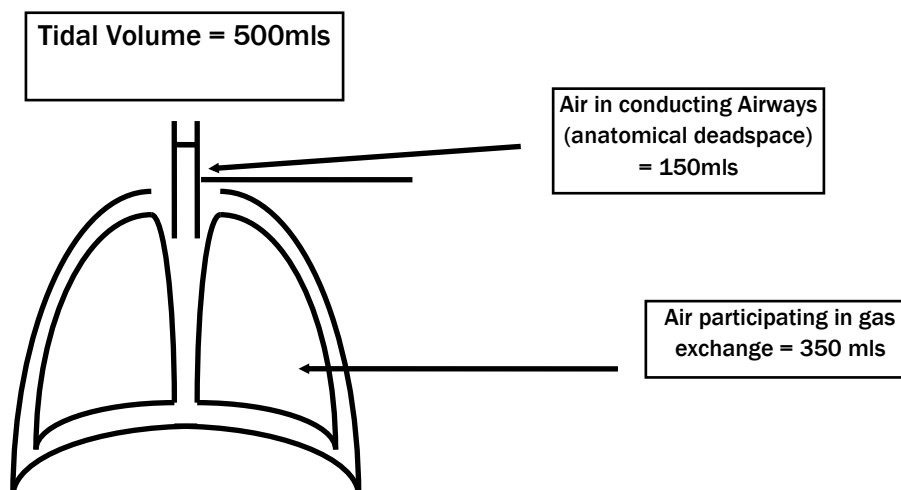
Reduction in lung volume below a certain level results in airway closure (small airways such as respiratory bronchioles). The lung volume at which this occurs is known as the closing capacity (CC). In older people and those with chronic lung disease, some of the lungs' elastic recoil is lost, with a resulting decrease in intrapleural pressure. Thus the volume at which airway closure occurs is higher (closer to FRC).

6. DEAD SPACE VENTILATION

Dead space is the amount of gas that is involved in ventilation but does not take part in gas exchange. (Not all the air in each breath is used for the exchange of oxygen and carbon dioxide. About a third of every resting breath is exhaled exactly as it came into the body.) There are four types of dead space⁷:

- Anatomic dead space – This refers to the amount of gas that fills the conducting passages of the airway and is not involved in gas exchange. In most adults, this value is estimated at 2 mL/kg of body weight. For the normal sized adult, it is usually about 150 mL. Therefore, if the normal tidal volume is 500 mL, only 350 mL of tidal volume is actually involved in gas exchange as illustrated below.
- Alveolar dead space – This is the amount of gas filling the alveoli that does not contribute to gas exchange.
- Mechanical dead space – This is the contribution to the patient's dead space through the addition of respiratory circuit attachments, etc.
- Physiologic / total dead space – This value is the sum of anatomic and alveolar dead space. It represents the total volume in the airways and alveoli not participating in gas exchange.

The relevance of anatomical dead space



As the dead space increases, the amount of gas that actually contributes to gas exchange decreases. The volume of gas that takes part in gas exchange is alveolar ventilation.

In the mechanically ventilated patient there is mechanical dead space that is contributed by the circuit. This is compressible dead space that can be reduced in patients with conditions such as ARDS and ALI, when it is difficult to achieve optimal tidal volumes.⁷

7. LUNG MECHANICS: COMPLIANCE AND RESISTANCE

The mechanical characteristics of the lung greatly influence both normal lung function and pulmonary disability. The two major factors involved in lung mechanics are compliance and resistance, as outlined below.⁵⁰

Compliance

Normally inspiration is an active process, accomplished through the expansion of the lungs and the thorax. The ease with which the lungs and thorax can be expanded, or distended, is referred to as compliance. Total compliance therefore depends not only on the elasticity of the lung tissue, but also on that of the thoracic cage.

Compliance determines the change in volume for a given change in pressure. For example, if a patient is able to sustain a large increase in tidal volume with a small fall in pleural pressure then their lungs are compliant. If a patient requires a large fall in pleural pressure for a relatively small increase in tidal volume then their lung tissue is non-compliant.

Compliance is reduced by any factor that:

Reduces the natural elasticity of the lungs, eg fibrosis, or interstitial oedema.

Reduces the total number of functional alveoli, eg atelectasis or airway obstruction.

Increases the stiffness of the chest wall, eg splinting because of pain.

Decreases the stiffness of the chest wall, eg post-sternotomy, resulting in decreased FRC.

Checks the ability of the thorax to increase in volume, eg abdominal distension.

Compliance is therefore a relationship between volume and pressure and can be estimated by dividing the change in volume by the change in pressure as follows:

$$\text{Compliance} = \frac{\text{change in volume}}{\text{change in pressure}}$$

For example, your patient is receiving the following ventilator parameters:

PEEP – 10 cm H₂O

Tidal volume – 1000 mL

End inspiratory hold or plateau pressure – 35 cm H₂O

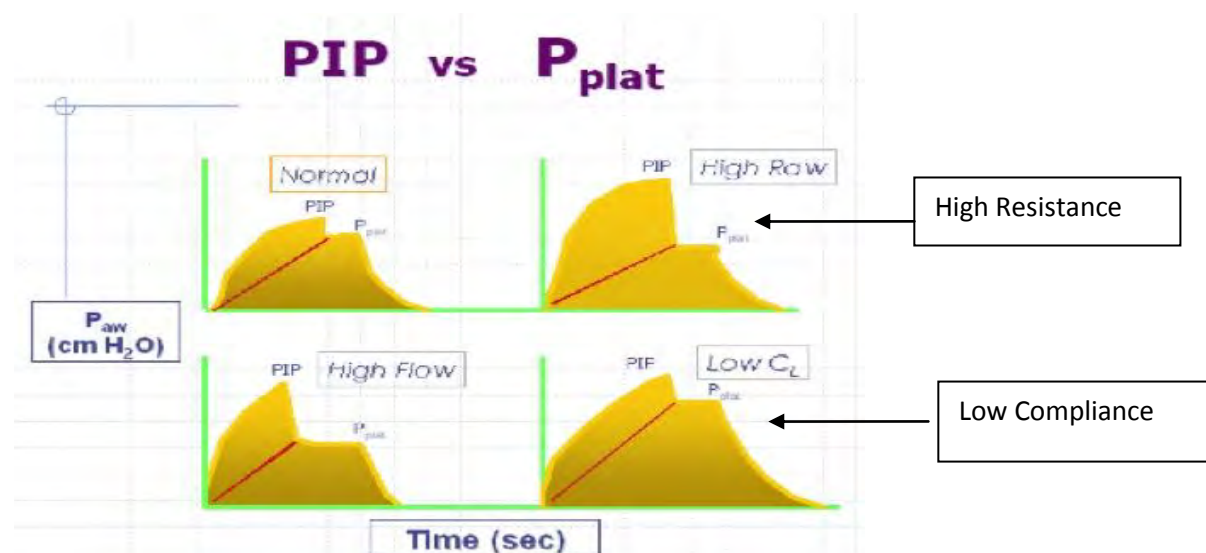
In this case, the change in volume is 1000 mL and the change in pressure is 25 cm H₂O. The change in pressure is determined by subtracting the level of PEEP (10 cm) from the end inspiratory hold pressure (35 cm). Remember, we are interested in the change in pressure and in this case the pressure is rising from a baseline of 10 cm to a total pressure of 35 cm of water, the resultant change in pressure is therefore 25 cm.

The total lung compliance (lung and chest wall) for this patient is:

$$\frac{1000}{35-10}$$

$$= \frac{1000}{25} = 40\text{mL /cmH}_2\text{O}$$

You will note that the estimated value for compliance is stated in mL/cm H₂O. In the above example, this means that for a 1 cm increase in pressure the patient would experience a 40 mL rise in volume. The normal value for adult compliance is a combination of lung and thoracic wall compliance and is 70–100 mL/cm H₂O.⁵⁰



Clinically, there are two types of compliance measurements that can be determined. These are dynamic compliance and static compliance.

Dynamic compliance is calculated by the following formula:

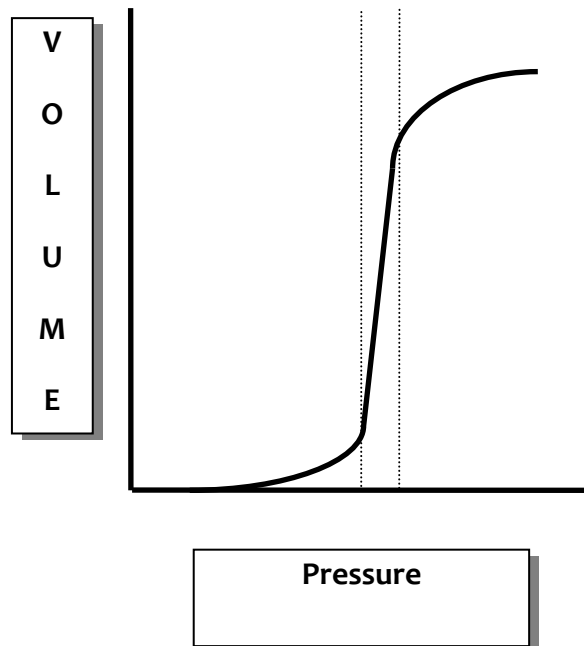
$$\text{Dynamic compliance} = \frac{\text{Tidal volume}}{\text{Peak inspiratory pressure} - \text{PEEP}}$$

Static compliance is calculated by the following formula:

$$\text{Static compliance} = \frac{\text{Tidal volume}}{\text{Plateau pressure} - \text{PEEP}}$$

To obtain the static compliance, an inspiratory pause must be initiated. This pause will result in a period of no gas flow and allow the pressure in the alveoli to equilibrate with the ventilator circuit pressure. The measurement of static compliance may be useful in eliminating the following variables that may influence compliance readings: resistance to flow, distribution of gases and recruitment time of closed lung units.

Compliance alters during phases of a maximal inspiration. At lung volumes near RV and TLC, the lung tissue is less compliant (ie not as distensible). This results in an 'S' shaped curve (see following figure). Conceptually, this is similar to blowing up a balloon. It is more difficult to inflate a balloon at the beginning of inflation. Once the balloon starts to inflate, less pressure (or work) is required to inflate the balloon. As the balloon reaches its total capacity and draws near to bursting, a greater pressure is required to achieve a unit volume increase. Thus the lung, like a balloon, requires greater pressures at the beginning (near functional residual capacity) and end of inspiration (near total lung capacity) for relatively small increments in tidal volume; this is a state of decreased lung compliance. In the middle of inspiration, little pressure is required for increases in volume – ie the lungs are more compliant.

Compliance 'S' curve

Pulmonary Surfactant increases compliance by decreasing the surface tension of water. The internal surface of the alveolus is covered with a thin coat of fluid. The water in this fluid has a high surface tension, and provides a force that could collapse the alveolus. The presence of surfactant in this fluid breaks up the surface tension of water, making it less likely that the alveolus can collapse inward. If the alveolus were to collapse, a great force would be required to open it, meaning that compliance would decrease drastically^{45,50}

Compliance^{45,50}

Compliance refers to the distensibility of the lung tissue.

A patient with a low compliance or non-compliant lungs is said to have 'stiff' lungs.

Signs of non-compliant lungs may include high airway pressures for a given tidal volume. Lungs that have decreased in compliance will require higher airway pressures to deliver a given tidal volume.

Potential complications of increased airway pressures include: barotrauma, mediastinal emphysema, pneumothorax, and tension pneumothorax.

Compliance is calculated by dividing the change in volume by the change in pressure.

The normal value (full size adult) for compliance (total lung) is approximately 70–100 mL/cm H₂O.

Compliance for a patient who is intubated and ventilated is approximately 40–60 mL/cm H₂O; this will vary depending on whether you are measuring static or dynamic compliance.

Compliance is related to lung size; larger lungs have higher compliance.

Elasticity is often mistaken to mean compliance. Elastance is the reciprocal of compliance and is defined as the force with which the lung fibres try to recoil.

Resistance⁵⁰

Resistance refers to impedance to flow. For gas to flow, a pressure difference must exist between the two ends of a tube. The relationship between the driving pressure and the resultant flow is termed the resistance. Airway resistance is the pressure difference between the alveoli and mouth divided by flow rate.

Resistance to flow may be inspiratory or expiratory. Factors that may increase both inspiratory and expiratory resistance include:

- Bronchial tone.
- Sputum.
- Oedema.
- External breathing circuits (eg ETT / tracheostomy tube and other circuit components).

Airflow obstruction can lead to gas trapping, which results in dynamic hyperinflation (auto PEEP, intrinsic PEEP, inadvertent PEEP). The possible effects of auto PEEP are:

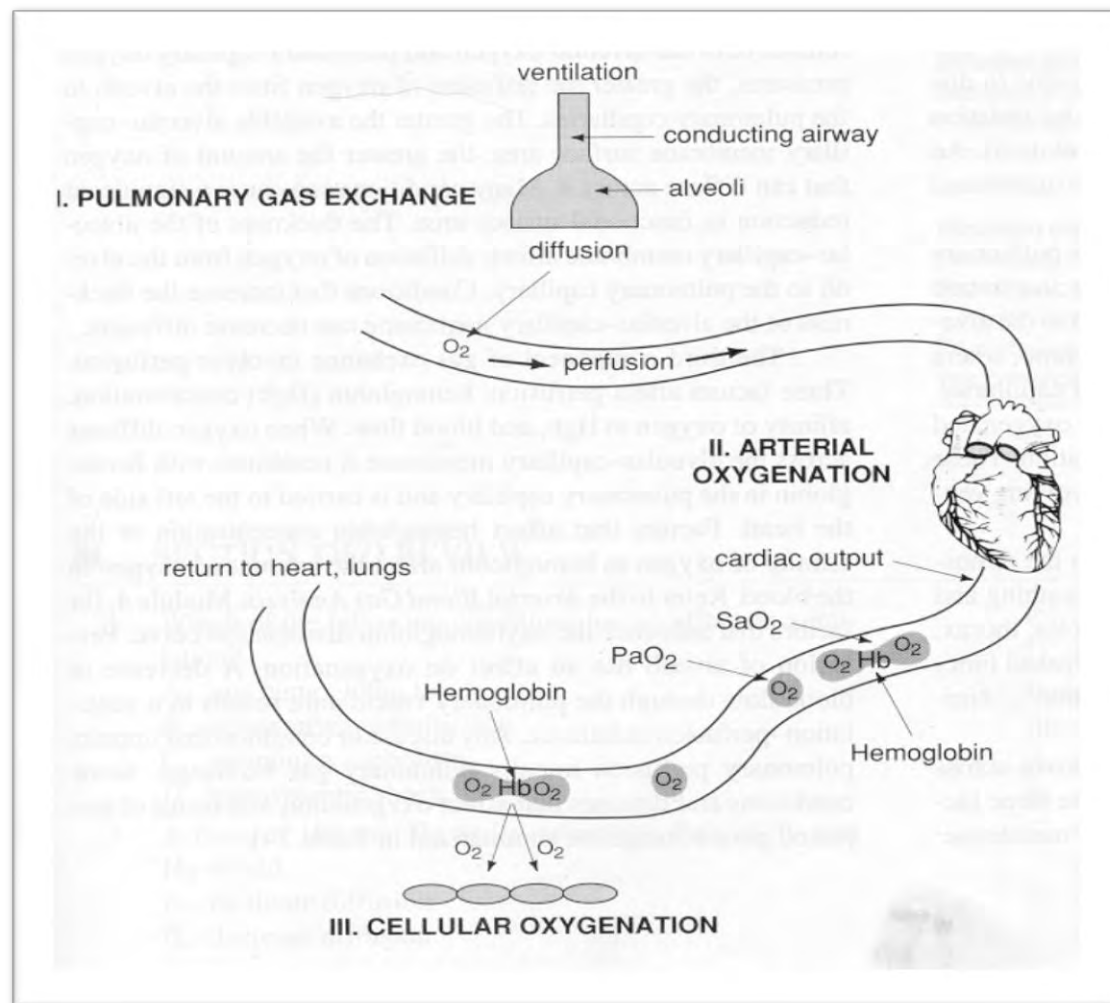
- Tidal volume may cycle close to total lung capacity (TLC), ie reduced lung compliance (increased risk of barotrauma).
- Increased effort required for ventilator triggering or initiation of gas flow.
- Increased work of breathing.
- Decreased preload and cardiac output.

8. OXYGENATION

Tissue oxygenation depends on oxygen delivery and consumption. Three processes involved in oxygenation are:¹⁷

- Intake of Oxygen (Pulmonary Gas Exchange or Pulmonary Ventilation).
- Delivery of oxygen to the cells (Arterial Oxygenation) – DO_2
- Oxygen consumption -use of oxygen by the cells for metabolism (Cellular Oxygenation) – VO_2 .

The components of oxygenation (Source: Kidd & Wagner, 1997, pg:159)



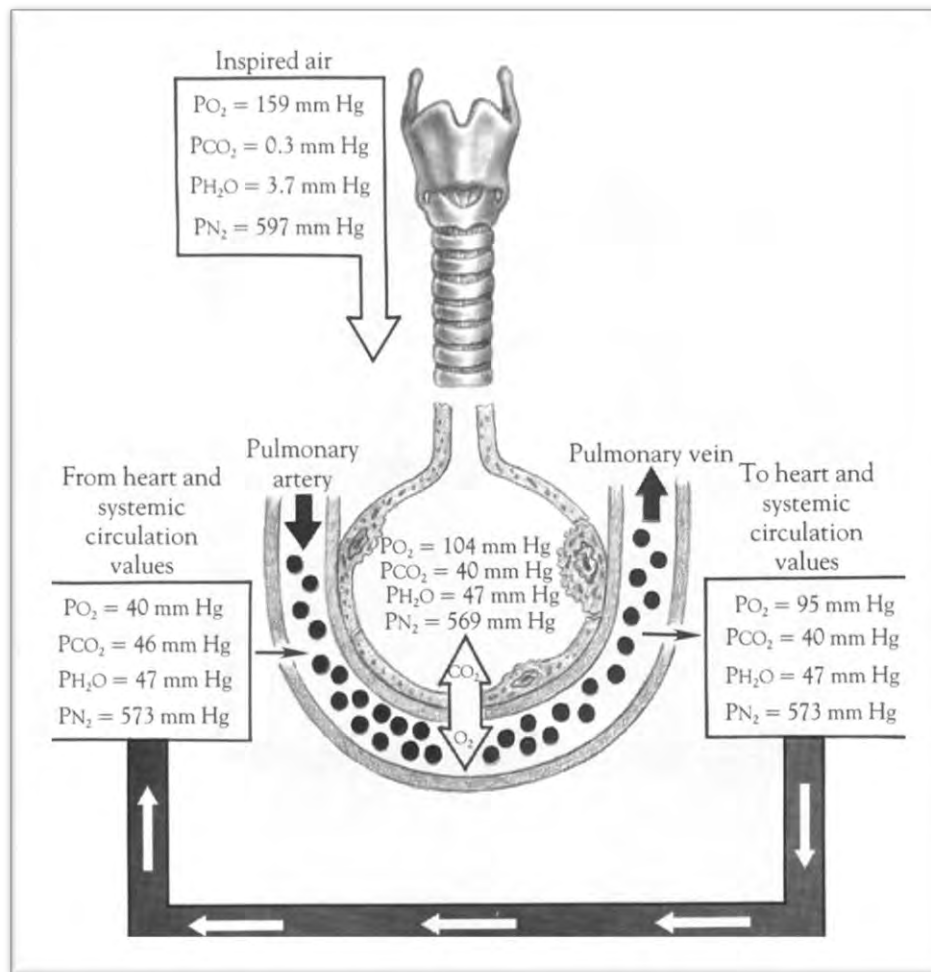
Pulmonary gas exchange:^{17,50}

Pulmonary gas exchange involves breathing O_2 in so it can be exchanged at an alveolar level with CO_2 . The O_2 is then attached to haemoglobin in the pulmonary capillaries.

Pulmonary gas exchange is dependent on three processes:

- Ventilation
- Diffusion – determined by:
 - Surface Area
 - Thickness
 - Length of exposure
- Perfusion- determined by:
 - Haemoglobin
 - Affinity of O_2 to haemoglobin
 - Blood flow /Cardiac output

Process of Respiration (Source: Thelan et al, 1998, pg:619)



Delivery of Oxygen (DO_2)¹⁷

Arterial oxygenation is the process of delivering oxygen to the cells (Kidd & Wagner, 1997).

Oxygen delivery to the cells depends on:

- Cardiac output
- The amount of Haemoglobin (Hgb).
- Oxygen saturation
- Oxygen binding capacity (normally 1.34 mls of oxygen binds to every 1g of haemoglobin)

Each of these factors need to be optimised to improve oxygen delivery.

The relationship between the oxygen carrying capacity of blood, cardiac output, concentration of haemoglobin and saturation of haemoglobin is expressed in the following equation.

$$DO_2 = \text{Cardiac output} \times \text{Hgb concentration} \times \text{Oxygen Saturation} \times 1.34$$

There is also a relationship between partial pressure of oxygen dissolved in the plasma (PaO_2) and saturation of Hgb with oxygen (SaO_2) on the oxyhaemoglobin curve.

Oxyhaemoglobin – dissociation curve.^{13,17}

Oxygen is transported in the blood in two ways. The majority of the blood (97%) is bound to haemoglobin, whereas the other 3% is dissolved in the plasma. Each haemoglobin molecule can combine with four oxygen molecules. When four oxygen molecules are bound to a haemoglobin it is said to be fully saturated. The extent to which haemoglobin is bound to haemoglobin depends largely on the PO_2 of blood. However, the relationship is not linear. When oxygen saturation is plotted against the partial pressure of oxygen an S-shaped oxygen haemoglobin curve results.

The oxyhaemoglobin curve shows the relationship between, percent of haemoglobin saturation (SaO_2) and partial pressure of arterial oxygen (PaO_2). A normal oxyhaemoglobin curve assumes certain parameters. These being a pH of 7.4; temperature of $37^\circ C$ and $PaCO_2$ of 40mmHg. At the arterial end of the curve the slope is gentle; this reflects the alveolar - capillary O_2 transfer site. PaO_2 drops markedly with little change in SaO_2 at this point. At the steeper portion of the curve, venous disassociation occurs as O_2 leave the haemoglobin and is transferred to the cells. At this site SaO_2 drops dramatically with little change in PaO_2 .

Oxygenation at the cellular level depends on the ability of O_2 to bind with and be released from the Hb in the blood.

P_a represents the partial pressure at which haemoglobin is 50% saturated

A shift to the left increases the affinity of Hb for O_2 with decreased tissue oxygenation and P_a .

Causes of a shift to the left:

- Alkalosis
- Hypothermia
- Decrease 2, 3 DPG - Massive blood transfusion and carbon monoxide poisoning
- Hypocapnia

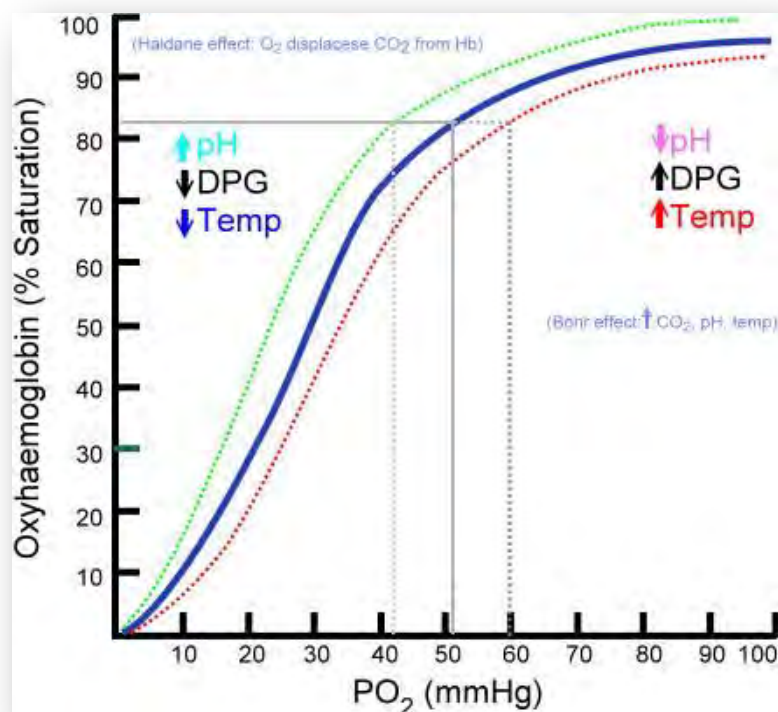
A shift to the right decreases the affinity of Hb for O_2 with increased tissue oxygenation and P_a .

Causes of a shift to the right:

- Hyperthermia
- Acidosis
- Increased 2, 3 DPG - A by product of metabolism, hyperthyroidism and hypoxia
- Hypercapnia

Oxyhaemoglobin – dissociation curve

(Source:http://en.wikipedia.org/wiki/File:Oxyhaemoglobin_dissociation_curve.png)



Oxygen Consumption $\dot{V}O_2$ – Cellular Oxygenation^{13,17}

Once oxygen is delivered to the cells it has to be utilised. It is essential for aerobic respiration.

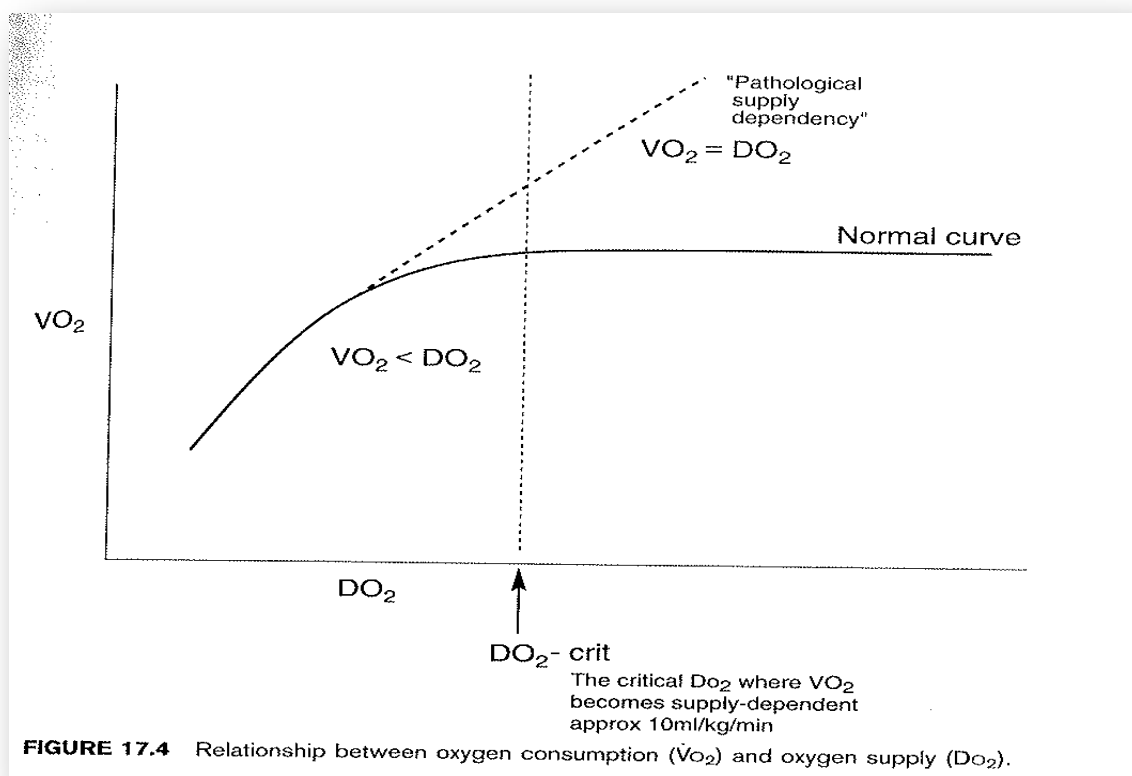
Aerobic metabolism: Oxygen combines with food (Kreb Cycle) to form CO_2 , H_2O and ATP.

Anaerobic metabolism: Glucose in the absence of O_2 is broken down to form ATP, pyruvate and lactic acid.

In normal conditions DO_2 is well matched to metabolic requirements. The oxygen consumption ($\dot{V}O_2$) is about 25% of DO_2 , which results in an extraction ratio of 0.25. This leaves a reserve and when DO_2 decreases the extraction ratio increases allowing the $\dot{V}O_2$ to remain constant. When oxygen extraction is maximum, further decrease in DO_2 will result in a fall in $\dot{V}O_2$. This is because tissue extraction cannot compensate for decrease in delivery of oxygen. When this oxygen debt occurs, anaerobic metabolism produces lactic acid. If this is not corrected tissue hypoxia will result leading to cell damage and death (Hillman & Bishop, 1996)

The use of supplemental oxygen is necessary to relieve hypoxaemia. This is combined with measures to:

- Reduce oxygen requirements – cooling, mechanical ventilation, sedation, paralysis
- Increase DO_2 - correct anaemia, low cardiac output and factors which shift the oxyhaemoglobin curve to the left.



(Source: Hillman & Bishop, 1996)

Oxygen Failure¹⁷

Oxygen failure is a respiratory crisis in which the primary problem is hypoxemia, $PaO_2 \leq 60\text{mmHg}$.

Hypoxemia (PaO_2) is defined as inadequate oxygen in arterial blood.

Hypoxia is defined as decreased oxygen supply to the cells or tissues.

PaO_2 , SaO_2 & SvO_2 are used to measure adequate oxygenation of the body. PaO_2 & SaO_2 can be measured by arterial blood gas, SvO_2 is mixed venous oxygen measured from a venous sample – true

mixed venous is obtained from the distal lumen of the pulmonary artery catheter. SpO₂ is measured using pulse oximetry.

Effective oxygenation of the blood and tissues in the body are governed by the following factors:

- Sufficient oxygen supply in the inspired air FiO₂.
- Sufficient ventilation to ensure oxygen is delivered to the lung alveoli.
- Adequate cardiac output (CO) to carry the oxygenated blood to the tissues.
- Adequate haemoglobin (Hgb) levels to carry sufficient oxygen in the blood.
- Immediate release of the Hgb molecule from oxygen and its diffusing ability to the tissues.

Oxygen delivery to the tissues can be compromised if any of the above factors are insufficient.

The four types of hypoxia are¹⁷:

1. Hypoxic hypoxia - Occurs when there is a decreased arterial blood saturation of oxygen (PaO₂). Hypoxic hypoxia may occur anywhere along the oxygen cascade from when the oxygen is inspired to when it reaches the mitochondria, the power house of the cell.

It may be due to the following:

- Decrease in inspired oxygen.
- Alveolar hypoventilation..
- Diffusion problems.
- Ventilation/Perfusion mismatch (the most common cause of hypoxia - *figure 4*).
- Shunt.
- Increased oxygen consumption.
- Hypoxic pulmonary vasoconstriction.

2. Anaemic Hypoxia - Occurs when there is a decrease or defective Hgb or Hct

3. Circulatory or Stagnant Hypoxia - Occurs as a result of decreased cardiac output or obstruction, which impedes oxygen getting to the tissues

4. Histotoxic Hypoxia - in which quantity of oxygen reaching the cells is normal, but the cells are unable to use the oxygen effectively, due to disabled oxidative phosphorylation enzymes. Hence intracellular oxygen utilisation is affected. Cyanide toxicity is one example.

Assessment of Oxygenation¹⁷

Signs and symptoms of hypoxia relate to anything that indicates reduced oxygen to the tissues.

Signs and symptoms include:

- Tachypnoea.
- Tachycardia to bradycardia.
- Altered level of consciousness.
- Confusion.
- Irritability.
- Hypertension to hypotension.
- Cyanosis being a very late sign.

Pulse Oximetry.⁶

Pulse oximetry is a simple non-invasive method of monitoring the percentage of haemoglobin (Hb), which is saturated with oxygen. The pulse oximeter consists of a probe attached to the patient's finger, toe, ear lobe or forehead, which is linked to a computerised unit. The unit displays the percentage of Hb saturated with oxygen together with an audible signal for each pulse beat, and calculated heart rate. A pulse wave related to flow is displayed graphically. SpO₂ is generally maintained > 94%. In certain patients, such as those with CAL lower saturation levels of 88-92% are the goal.

9. ARTERIAL BLOOD GASES

Measurements of arterial blood gases (ABGs) are obtained to assess adequacy of oxygenation and ventilation, to evaluate acid-base status by measuring the respiratory and non-respiratory components, and to monitor effectiveness of therapy.

Arterial blood gas measures the following parameters: pH, PaO₂, PaCO₂. The ABG derives the base excess or deficit, HCO₃ and SaO₂.

Measured Parameters:¹⁷

- **pH** – this reflects the hydrogen ion concentration in the blood.
- **PaO₂** – reflects the partial pressure of oxygen in the arterial blood
- **PaCO₂** – reflects the partial pressure of carbon dioxide in the arterial blood

Derived Parameters:^{4,17}

- **Base excess or deficit** - This test quantifies the patient's total base excess or deficit so that clinical treatment of acid-base disturbances (specifically those that are not respiratory in nature) can be initiated. It is also referred to as the *whole blood buffer base* and is the sum of the concentration of buffer anions (in milliequivalents per litre) contained in whole blood. These buffer anions are the bicarbonate ion (HCO₃⁻) present in plasma erythrocytes, and the haemoglobin, plasma proteins, and phosphates in plasma and red blood cells.
- **HCO₃⁻** – reflects the bicarbonate content in the plasma of arterial blood
- **SaO₂** – this is the haemoglobin oxygen saturation of arterial blood.

Oxygenation assessment on ABG^{4,17,50}

When assessing oxygenation on the ABG, the A-a gradient is used to assess presence of hypoxemia.

The **Alveolar-arterial gradient** (A-a gradient), is a measure of the difference between the alveolar concentration of oxygen and the arterial concentration of oxygen. It is used in diagnosing the presence of shunt. This test gives an approximation of the difference in the partial pressure of O₂ between the alveoli and arteries. The alveolar-to-arterial (A-a) oxygen gradient assesses oxygen delivery by comparing the arterial oxygen level to the theoretical maximal alveolar oxygen level. It identifies the cause of hypoxemia and intrapulmonary shunting as either (1) ventilated alveoli but no perfusion, (2) unventilated alveoli with perfusion, or (3) collapse of both alveoli and capillaries.

In general the A-a gradient can be calculated by: (Hillman & Bishop, 1996)

$$\text{A-a gradient} = \text{PA}_{\text{O}_2} - \text{Pa}_{\text{O}_2}$$

Where:

- **PA_{O2}** = alveolar PO₂ (calculated from the alveolar gas equation)
- **Pa_{O2}** = arterial PO₂ (measured in arterial blood A-a gradient)

$$\text{PA}_{\text{O}_2} = \text{FiO}_2 \times (\text{pAtm} - \text{pH}_2\text{O}) - \frac{\text{PaCO}_2}{\text{R}}$$

PA_{O2} = Alveolar pressure of O₂

FiO₂ = Fraction of inspired oxygen

pAtm = Atmospheric Pressure (760mmHg @ sea level)

pH₂O = Water Vapour pressure (47mmHg @ 37°C)

R = Respiratory Quotient = 0.8 (ratio of carbon dioxide production to oxygen consumption)

PaCO₂ = Partial pressure of carbon dioxide measured on the arterial blood gas

PaO₂ = Partial pressure of oxygen measured on the arterial blood gas

The normal A-a gradient varies with age and ranges from 7-14mmHg when breathing room air.

Normal A-a gradient = (Age+10) / 4. A-a increases 5 to 7 mmHg for every 10% increase in FiO₂

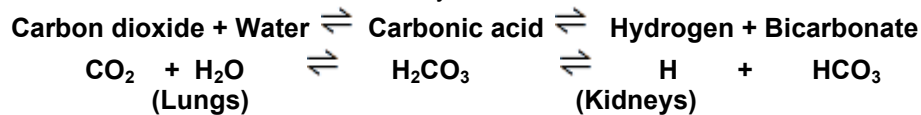
On 100% oxygen, the A-a gradient is 25-65mmHg.

An abnormally increased A-a gradient suggests a defect in diffusion, V/Q (ventilation/perfusion ratio) mismatch, or right-to-left shunt.

Acid base imbalance assessment of ABG^{4,17}

Buffers. The body will always try to maintain homeostasis and restore the pH to normal. This process is called compensation and is controlled by the use of buffers. Buffers are weak acids or bases that prevent sudden change in pH. Examples of buffer systems are:

- Phosphate Buffer System
- Hb/OxyHb System - Hb releases O₂ and attracts H ions
- Protein buffer system - Carboxyl (COO-H⁺) and Amine (NH³⁻) groups.
- Bicarbonate - Carbonic Buffer System



Acidosis (pH < 7.35) is the abnormal increase in H ions (acid) or loss of HCO₃⁻ (base).

Alkalosis (pH > 7.45) is the abnormal increase in HCO₃ (base) or loss of H ions (acid).

Acidosis and alkalosis may be **RESPIRATORY** or **METABOLIC** in nature:

Conditions leading to Acidosis/Alkalosis involve a multitude of physiological processes such as:

- Respiratory/renal dysfunction
- Disturbances of tissue oxygenation, circulation
- Substance ingestion
- Electrolyte loss/gain

Respiratory component - PaCO₂ can be elevated or decreased.

A PaCO₂ that is **elevated** could indicate a **respiratory acidosis** due to hypoventilation (e.g. retention of CO₂).

A PaCO₂ that is **decreased** could indicate a **respiratory alkalosis** due to hyperventilation e.g. loss of CO₂

Metabolic Component - HCO₃ can be elevated or decreased.

A HCO₃ that is **decreased** could indicate **metabolic acidosis** due to acid being added to the system thus HCO₃ being used up or HCO₃ being lost (e.g. diarrhoea, renal failure – where the Kidney excrete H⁺, ketoacidosis, or Salicylate poisoning)

A HCO₃ that is **elevated** could indicate a **metabolic alkalosis** due to acid being lost (e.g. vomiting of GI, suction, diuretic therapy and loss of K⁺) or HCO₃ gained.

Compensation (Hillman & Bishop, 1996)

- A PaCO₂ that is **elevated** could also indicate a compensatory response to **metabolic alkalosis**. In this case the pH will be returning to normal.
- A PaCO₂ that is **decreased** could indicate a compensatory response to **metabolic acidosis**. In this case the pH will be returning to normal.
- A HCO₃ that is **decreased** could indicate a compensatory response to **respiratory alkalosis**. In this case the pH will be returning to normal.
- A HCO₃ that is **elevated** could indicate a compensatory response to **respiratory acidosis**. In this case the pH will be returning to normal.

Interpretation

- Oxygenation
- Look at PaO₂ & SaO₂ & FiO₂ – Calculate the A-a gradient.
- Acid-Base Status
- Look at the PH - Acidosis - PH < 7.35
- - Alkalosis - PH > 7.45
- Acidosis - Respiratory or Metabolic?
- Respiratory Acidosis - PaCO₂ > 45mmHg (PH < 7.35)
- Metabolic Acidosis - HCO₃ < 22mmHg (PH < 7.35)
- Alkalosis - Respiratory or Metabolic?
- Respiratory Alkalosis - PaCO₂ < 35mmHg (PH > 7.45)
- Metabolic Alkalosis - HCO₃ > 28mmHg (PH > 7.45)

Table 11.22 Some simple rules for blood gas analysis interpretation

Acid–base disorder	Rule*
Primary acute respiratory acidosis	HCO ₃ ⁻ increases 1 mmol/L per 10 mmHg rise in Paco ₂ above 40 mmHg (up to 30 mmHg)
Primary chronic respiratory acidosis	HCO ₃ ⁻ increases 4 mmol/L per 10 mmHg rise in Paco ₂ above 40 mmHg (up to 36 mmol/L)
Primary acute respiratory alkalosis	HCO ₃ ⁻ decreases 2 mmol/L per 10 mmHg fall in Paco ₂ below 40 mmHg
Primary chronic respiratory alkalosis	HCO ₃ ⁻ decreases 5 mmol/L per 10 mmHg fall in Paco ₂ below 40 mmHg
Primary metabolic acidosis	Paco ₂ should be within 5 mmHg of the number denoted after the decimal point in the pH (down to a Paco ₂ 10) (e.g. pH 7.10—Paco ₂ should be 10)
Primary metabolic alkalosis	Paco ₂ should be within 5 mmHg of the number denoted after the decimal point in the pH (up to a Paco ₂ 60) (e.g. pH 7.6—Paco ₂ should be 60)

HCO₃⁻ = bicarbonate ion.
 *Assume normal Paco₂ = 40 and HCO₃⁻ = 24.
 With a pure metabolic acidosis the Paco₂ = 1.5(HCO₃⁻) + 8.
 With a pure metabolic alkalosis the Paco₂ = 0.9(HCO₃⁻) + 9.

(Source: Blackwell et al, 2007)

10. AIRWAY MANAGEMENT

Basic Airway Management

Airway management is an important first step in the care of all patients, as an obstructed airway ultimately leads to problems with oxygenation. Failure to manage a patient's airway successfully can result in severe adverse outcomes, including brain injury, myocardial injury, airway trauma, and death.

Complete and partial obstruction of the airway^{8,10}

Airway obstruction can be partial or complete. When an airway is completely obstructed, there are no breath sounds at the nose or mouth, as air is unable to move past the obstruction. When an airway is partially obstructed, noises can be heard which indicate the level of obstruction.

Partial obstruction of the airway may be indicated by subtle signs only, so thorough assessment is important. The noises that may be heard include:

- Gurgling – may indicate the presence of fluid in the mouth or upper airway.
- Snoring – may indicate pharynx is partially obstructed by the tongue.
- Crowing – may indicate laryngeal spasm.
- Inspiratory stridor – may be caused by an obstruction above or at the level of the larynx.
- Expiratory wheeze – may be caused by airway collapse during expiration (eg asthma).

Upper airway obstructions may be caused by:

- Obstruction of the pharynx by the tongue – caused by sedation or the patient being neurologically compromised.
- Vomit, secretions, blood or gastric fluid.
- Tissue swelling from trauma, allergy or infection.
- Lower airway obstructions may be caused by:
 - Laryngeal oedema – due to burns, inflammation or allergy.
 - Laryngeal spasm – due to a foreign body, airway stimulation or secretions/blood in the airway.
 - Tracheobronchial obstruction – due to secretions, inhaled gastric contents, pulmonary oedema fluid or bronchospasm.

The following information describes guidelines and equipment for effective airway management. Knowledge of this information will be required in your assessment of emergency patients. The key steps involve;

- Assess the airway.
- Clear the airway.
- Maintain an open airway.
- Assess the effectiveness of your interventions.

(As cervical spine management is assessed and managed at the same time as airway it is also included in this module.)

Assess the airway^{8,10}

Assessing a patient's airway involves the following steps.

Is the patient responsive?

When you approach the patient, first assess for responsiveness, namely:

A good initial indication that a patient is able to maintain their airway is if they are awake and can talk to you. If you walk into a patient's room and ask them how they are and they respond appropriately you will know that their airway is patent, they are breathing normally and their brain is still perfused, thus indicating no life-threatening emergency.

If a patient has a decreased level of consciousness, their ability to maintain their airway may be compromised. The most common cause of airway obstruction is when the tongue falls posteriorly into the oropharynx.

Generally, a patient with a Glasgow Coma Scale of 8 and below is unable to protect their airway and will require airway management of some description. Patients who are unable to maintain their airway are at increased risk of vomiting and aspirating GIT material into their airway/lungs.

Look

Observe the patient's chest rise and fall and evidence of respiratory effort

- Are their respirations slow or fast?
- Observe the patient's colour; look for signs of cyanosis.
- Open the patient's mouth and inspect the airway for signs of obstruction (eg vomitus, loose teeth or other foreign bodies).
- Observe how the patient is positioned.

Listen

Listen for breath sounds – are they present? Note the rate, rhythm and depth of ventilation.

- Is the patient's breathing noisy?
- What noises are coming from the patient's airway?
- Ask the patient if their voice sounds normal to them 'Dysphonia' (hoarse voice) may be heard in relation to trauma (blunt, thermal) and may lead to airway oedema.

Feel

Feel at the mouth and nose for expired air.

Feel for chest rise and fall.

Clear the airway

In a high proportion of cases, simple airway-opening manoeuvres such as 'Head Tilt Chin Lift' and 'Jaw Thrust' will relieve an airway obstruction. These should be familiar to you from Basic Life Support.

The jaw thrust manoeuvre is to be used in patients with a suspected spinal injury as the cervical spine is not hyper-extended.

Depending on the type of obstruction, the following instruments should be used:

- To remove large objects – use Magill's forceps.
- To suction fluid – use a wide-bore rigid (Yankauer) catheter.
- To remove secretions lower than the pharynx – use a flexible (Y-suction) catheter inserted down an airway adjunct (eg oropharyngeal/nasopharyngeal airways).

(Australian Resuscitation Council, 2010)



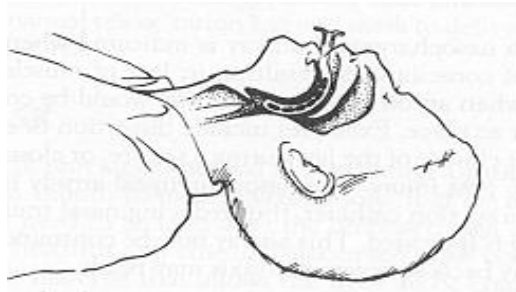
Maintain an open airway with airway adjuncts⁸

Once an open airway has been established, the physician may choose to use either an oropharyngeal or nasopharyngeal airway to make it easier to maintain an open airway. Both of these devices prevent the tongue from occluding the airway and thereby provide an open conduit for air to pass.

- Airway adjuncts are used for:
- Airway maintenance for an unconscious patient.
- Bag-valve-mask ventilation.
- Preventing the patient from biting the ET tube.
- Suctioning.

When using an oropharyngeal (Guedels) airway:

- Exercise caution when inserting.
- Measure from corner of mouth to angle of jaw.
- Insert concave up then rotated 180°.
- Remember that the Guedels airway sits in the oropharynx and induces gag by touching the soft palate, but a nasopharyngeal does not touch there.



Oropharyngeal Airway

When using a nasopharyngeal (Guedels) airway:

- Use in patients with seizure activity.
- Remember that it may cause epistaxis.
- Do not for use in patients with facial trauma or Base of Skull fractures.
- Measure from nasal nare to the pinna of ear.
- Lubricate well, bevel facing septum, direct posteriorly and rotate slightly.

Remember that this type of airway may be better tolerated in patients that have a gag reflex as it is less likely to induce gag or vomiting.

Assess the effectiveness of your interventions

Once you have cleared the airway, return to your assessment of the patient to see if there has been a change in their condition. Reassess their level of consciousness and then look, listen and feel as described above.

Following assessment of the airway and implementation of airway management strategies, apply oxygen using an oxygen mask and/or bag-valve-mask (BVM) ventilation.

When using an oxygen mask:

- The oxygen delivery device and amount delivered (L/min) will be guided by the patient's current condition and the reason for applying the oxygen.
- If the patient is not breathing after the airway has been established, ventilation will need to be commenced via BVM ventilation, which allows for oxygenation and ventilation of patients until a more definitive airway can be established

11. OXYGEN THERAPY AND DELIVERY SYSTEMS

Oxygen is the first line management for hypoxia. Oxygen therapy is indicated whenever tissue oxygenation is impaired.

Increased FiO_2 can be delivered with the following devices: ^{10,17}

Nasal Cannulae

Delivers oxygen through a low flow oxygen delivery system. It allows supplemental oxygen to be given whilst your patient is eating or talking. Maximum oxygen flow should not exceed 4 lpm.

Hudson Mask

Delivers concentrations of 35 – 65%, depending on the patient's respiratory rate and tidal volume. It should never be used at flow rates of less than 6L/min or rebreathing will occur.

Venturi Mask

Is suited for patients who need precise O_2 concentrations of between 24 – 50%. FiO_2 is adjusted using the gas flow.

Oxygen Reservoir Mask (Non-rebreather Mask)

Delivers 90 – 100% O_2 , provided there is no leak in the system. Ensure that the reservoir bag does not collapse with inspiration. It is a precise method of delivering high O_2 concentration for a short period.

Bag-Valve Mask Ventilation (BVM).

When using bag-valve-mask ventilation, remember that BVM ventilation requires a good seal and a patent airway.

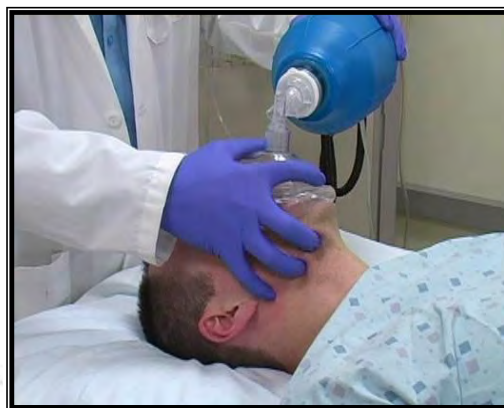
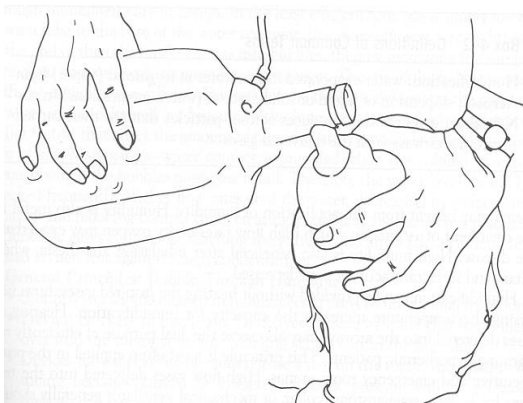
To create a good seal while maintaining the airway, use the thumb and index finger to apply downward pressure on the mask then use 3rd, 4th and 5th fingers to pull patient's jaw up and open the airway.

Airway adjuncts can assist the operator maintain a seal.

Certain factors predict difficult BVM ventilation. These include the presence of facial hair, lack of teeth, a body mass index (BMI) greater than 26, age older than 55 years, and a history of snoring.

Obtaining a good seal with the mask while maintaining the airway to allow for ventilation, is a skill that takes practice to master.

Administer one breath every 4 seconds.



www.procedureconsult.jp

12. TRACHEAL INTUBATION

When physical manoeuvres and oro/nasopharyngeal airways are unsuccessful in establishing and maintaining a patent airway or the patients level of consciousness is compromised, endotracheal intubation is required.

Indications

- Upper airway obstruction (secondary to swelling, trauma, bleeding, infection, space occupying lesion).
- Decreased level of consciousness (GCS < 9).
- Apnoea; requiring mechanical ventilation.
- Prevention of aspiration.
- Respiratory distress/failure requiring mechanical ventilation.
- To enable tracheal suctioning.

Contraindications

- Where a valid order for 'Do not resuscitate' or 'Do not intubate' exists and there have been no changes to the patient's circumstances since the order was made.

Precautions

- Potential spinal cord injury.
- Previous intubation difficulty, adverse drug reactions.
- Full stomach with the risk of regurgitation and pulmonary aspiration of gastric contents.
 - Resuscitation trolley and Difficult Intubation Trolley
 - C-MAC with Blades C-MAC (default blade) & C-MAC D (for potential / known difficult airway).
 - Standard personal protection equipment

Prepare the following equipment and drugs⁸

Airway	Breathing
<ul style="list-style-type: none"> ▪ Airway masks: small, medium , large ▪ Oropharyngeal airways: Guedel airways: sizes 2, 3, 4. ▪ Nasopharyngeal airways: size 6, 7. ▪ Laryngoscope Handle & laryngoscope blades size #3 & #4 ▪ Alternate blades: C-MAC & C-MAC D blade, Airtraq ▪ Endotracheal tubes: 7.0, 8.0, 9.0mm ▪ LMA ▪ Bougie (Frova airway intubating catheter) ▪ Introducer ▪ Magill Forceps 	<ul style="list-style-type: none"> ▪ Bag Valve Mask device connected to 15L oxygen ▪ Functioning High wall suction attached to Yankauer sucker ▪ EtCO2 monitoring ▪ Stethoscope for auscultation <p style="text-align: center;">Other</p> <ul style="list-style-type: none"> ▪ 10ml syringe ▪ Lubricant ▪ Tape for securing ETT ▪ Flex Tube ▪ Closed suction for ETT
Circulation	Drugs
<ul style="list-style-type: none"> ▪ Functioning IV access ▪ IV fluid on a pump set ▪ 0.9% sodium chloride (20ml flush) 	<p>Anaesthetic / sedative agent : <i>(these are guidelines only, anaesthetic induction agents are only to be used by those trained in their use)</i></p> <ul style="list-style-type: none"> ▪ Propofol – 200mg/20ml – draw up neat. Administer 1-2mg/kg (titrate dose). ▪ Thiopentone – 500mg vial – draw up in 20ml H₂O for injection. Administer 1-3mg/kg.

	<ul style="list-style-type: none"> ▪ Midazolam – 5mg/5ml – draw up neat. Administer 1-2mg bolus (titrate dose). ▪ Fentanyl – 100micrograms/2ml – draw up 100-200micrograms neat. Administer 100-200 micrograms. ▪ Ketamine – 200mg/2ml – administer 0.5 to 2mg/kg ▪ (Consider and draw up infusions of drugs to maintain sedation and analgesia post intubation. Eg: Propofol & Fentanyl). <p>Muscle relaxant:</p> <ul style="list-style-type: none"> ▪ Suxamethonium – 100mg/2ml – draw up neat and administer 1-1.5mg/kg (avoid if K+ > 6.0mmol /L) ▪ Vecuronium 10mg/10ml – draw up in 10ml H₂O for injection. Administer 0.1mg/kg. ▪ Rocuronium 50mg/5ml – draw up neat. Administer 0.7mg/kg. <p>Vasopressor:</p> <ul style="list-style-type: none"> ▪ Metaraminol – 10mg/1ml – draw up 10mg in 20ml H₂O for injection, administer 0.5mg increments.
--	---

Prepare Patient:⁸

- Where possible, inform patient and family of the need for intubation, assess for issues of consent, and inform that speech is not possible whilst the patient is intubated.
- Assess patient for potential difficulty of intubation. Use the MACOCHA³ score to assess difficulty.

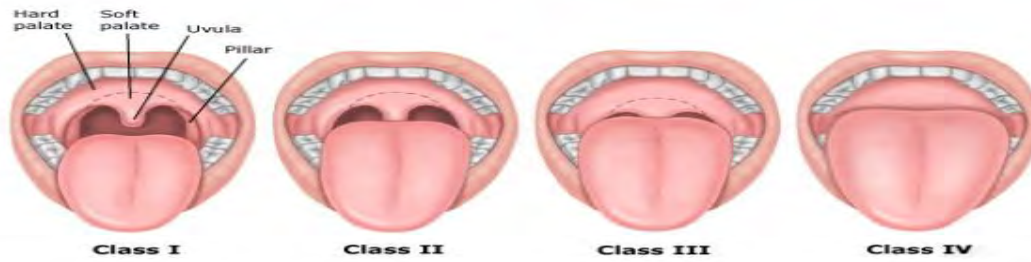
TABLE 5. MACOCHA SCORE CALCULATION WORKSHEET

	Points
- Factors related to patient	
Mallampati score III or IV	5
Obstructive Sleep Apnea Syndrome	2
Reduced mobility of Cervical spine	1
Limited mouth opening <3cm	1
- Factors related to pathology	
Coma	1
Severe Hypoxemia (<80%)	1
- Factor related to operator	
Non Anesthesiologist	1
Total	12

M. Mallampati score III or IV
A. Apnea Syndrome (obstructive)
C. Cervical spine limitation
O. Opening mouth <3cm
C. Coma
H. Hypoxia
A. Anesthesiologist Non trained

Coded from 0 to 12
0 = easy
12 = very difficult

8 -12 = High risk

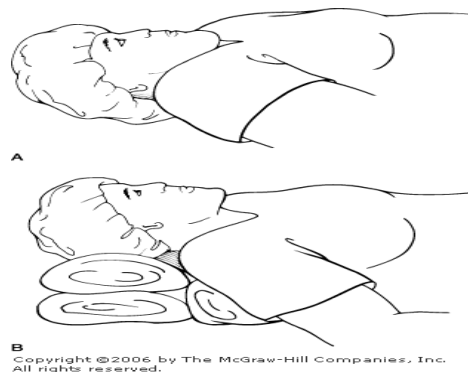


Mallampati Score

- Assess for ease of BVM ventilation, intubation, LMA insertion, cricothyroidotomy.
- Ensure adequate pre-oxygenation (3 minutes is feasible)– SpO₂ >90%
- Position patient optimally:
 - Sniffing position



- For obese patients, they will need to be elevated so that the tragus is at the level of the sterna notch.



- For patients with suspected spinal injuries, remove cervical collar and maintain manual in-line stabilisation (MILS).
- Can the patient's condition be optimised any further before intubation?
- Draw up what will be used to maintain sedation and analgesia post intubation (eg: propofol infusion and fentanyl infusion).

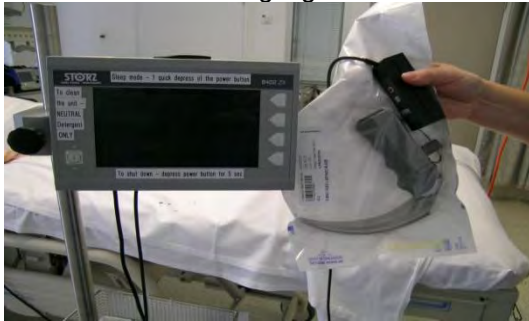
Prepare Equipment:⁸

- Ensure patient is monitored: ECG, blood pressure, SpO₂, EtCO₂.

A - Airway equipment (MABLES)

- M – Mask – appropriate size: small, medium, large
- A – Airway – oropharyngeal, nasopharyngeal, LMA (Laryngeal mask airway).
- B – Bougie. Have blue bougie (Frova airway intubating catheter) available – lubricate prior to insertion

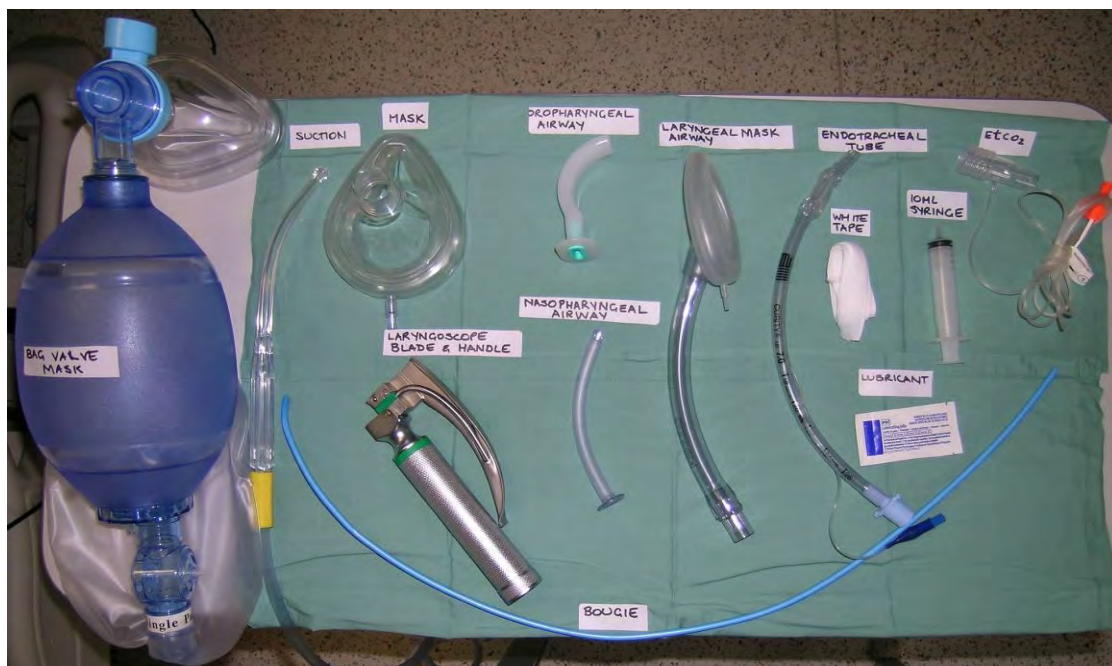
- L - CMAC, CMAC D Laryngoscope handle and blades & Airtraq. Alternate blades: Laryngoscope handle and Blades (Size 3 & 4), Connect blade to laryngoscope, assess that the light functions and the bulb is secure within the fixture.
- E – Endotracheal tubes (Inflate cuff to check for leak, deflate cuff). Lubricate the distal end of the ETT.
 - ⇒ adult male 8.0mm
 - ⇒ adult female 7.0mm
 - ⇒ adolescent/small adult 6.0 – 7.0mm
 - ⇒ white tape to secure ETT, 10ml syringe, lubricant
- S – Suction: working high wall suction connected to Yankauer sucker, surgical cricthyroidotomy kit.



C MAC



C MAC D BLADE



B – BVM (Bag Valve Mask) device

C – Circulation – ensure adequate IV access, fluid primed on pump set

D – Drugs

Anaesthetic / sedative agent: (dose needs to be adjusted for a lower dose in shocked patients and the elderly).

- Propofol – 200mg/20ml – draw up neat. Administer 1-2mg/kg (titrate dose).
- Thiopentone – 500mg vial – draw up in 20ml H₂O for injection, administer 1-3mg/kg (lower dose for shocked and elderly patients).
- Midazolam – 5mg/5ml – draw up neat. Administer 1-2mg (titrate dose).

- Fentanyl – 100micrograms/2ml – draw up 100-200micrograms neat. Administer 100-200 micrograms.
- Ketamine – 200mg/2ml – Administer 0.5 to 2mg/kg

Muscle relaxant:

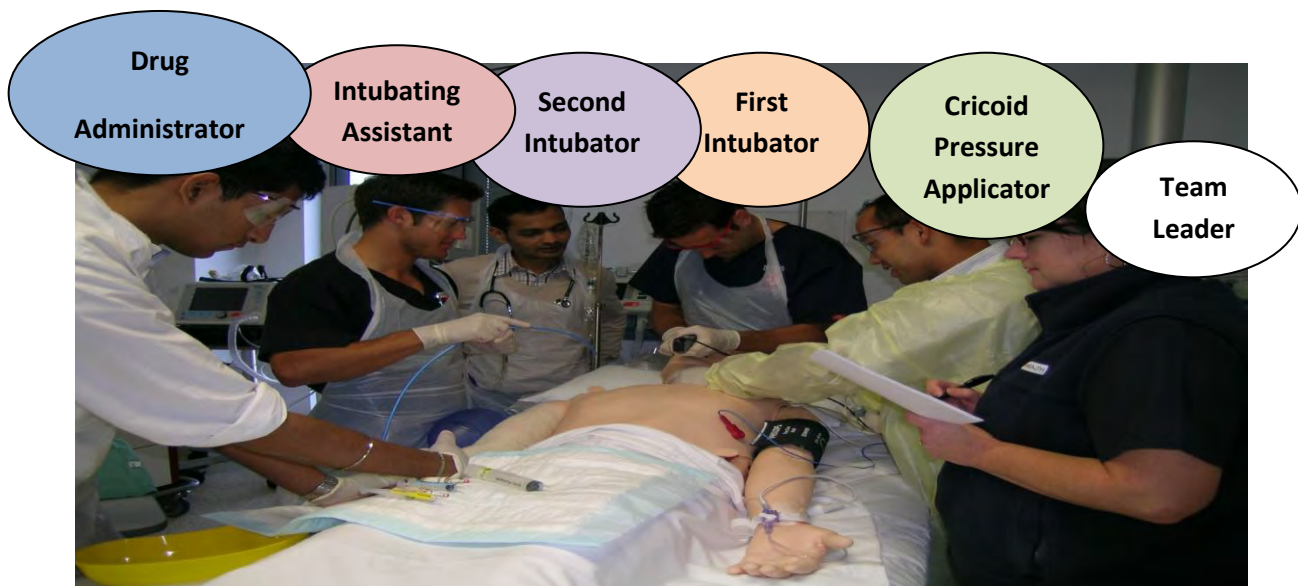
- Suxamethonium – 100mg/2ml – draw up neat and administer 1 -1.5mg/kg (avoid if K+ > 6.0mmol /L)
- Vecuronium 10mg/10ml – draw up in 10ml H₂O for injection. Administer 0.1mg/kg.
- Rocuronium 50mg/5ml – draw up neat. Administer 0.7mg/kg.

Vasopressor:

Metaraminol – 10mg/1ml – draw up 10mg in 20ml H₂O for injection. Administer 0.5mg increments to reverse vasodepressor effect of induction agents.

Prepare Team⁸

- It is essential to have two operators experienced in intubation present for planned intubation (the exception being emergency situations where if possible it is preferable to have two operators experienced in intubation).
- Allocate roles appropriately – team leader, first intubator, second intubator, cricoid pressure applicator, intubating assistant, drug administrator (as a minimum 4 people are needed).
- Have an escalation plan in place – Plan A, Plan B and Plan C (Refer to Appendix 2).
- Part of the plan should include who to contact in case of difficulty with intubation – ICU SR →ICU Staff Specialist →Duty Anaesthetist.

**Prepare for Difficulty⁸**

- If view not optimal consider the following optimisation steps:
 1. Release cricoid pressure.
 2. Insert laryngoscope deeper
 3. Pull laryngoscope out further
 4. BURP (External laryngeal manipulation, Backwards Upwards Right Pressure). Apply pressure over the thyroid cartilage.
 5. Use bougie
 6. Reposition head (place folded sheet under head to improve sniffing position).
 7. Use alternate laryngoscope blade – CMAC, CMAC D blade, or Airtraq.
 8. Change intubator
 9. Ensure adequate suction to clear secretions
- Is it possible to wake patient up if airway is difficult?

- Access to difficult airway trolley and awareness of difficult airway drill
- Are there any specific complications anticipated?

Predicted difficult airway / Grade III to IV Intubation

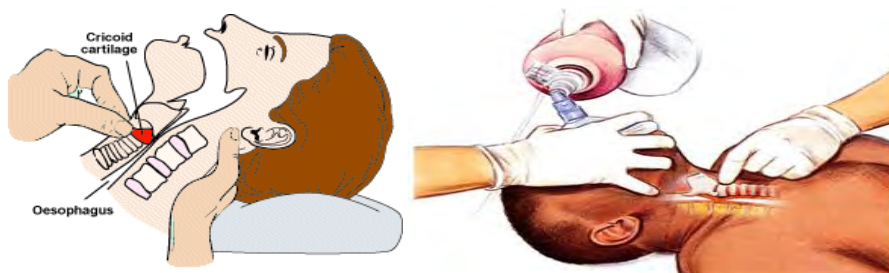
The following options should be considered for a patient with known difficult airway:

- Awake direct laryngoscopy
- Awake fiberoptic bronchoscopy
- Gaseous induction maintaining spontaneous ventilation (only done in OT).
- Awake surgical airway.

All of the above options must be considered only after consultation with an experienced airway operator.

Procedure: method

- Put on protective eyewear, mask, gloves and gown.
- Pre-oxygenate the patient with 100% oxygen by self inflating Bag-Valve-Mask device, for 3-5 minutes if possible.
- Position the patient flat with the head raised on a small pillow, tilted backwards (sniffing position).
- ***If cervical spine injury is suspected, an assistant should stabilise the head and neck in a neutral position, maintaining manual in-line immobilisation. Intubation should then be performed without flexion or extension. A Hard Collar if present is temporarily removed for intubation. Avoid the use of suxamethonium if SCI > 24hours.***
- Medical Officers (on RN as directed by MO): administer induction agent and then neuromuscular blocking agent (as per the guidelines for those drugs). Flush medications well with sterile 0.9% sodium chloride in between administration.
- An assistant should apply cricoid pressure if requested by the person intubating the patient.



Cricoid Pressure (<http://www.tovatech.com>)

- Open the mouth with the fingers of the right hand.
- With the left hand, insert the blade into the right side of the mouth, using it to push the tongue to the left. The epiglottis should then be visible.
- Suction any secretions with a Yankauer sucker.
- Place the tip of the laryngoscope blade in the depression between the base of the tongue and the epiglottis (vallecula). Note that the technique is different for infants and small children, where the blade is inserted beyond the long floppy epiglottis.
- Lift vertically upwards to the ceiling, in the direction of the laryngoscope handle, to expose the larynx.
- Do not lever the laryngoscope on the front top teeth.
- Intubate with the introducer and rail road the tube over it under direct vision; insert the tube through the vocal cords, positioning the black marker line at the level of the cords.
- If laryngoscopy is suboptimal **follow the optimisation steps as outlined above.**
- If intubation is not possible within 30 seconds, stop and re-ventilate the patient with 100% oxygen by BVM, **and proceed to difficult airway drill**
- If intubation is successful - Remove the introducer.
- Inflate the cuff with air until a seal is obtained.
- Connect EtCO₂ and ensure tube position with capnography waveform. If in doubt or no capnography waveform remove ETT.

- Ventilate the patient with 100% oxygen by BVM device, and:
 - ⇒ Observe that both sides of the chest are moving.
 - ⇒ Auscultate in both axillae to exclude endobronchial intubation.
 - ⇒ Auscultate over the epigastrium to exclude oesophageal intubation.
 - ⇒ Check that gastric distension is not occurring.
- Cricoid pressure may be released after the person intubating confirms correct tube position and directs it can be removed.
- Note the position of ETT at teeth (cm marking on the endotracheal tube).
- Tie the tube in securely:
 - ⇒ Wind tape around the tube once, and tie a half-knot. This should be tied on the top of the tube.
 - ⇒ Pass the tape behind the head, and tie a firm reef knot at the centre of the mouth. Apply protective gauze or foam squares or commercial foam non-adherent dressing (eg Biotain dressing) to avoid lip/mouth pressure.
- Maintain adequate level of sedation after intubation to facilitate tube tolerance and ventilation compliance (RASS 0 to -1). For patients with spinal injury - re-apply hard collar at this point and release in-line neck stabilisation.
- Suction the trachea.
- Insert a nasogastric/ orogastric tube (nasogastric tube insertion is contraindicated in potential base of skull fracture).
- Confirm tube position with a chest X-Ray.
- Measure cuff pressure and record in mmHg the pressure required to maintain a seal (16mmHg – 25mmHg).

Complications^{10,17}

Failed intubation

- If intubation is not possible within 30 seconds, stop and re-ventilate the patient with 100% oxygen by BVM, **and proceed to difficult airway drill (see APPENDIX 2).**

Obstruction

- Biting, which is prevented by adequate sedation to facilitate tube tolerance.
- Herniation of the cuff, which can be prevented by avoiding overinflation and maintaining cuff pressure in the range of 16mmHg – 25mmHg (use minimal pressure required to obtain a good seal).
- Kinking of the tube.
- Blood or mucus obstruction may be avoided by using humidification (HMEF or Wet circuit humidification as clinically indicated).

Dislodgment

- Ensure tube is securely secured and positioned.
- When turning / repositioning patient, the ETT must be supported during the procedure.
- Observe patient and manage their anxiety, agitation to reduce episodes of accidental extubation.
- If tube should become dislodged, maintain Airway, Breathing and Circulation and call a MET if ICU medical staff are not present.

Malposition

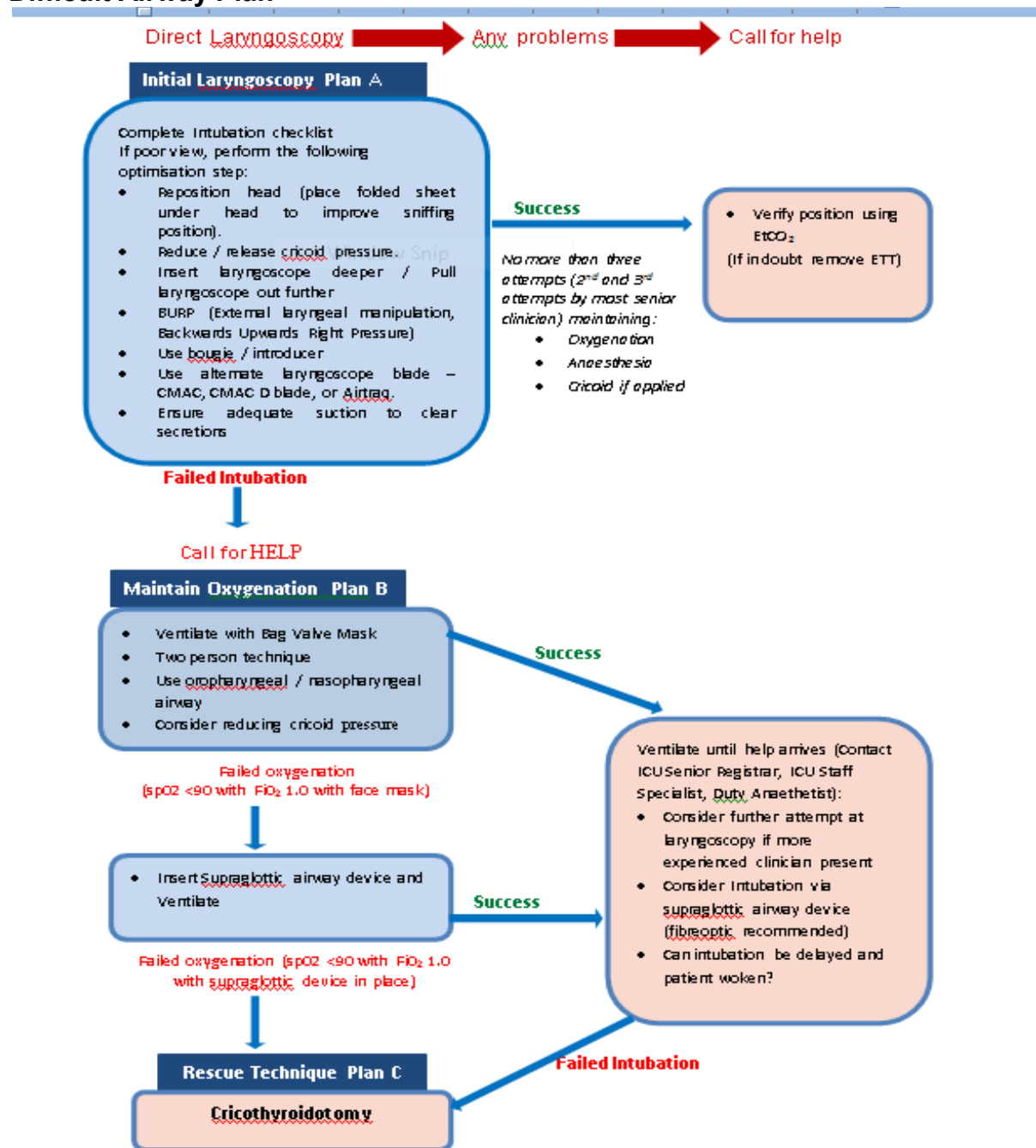
- If oesophageal intubation is suspected, remove the ETT, ventilate the patient by self inflating Bag-Valve-Mask device, and attempt intubation again as per algorithm in Appendix 2.
- Endobronchial intubation (usually the right main bronchus) – if there is decreased breath sounds on the left, assess if the tube needs to be pulled back.
- Inflation of the cuff between the vocal cords causes pain and laryngeal damage. Always confirm visually that the cuff has passed through the cords. The position of the tube is marked with a black line for this purpose.

Clinical Issues:

- Document procedure in the patient's health care record and the results of the Chest X-Ray.
- Confirm EtCO₂ capnography waveform

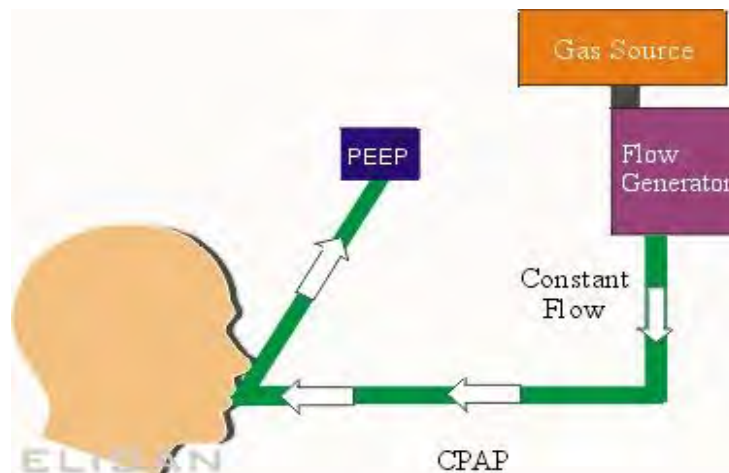
- Check placement of ETT on chest x-ray - confirm correct placement above the carina (2-4cm or in line with the upper 1/3 of the aortic knuckle)
- Document position of the tube at the teeth on the ICU flow chart.
- Ensure the ETT is well secured and connected to the mechanical ventilator which is set up with desired mode and ventilation settings.
- Document the laryngeal view at laryngoscopy.
- Maintain adequate sedation – titrate to desired RASS (Richmond agitation sedation score).
- Mouth care with regular brushing of teeth is essential. Protect the corners of the mouth from pressure from the ETT.
- Intubated patients must NEVER be left unattended

Difficult Airway Plan⁸



13. MECHANICAL VENTILATION.

Ventilation therapy is provided by non-invasive or invasive means and with positive pressure breaths. The principle of positive pressure ventilation is gas flow along a pressure gradient between the upper airways and the alveoli (<http://www.ccmtutorials.com/rs/mv/page2.htm>).



A mechanical ventilator is a machine that generates a controlled flow of gas into a patient's airways. Oxygen and air are received from cylinders or wall outlets, the gas is pressure reduced and blended according to the prescribed inspired oxygen tension (FiO_2), accumulated in a receptacle within the machine, and delivered to the patient using one of many available modes of ventilation.⁷

Gas flow is actively delivered to the lungs with the volume delivered being dependant on inspiratory time, gas flow and pressure applied at the airway.

The magnitude, rate and duration of flow are determined by the operator. Flow is either volume targeted and pressure variable, or pressure limited and volume variable. The pattern of flow may be either sinusoidal (which is normal), decelerating or constant. Lung elasticity and chest wall characteristics determine compliance.⁷

The two main goals of mechanical ventilation, is to facilitate ventilation and to facilitate oxygenation. The treatment for improving ventilation is by increasing alveolar ventilation, which is achieved by increasing the rate of breathing and the Tidal Volume.

Causes of failure to oxygenate are:⁷

- Decreased alveolar oxygen tension
- Reduced O_2 diffusion capacity
- Ventilation perfusion mismatch

The treatment for failure to oxygenate includes increasing FiO_2 , restoration and maintenance of lung volumes, using recruitment manoeuvres and PEEP to increase baseline airway pressures.

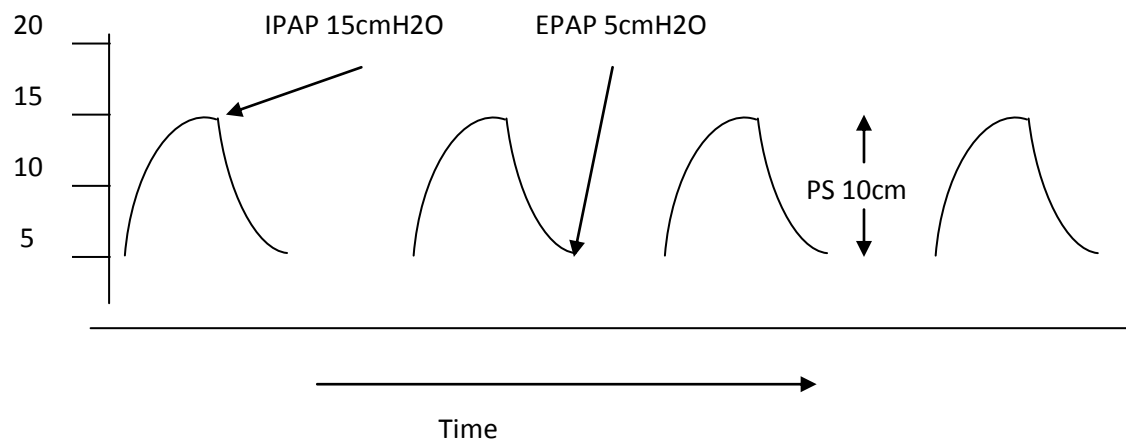
Non-Invasive ventilation

NIV delivers mechanical ventilatory support to the spontaneously breathing patient, who is able to protect their airway in the absence of endotracheal intubation. A well-fitting mask over the face or nose is used to provide either CPAP (Continuous Positive Airway Pressure) or bi-level support (BiPAP) which assists both the inspiratory and expiratory phases of breathing. BiPAP can actively assist respiration through augmentation of alveolar ventilation²³.

Bi level positive airway pressure (BiPAP) is a mode of NIPPV that supports both the inspiratory and expiratory phases of spontaneous breathing. A positive pressure known as **IPAP** (inspiratory positive airway pressure) is generated when the patient initiates a breath. IPAP increases the patient's tidal volume and supports alveolar ventilation. When exhalation commences a pressure is applied at end expiration (**EPAP**: expiratory positive airway pressure), otherwise known as CPAP/PEEP. EPAP increases functional residual capacity of the lungs and decreases airway closure. Areas of atelectasis can be re-expanded and fluid accumulation can be prevented/reduced. This aids in improved gas exchange and increase in arterial oxygen levels.²³

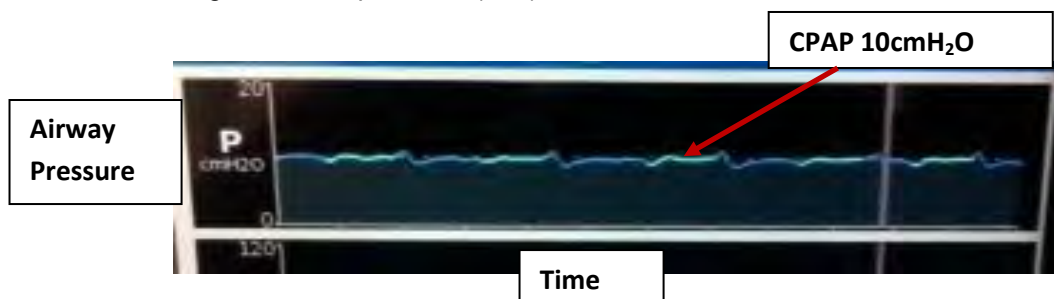
The difference between IPAP and EPAP is commonly referred to as Pressure Support (**PS**). $IPAP - EPAP = PS$.

Pressure support is a preset amount of inspiratory pressure that augments the patient's spontaneous inspiratory breath.



Continuous positive airway pressure (CPAP) Continuous positive airway pressure (CPAP) is a form of Non Invasive Ventilation (NIV) which provides positive pressure via a mask throughout the respiratory cycle (i.e. on inhalation and exhalation) to the spontaneous breathing patient who has sufficient respiratory drive and muscle strength. A supply of gas provides the positive pressure and a restrictive valve placed within the circuit provides PEEP. This delivers a pressure above atmospheric through the entire respiratory cycle. This positive pressure improves oxygenation and lung compliance and reduces the work of breathing (WOB) by¹⁸:

- Enabling the patient to take larger tidal volumes for the same amount of effort
- Increasing Functional Residual Capacity (FRC) i.e. preventing alveolar collapse and thereby increasing the surface area for oxygen exchange to occur
- Reducing ventilation/perfusion (V/Q) mismatch



Tidal volume is the volume of air moved into or out of the lungs in one normal resting breath. In the spontaneously breathing patient this equates to 5 – 8mls/kg.

Minute volume is the volume of air moved into or out of the lungs in one minute. $Respiratory\ rate \times tidal\ volume = minute\ volume$.

Physiological Benefits of NIV¹⁸

- Improved oxygenation
- Decreased work of breathing
- Improved V/Q Matching
- Decreased fatigue
- Increased minute ventilation

Assessment prior to commencement of NIV¹⁸

Prior to commencement of NIV patients are to be assessed for:

- Capacity to protect own airway;
- Adequate level of consciousness (may be selectively used for hypercapnic COPD patients that are "not for intubation" obtunded patients);
- Anticipated compliance with the mask;
- Capacity to manage their respiratory secretions
- Potential reversibility.

Indications for NIV¹⁸

- Severe (acute) exacerbation of COPD (pH<7.35 and relative hypercarbia)
- Acute respiratory failure and acute pulmonary edema in the absence of shock or acute coronary syndrome requiring acute coronary revascularization
- Immunosuppressed patients with acute respiratory failure
- Post extubation ventilator support
- Acute respiratory failure post lung resection surgery or post abdominal surgery
- Asthma
- Acute respiratory failure in selected 'not for intubation' patients
- Acute deterioration of disorders associated with sleep hypoventilation such as neuromuscular and chest wall restrictive disorders and obesity hypoventilation syndrome
- Temporary palliation for dyspnoea, to be replaced by combinations of opioids and benzodiazepines. A medical team decision will be made when NIV is deemed no longer beneficial to the patient's management

Contraindications for NIV¹⁸

- Life threatening hypoxemia (e.g. PaO₂ <60mmHg on FiO₂ 100%) where the patient requires invasive ventilation
 - Respiratory arrest
 - Untreated pneumothorax
 - Inability to protect own airway
 - Copious, unmanageable respiratory secretions
- Facial burns/trauma/recent facial, neck or upper airway surgery that prevents mask use

Adverse Effects¹⁸

Complication	Treatment	Rationale
Skin necrosis (nose and chin)	Apply soft dressing such as comfeel	Cushions and protects the area
Mask intolerance and leaks	Select appropriate mask size	Provides better seal and comfort and prevents gas from escaping
CO ₂ rebreathing	Ensure minimal dead space is present.	Reduces resistance in the circuit and allow CO ₂ to escape
Nasal congestion and sinusitis	Ensure adequate humidification	Prevents build up and increased viscosity of secretions

Eye irritation and conjunctivitis	Avoid mask leaks, apply eye drops as appropriate	Reduced air flow into the eyes.
-----------------------------------	--	---------------------------------

Masks / Interfaces for NIV¹⁸

Some of the available interfaces for NIV include:

- Full Face Mask – Oronasal mask
- Nasal Mask
- Total Face Mask



Full Face Mask
(Oronasal Mask)

Nasal Mask

Total Face Mask

Mask / Interface Application¹⁸

- Assessment of mask fit, interface type, head strap tightness, skin integrity of mask contact point, ventilation synchrony and degree of mask leak are to be completed each time the interface is adjusted and minimally second hourly
- Interventions to prevent pressure injury secondary to the interface are to be implemented on commencement of NIV. These include use of specialist devices to prevent tubing (e.g. NG tube) being pressed into the skin and protective interfaces such as a hydrocolloid dressing.
- When deterioration in skin integrity is identified, immediate strategies are to be employed to reduce further injury. These may include:
 - Change the interface (consider full face mask)
 - Reposition the interface ensuring that the mask is not pressing on the bridge of the nose or that the straps are not pressing into the skin



Position of patient

The patient should be in a sitting or semi-recumbent position in bed. Consider side-lying position to remove pressure from pendulous abdomen (obesity / pregnancy).

NIV Parameter Setting¹⁸

- **FiO₂ (% O₂)** – Set between 21% to 100% depending on patients clinical condition, oxygen requirements, PaO₂ and Sa O₂.
- **CPAP** – Set in the CPAP mode. It determines the amount of PEEP that will be delivered. This can be set between 4cmH₂O to 20cmH₂O pressure. Usual start up setting is 5cm H₂O.

- **IPAP** – Inspiratory Positive Airway Pressure. This is the positive pressure delivered on inspiration. IPAP cannot be set lower than the EPAP. This can be set between 4cmH₂O to 40cmH₂O pressure. IPAP cannot be set lower than the EPAP. Usual start up is about 10cm H₂O.
- **EPAP** – Expiratory Positive Airway Pressure. It determines the amount of PEEP that will be delivered. EPAP cannot be set higher than IPAP. This can be set between 4cmH₂O to 20cmH₂O. Usual start up setting is 5cm H₂O.
- **PS** - Pressure Support. This is the difference between IPAP and EPAP, e.g.: IPAP 10 – EPAP 5= PS 5. It helps in reducing the work of breathing.
- **RATE** – Can be set to deliver a mandatory rate in the BiPAP mode. (It is intended as a **back up rate**, and should be set so that it does not interfere with a spontaneously breathing patient). The rate control and the timed inspiration are linked so that the inspiratory time is never longer than the expiratory time.
- **Timed Inspiration** – this is the amount of time for inspiration (only in a rate controlled breath) and determines the I:E ratio (Inspiratory: Expiratory ratio). This may be limited by the rate setting when the rate is set higher than 10bpm.
- **IPAP Rise Time** – This setting controls how quickly the ventilator increases inspiratory pressure EPAP to IPAP. It can be set from 1 – 5 (The numbers correspond to 0.1, 0.2, 0.3, 0.4, 0.5 of a second). For example, those patients with very fast breathing may require a fast Rise time (1 which is equal to 0.1 sec) to ensure an adequate amount of gas flow to prevent gas starvation. Usually start with a 0.2 or 0.3 sec.
- **C-flex**: The optional C-Flex setting enhances traditional CPAP by reducing the pressure at the beginning of exhalation – a time when patients may be uncomfortable with CPAP – and returning it to the set CPAP level before the end of exhalation. The amount of pressure relief is determined by the C-Flex setting and the expiratory flow. The higher the setting number (1, 2 or 3) and the greater the expiratory flow, the greater the pressure relief (during the active part of exhalation only).

Monitored Parameters¹⁸

- **Rate**: Respiratory rate or total breathing frequency. Moving average over the last 6 breaths (or 15 seconds).
- **V_T**: Estimated exhaled tidal volume. Moving average over the last 6 breaths. It is body temperature pressure saturated (BTPS) compensated.
- **V_E**: Estimated minute ventilation. The product of tidal volume (spontaneous and timed) and rate (spontaneous and timed). Moving average over the last 6 breaths.
- **PIP**: Peak inspiratory pressure. The highest patient pressure during the previous breath cycle.
- **T_I / T_{TOT}**: Inspiratory duty cycle or inspiration time divided by total cycle time. Moving average over the last 8 breaths. Ti/Ttot represents the percentage of time spent in inspiration. I.e: we normally breathe at a ratio of 1:2, spending 1/3 of our time in inspiration, therefore a “normal” inspiratory time is considered 33%.
- **Pt. Trig**: Patient-triggered breaths, as a percentage of total breaths over the last 15 minutes. A spontaneously breathing patient should be triggering 100%
- **Pt. Leak**: Estimated patient leak or unintentional leak. Average during the previous breath cycle. Displayed only after a suitable exhalation port and mask/patient interface are selected. (this is done through Menu – Mask/Port). Acceptable leak is 7-25 L/min, however the device can compensate efficiently for a leak up to 60 L/min. A leak lower than 7 means the mask is too tight and a leak higher than 60 means that you need to fit your mask better.

CPAP Settings



BiPAP /ST Settings



Alarm Parameter Settings¹⁸

- **High Inspiratory Pressure Limit (HIP)**– High Pressure Limit. This is set at 30cmH₂O pressure (range is 5 to 50cmH₂O). Should be set above the IPAP level.
- **Low Inspiratory Pressure Limit (LIP)**– Low Pressure Limit. This is set at 5 - 15 cmH₂O (range is 'disabled' to 40 cmH₂O). Should be set below the IPAP level and above the EPAP level. It works in conjunction with the low pressure delay to indicate if there is a failure to trigger between the two pressure levels.
- **Low Inspiratory Pressure Delay Time (LIP T)**– Low Pressure Delay. Set at 15 – 20 secs (range is 0-60 seconds). Set this for the maximum acceptable time the pressure can drop below the LIP set value before the alarm is activated.
- **High Rate** – High Rate. Assess the patient's rate. Set at 35 BPM. This may vary depending on the patient's respiratory rate and clinical condition (range is 4 BPM to 120 BPM).
- **Low Rate** – Low Rate. Assess the patient's rate. **Due to no set Apnoea alarm – always set the Low Rate 1 above your set mandatory/ backup rate.** i.e. If the mandatory / backup breath rate set at 6, low rate alarm should be set at 7. This way the device will alarm prior to commencing timed breaths as set. This will vary depending on the patient's respiratory rate and clinical condition (range is 4 BPM to 120 BPM).
- **Low Minute Ventilation** – Low minute ventilation. Assess the patients exhaled Minute Ventilation and set the alarm at lower limit of not less than 3L/min and the upper limit at 5L above the patients exhaled Minute Ventilation. This provides an alert of low minute ventilation which could result in raised levels of PaCO₂.

Criteria for Failure of Non-invasive ventilation.^{2,35}

- Failure to reduce PaCO₂ within 1-2 hours of initiating non-invasive ventilation. Worsening PaCO₂ that is not resolving with Non-invasive ventilation.
- Presence of severe hypoxemia. PaO₂:FiO₂ ratio ≤ 146 after 1 hour of treatment with non-invasive ventilation.
- Worsening metabolic acidosis in patients with Acute Lung Injury.

Clinical issues^{2,18,35}**Nursing considerations when caring for the patient on NIV.**

As the nurse caring for the patient requiring NIV, it is imperative to assess and maintain patient comfort and compliance. These are key factors in determining successful application of Non-invasive ventilation. Some of the factors that contribute to patient comfort and compliance include:

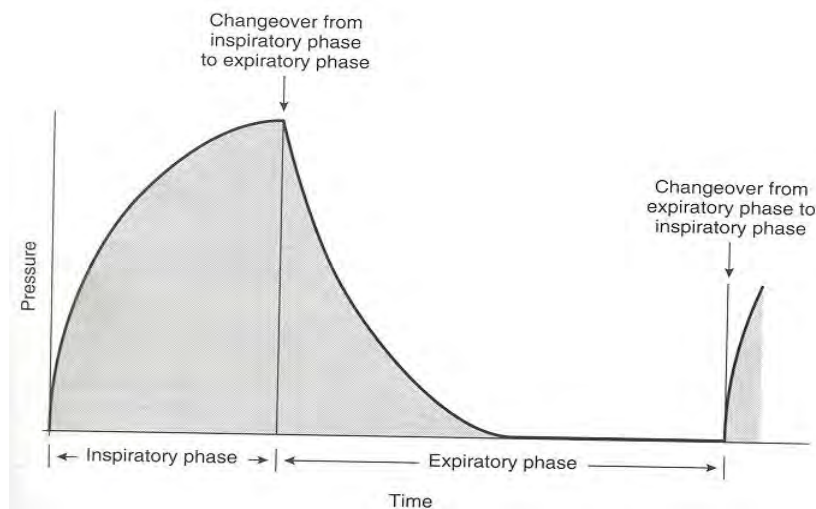
- **Choice of suitable interface and levels of pressure applied.**
- **Position of the patient:**
 - The patient should be in a sitting or semi-recumbent position in bed to achieve maximal chest wall movement and prevent upper airway obstruction
 - Assessment of patient comfort and pain is to be completed minimally second hourly and documented
- Encourage mobilisation of patient out of bed.
- Synchrony of Ventilation - Assess and monitor chest wall movement, and accessory muscle use.
- Assessment of patient comfort and pain.
- Pharmacotherapy for dyspnoea, anxiety and pain.
- **Humidification** – this will enhance normal airway humidification, thereby contributing to patient's tolerance to therapy and comfort
 - All NIV circuits are to be actively humidified if the patient is expected to require NIV for more than 24 hours.
 - Gas temperatures during NIV are to be based on patient comfort
- **Nutrition and Hydration**
 - Oral feeding is to be initiated if the patient is able to tolerate small periods off NIV.
 - No oral intake is to be implemented if the patient has a decreased LOC or in respiratory distress with an increased work of breathing
 - Nutrition assessment and plan is to be undertaken and documented for the patient receiving NIV after 24 hours of the initiation of therapy

- Palliation of symptoms – may sometimes be used for symptom relief and comfort care.
- Oral hygiene is to be attended every two hours as long as the patient's tolerance to ceasing NIV is longer than five minutes
- Eye care is to be attended every two hours.
- Ensure patients receive pressure area care – provide extra attention to pressure areas that can be created by the interface/mask e.g.: the bridge of the nose. Protective dressings over these areas may be indicated.
- A clear plan for the parameters indicating escalation to intubation and ventilation in the event of NIV failure is to be documented on clinical presentation or initiation of therapy.

Invasive Ventilation Principles

Functional characteristics of the Ventilator.^{32,33}

- It would be ideal if mechanical ventilators could mimic the mechanics and physiology of spontaneous breathing. However as all modern ventilators are positive pressure ventilators, they use a power source known as the drive mechanism, to force air into the lungs during inspiration. Expiration occurs passively during positive pressure ventilation.
- In spontaneous breathing, no conscious effort is required to perform the phases of a respiratory cycle.
- However, if a machine is to perform a respiratory cycle, it must be told what and how to carry out each of the 4 respiratory phases of ventilation:
- **Trigger inspiration** (initiation of inflation/triggering a breath).
- **Limit inspiration** (how the lungs are inflated).
- **Cycle** (ends inspiration).
- **Expiration** (deflates lungs & prepares for next inflation).



Four Phases of the Respiratory Cycle during a volume controlled breath
(Pierce, 1995)

Control³³ The 3 ways in which a ventilator can deliver a controlled breath are:

- **Volume controlled** (volume limited, volume targeted) and pressure variable
- **Pressure Controlled** (pressure limited, pressure targeted) and volume variable.
- **Dual controlled** (volume targeted (guaranteed) pressure limited)

Cycling³³ The 3 ways in which a ventilator cycles from inspiration to expiration:

- **Time cycled** – cycles based on set inspiratory time
- **Flow cycled** – based on lung recoil and flow sensor settings such as in pressure support
- **Volume cycled** – cycles once a set tidal volume has been delivered, such as in volume controlled ventilation.

Triggering³³ The 3 types of triggering that cause the ventilator to cycle to inspiration:

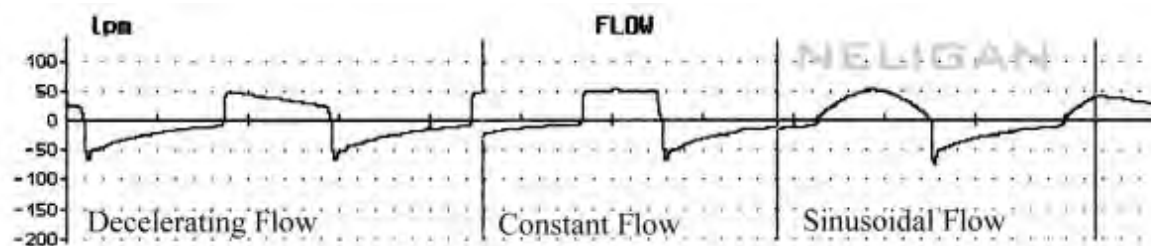
- **Time:** The ventilator cycles at a set frequency as determined by the controlled rate.
- **Pressure:** the ventilator senses the patient's inspiratory effort by way of decrease in baseline pressure.
- **Flow:** modern ventilators deliver a constant flow though the circuit throughout the respiratory cycle. A deflection in this flow by patient inspiration, is monitored by the ventilator and this triggers it to deliver a breath. Requires less effort than pressure triggering.

Breaths³³ The 3 types of breaths that can be delivered:

- **Mandatory** (controlled) breath – determined by set respiratory rate.
- **Assisted** (as in assist control, SIMV, PSV)
- **Spontaneous** (no additional assistance, as in CPAP)

Flow pattern: (<http://www.ccmtutorials.com/rs/mv/page2.htm>).

- **Sinusoidal** = this is the flow pattern seen in spontaneous breathing and CPAP. It matches the normal flow waveform of a spontaneously breathing patient.
- **Decelerating** = the flow pattern seen in pressure targeted ventilation. There is an initial high flow to the point of peak flow at the beginning of inspiration. After this point has been reached, flow will decrease until the flow reaches a percentage of the peak flow and inspiration will end. It results in a lower peak airway pressure than constant and accelerating flow, and better distribution characteristics
- **Constant** = flow continues at a constant rate until the set tidal volume is delivered. The square waveform reaches peak flow rapidly and maintains this rate of flow until the breath has been delivered.
- **Accelerating** = flow increases progressively as the breath is delivered.



(<http://www.ccmtutorials.com/rs/mv/page2.htm>)

The goals of therapy with mechanical ventilation are:

- Improve oxygenation and ventilation (optimise pulmonary gas exchange).
- Reversal of severe hypoxemia.
- Reversal of acute, severe respiratory acidosis.
- Relief of respiratory distress.
- Prevention or reversal of atelectasis.
- Reversal of respiratory muscle fatigue.
- Decrease in systemic or myocardial O₂ consumption.
- Decrease work of breathing.

Some of the indications for mechanical ventilation are:^{7,33}

- Failure to ventilate as characterised by increased arterial CO₂ tension.
- Failure to oxygenate characterised by decreased arterial O₂ tension.
- Respiratory muscle fatigue or its potential.
- Acute respiratory muscle failure from a disease process such as ARDS, acute lung injury, congestive heart failure and sepsis.
- Acute exacerbation of CAL that is refractory to conservative therapies.
- Inability to protect airway as a result of trauma, neurological dysfunction, anaesthesia or drug overdose.

Modes of Ventilation.

SIMV (Synchronised Intermittent Mandatory Ventilation). ^{7,17,33}

SIMV is available in both pressure control or volume control ventilation depending on the type and model of the ventilator available. This mode of ventilation ensures the patient will receive a preset rate and tidal volume or inspiratory pressure. In between these mandatory breaths the patient may initiate spontaneous breaths in synchrony with the ventilator.

- Preset Rate x Tidal volume or Inspiratory Pressure
- Mandatory, assisted and or spontaneous breaths
- Spontaneous tidal volume variable, which is dependent on patient effort and pressure support level set, lung characteristics, compliance and resistance
- Synchrony between Patient and Ventilator

SIMV utilises a window of time in which a breath is due and will look to deliver this breath within this timing window. The ventilator delivers the mandatory breath simultaneously as it senses the patient's inspiratory effort. ^{14,33}

There are 3 types of breaths delivered during SIMV. ^{14,17, 33,43}

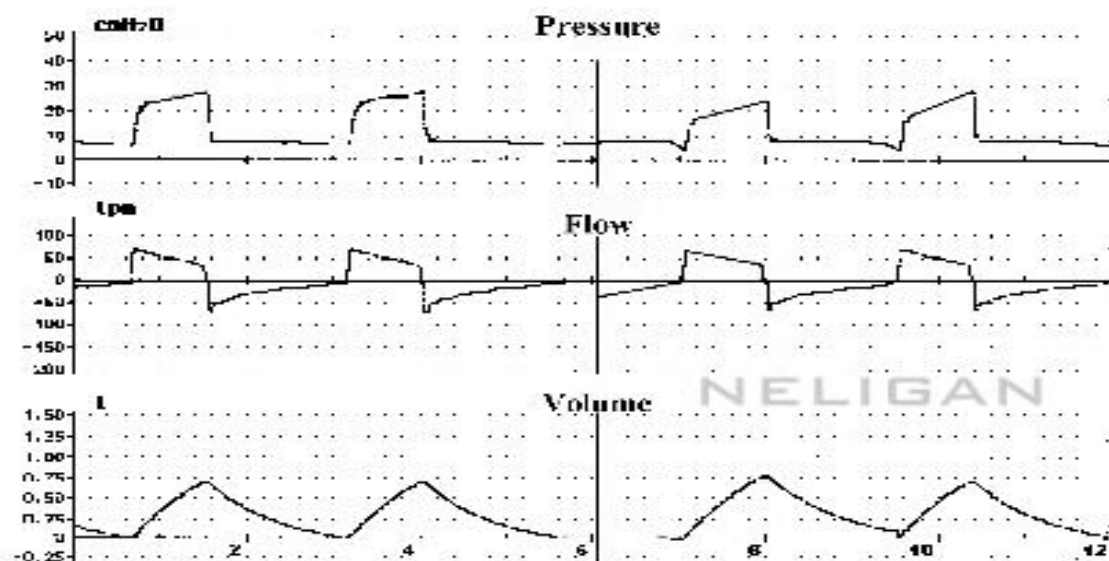
- **Mandatory Breath:** If the patient does not initiate sufficient inspiratory effort within the timing window, the mandatory SIMV breath is delivered at the scheduled time. The ventilator will then reset to respond to the next spontaneous inspiratory effort.
- **Assisted Breath:** When a mandatory SIMV breath is due (as determined by the RATE control), the assist or timing window opens and waits for the patient's inspiratory effort. Upon sensing the patient's inspiratory effort, the ventilator delivers the preset TIDAL VOLUME or INSPIRATORY PRESSURE in synchrony with the patient. As soon as the mandatory breath has been triggered, the assist window closes. As a consequence, once the assisted breath has been delivered, subsequent patient effort, results only in spontaneous (or pressure supported) breaths, until the next mandatory breath is due.
- **Spontaneous Breath:** In between these mandatory or assisted breaths, the patient may initiate spontaneous breaths in synchrony with the ventilator. The volume of the spontaneous breath is dependent on the respiratory muscular effort that the patient is able to generate, the level of pressure support set and resistance and compliance.

Volume Controlled Ventilation ^{14,17, 33,43}

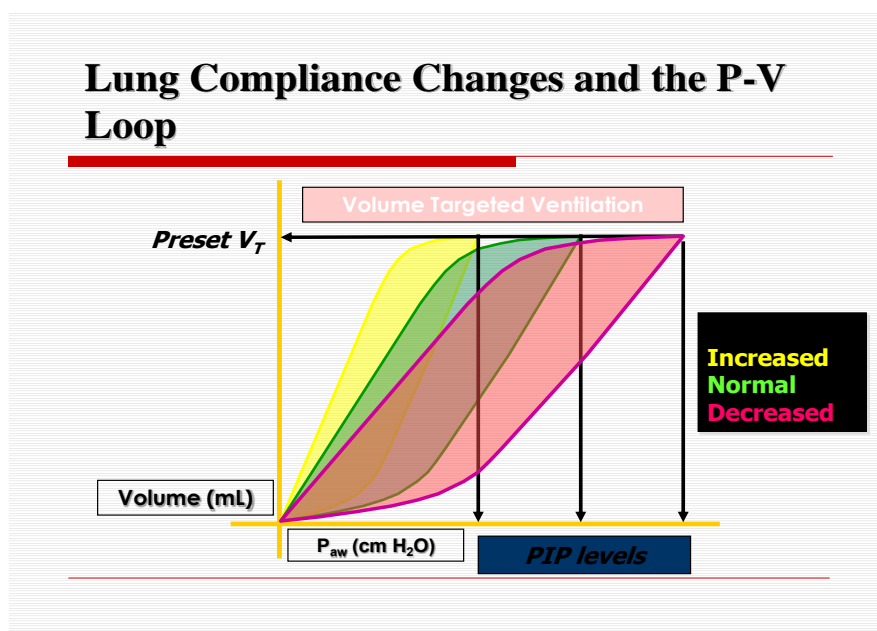
This is the default mode of ventilation (called SIMV on the Draeger ventilators) used for ventilation post anaesthetics, post intubation for respiratory failure and to protect the airway as a result of trauma or neurological dysfunction.

With volume controlled ventilation a preset tidal volume is delivered to the patient at a set rate. Once the preset inspiratory time (i.e. you set a plateau time or inspiratory pause time or I:E ratio) or tidal volume is reached inspiration ends and exhalation will begin. Unlike PCV, the flow rate or peak flow on the ventilator must be set. Peak inspiratory and plateau pressures will vary in response to resistance and compliance of the lungs (Henderson, 1999, 51 and Pilbeam, 1993)

- Rate x Tidal Volume x Inspiratory Time (Set Parameters)
- Peak Inspiratory Pressure and Plateau Pressure (Varies with every breath, depending on resistance and compliance)



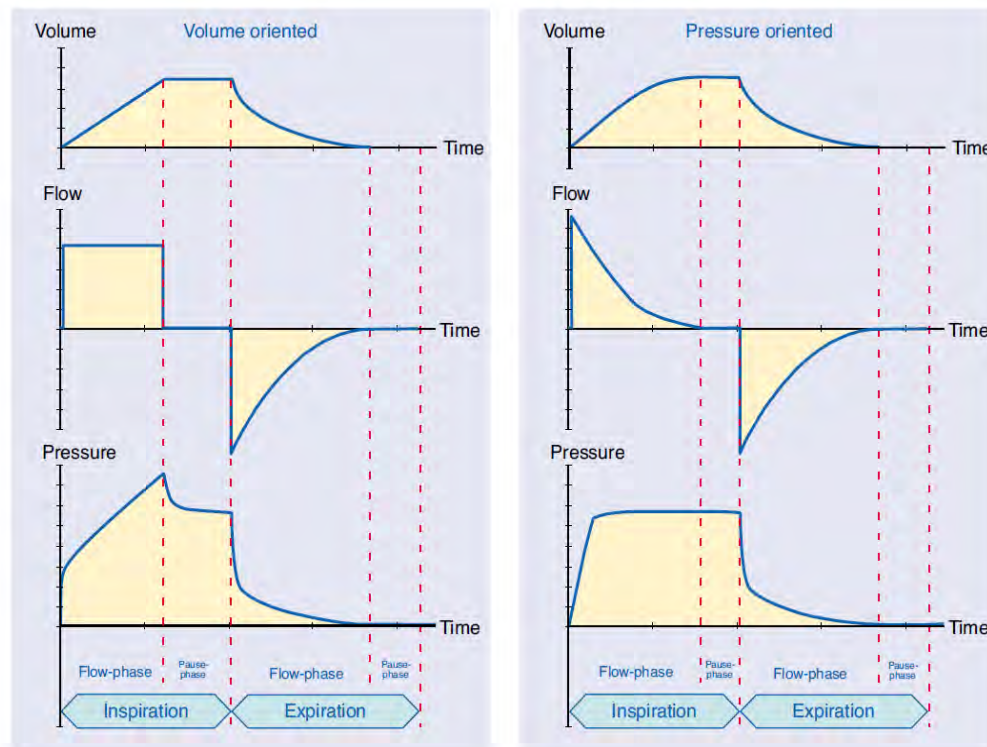
<http://www.ccmtutorials.com/rs/mv/page2.htm>



Volume Controlled, Pressure Limited Ventilation. ^{14,17, 33,43}

This is the safety mode for SIMV on the Draeger ventilators. The mode is called SIMV, but is volume controlled and pressure limited.

Safety features are available on most ventilators in use today, to prevent excessive airway pressure and hence the onset of barotrauma. A high airway pressure alarm limit is set on the ventilator. Once the peak inspiratory pressure reaches the preset high airway pressure alarm limit (usually set 10 cm H₂O above the patient's current peak inspiratory pressure), inspiration is terminated and expiration begins. An audio and or visual alarm gives a warning and the remaining tidal volume is not delivered but vented to the atmosphere. The alarm is "Volume not Constant".



(Rittner & Doring, 2005)

PSV – Pressure Support Ventilation. ^{14,17, 33,43}

Patients are weaned from the SIMV volume controlled mode to Pressure Support mode of ventilation as soon as they are able to initiate spontaneous breaths and maintain an adequate minute ventilation.

Patient has control over all aspects of breathing such as rate and tidal volume. The breath is assisted by a preset pressure.

- Used only for spontaneously breathing patients
- The patient controls tidal volume, respiratory rate and inspiratory flow rate
- Inspiratory time is determined by patient effort and partially by ventilator's strategy to signal exhalation
- Pressure support is adjusted to pts respiratory function and CO₂

This mode of ventilation is only functional when the patient is breathing spontaneously. Pressure support is active only during inspiration and is triggered by the sensitivity control on the ventilator. This is normally a flow trigger in most modern ventilators.

Respiratory effort is augmented by the delivery of a preset level of inspiratory positive pressure. The patient will initiate a breath and the ventilator will deliver a constant preset pressure support level throughout inspiration, promoting the flow of gas into the lungs. One important variant to monitor is the patient's VT, there is no set VT. Therefore the VT is dependent on, level of pressure support, patient effort, lung characteristics, compliance and resistance of the system (patient and ventilator).

PSV is used in most units in conjunction with SIMV. One advantage of using the two modes together is that if the patient is apnoeic they will still be guaranteed mandatory breaths. Realistically PSV should only be used in patients with an intact respiratory drive, as all breaths are patient initiated.

With PSV the pressure limit is not the mechanism that routinely causes cycling from inspiration to expiration. PSV is a flow cycled mode of ventilation, as inspiration ends on the basis of flow rather than pressure, time or volume. The patient retains control over respiratory pattern and volume.

Pressure support ventilation is terminated when, a certain percentage of the peak inspiratory flow rate usually 25% has been reached.

Advantages

- Pressure support decreases the work of breathing imposed by the ventilator circuit and in particular the ETT.
- Decreasing the work of breathing increases the patient's tolerance to weaning
- Improves patient ventilator synchrony and patient comfort, because the patient has control over the process of ventilation.
- The amount of assistance given to the patient and the quality and quantity of work applied to respiratory muscles for reconditioning are more titratable than with other weaning modes (Pierce, 1995)

PSV is generally used in conjunction with at least 5 cm H₂O of Positive End Expiratory Pressure (PEEP)

In PSV the peak inspiratory pressure (PIP) is equal to the PS level plus the amount of applied PEEP.

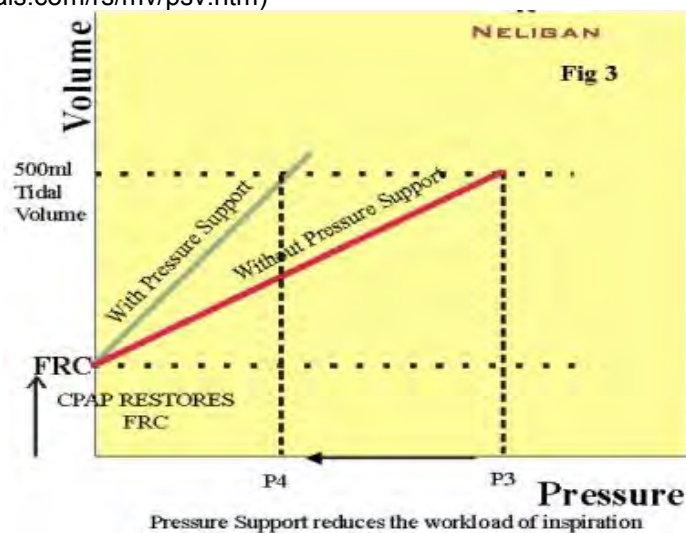
$$\text{PS Level} + \text{PEEP} = \text{PIP}$$

Example : Lucinda's pressure support level is set at 15 cm H₂O and PEEP 10 cm H₂O

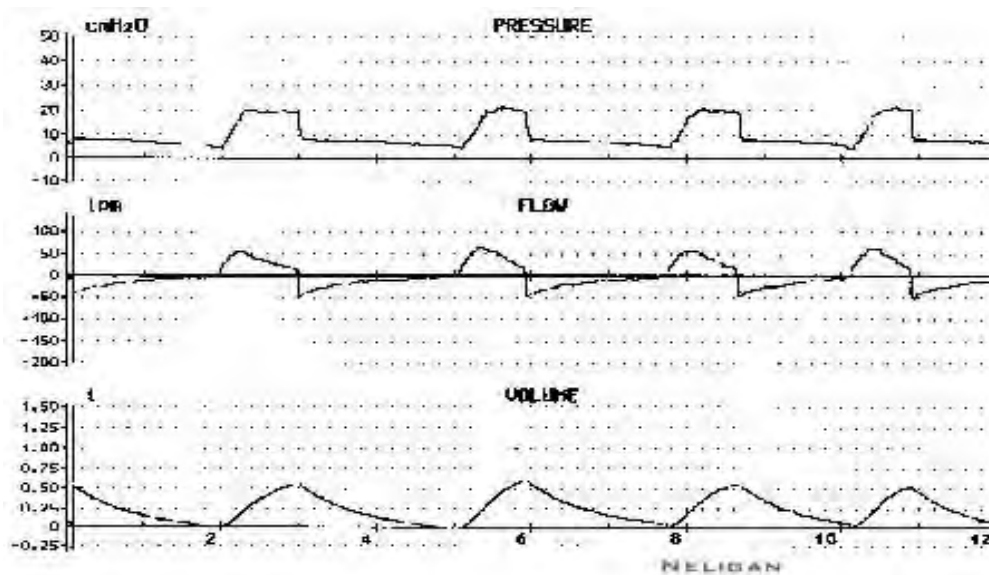
$$\text{PS Level } 15 \text{ cm H}_2\text{O} + \text{PEEP } 10 \text{ cm H}_2\text{O} = \text{PIP } 25 \text{ cm H}_2\text{O}$$

In the figure below you can see that application of CPAP has returned the resting FRC to normal, but the work of breathing remains high due to the loss of lung compliance (P3 is required to achieve the target tidal volume in this patient of 500ml). The solution to this problem is to administer pressure support in inspiration, in order to reduce the workload of breathing, and achieve the targeted tidal volume, with lower intrapleural pressures (P4).

(<http://www.ccmtutorials.com/rs/mv/psv.htm>)



The figure below is a screen display of a patient on pressure support ventilation. The pressure support is set at 12cmH₂O. Note the decelerating flow pattern and the termination of flow before the end of inspiration. The flat topped appearance of the pressure waveforms indicates a pressure controlled breath, and the slight variance in tidal volumes is typical of pressure support and, indeed, normal breathing (<http://www.ccmtutorials.com/rs/mv/psv.htm>)



(<http://www.ccmtutorials.com/rs/mv/page2.htm>)

Pressure Control Ventilation PCV^{14,17, 33,43}

This mode of ventilation is used in patients with poor lung compliance as demonstrated by raised airway pressures. In these patients it is difficult to deliver adequate tidal volumes whilst limiting airway pressure to maintain a plateau pressure of < 30. This could be due to Acute Lung Injury or ARDS.

With pressure controlled ventilation, a volume of gas is delivered to the lungs at a constant pressure during a set inspiratory time. Once the inspiratory time is reached, inspiration ends and exhalation will begin.

The Tidal volume delivered depends on:

- Set Inspiratory pressure
- Respiratory rate
- Inspiratory time / I:E ratio
- Airway Resistance
- Lung compliance

Rate x Inspiratory Pressure x Inspiratory Time (Set Parameters)

Tidal Volume (Varies with every breath, depending on resistance and compliance)

Aims:

- Provide a constant pressure during the entire inspiratory phase
- Avoid high peak airway pressures
- Provide a decelerating flow wave pattern

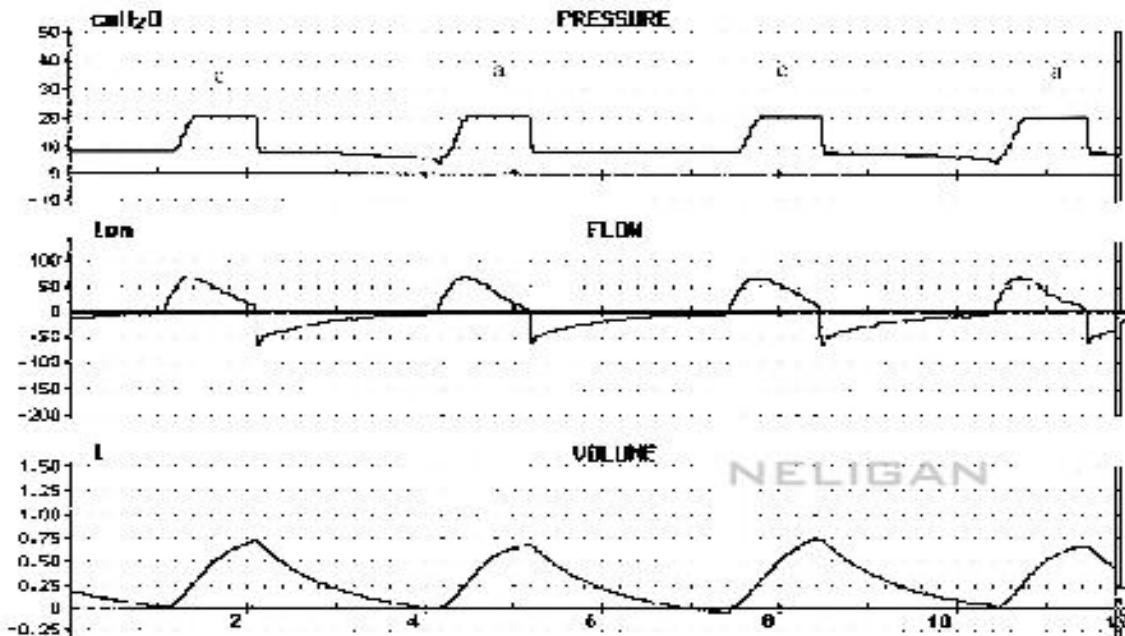
Clinical Advantages of PCV

- Improved gas exchange
- Laminar flow at the end of inspiration
- Reduces V/Q mismatch
- Peak inspiratory pressures are lower
- Flow is responsive to patient demand

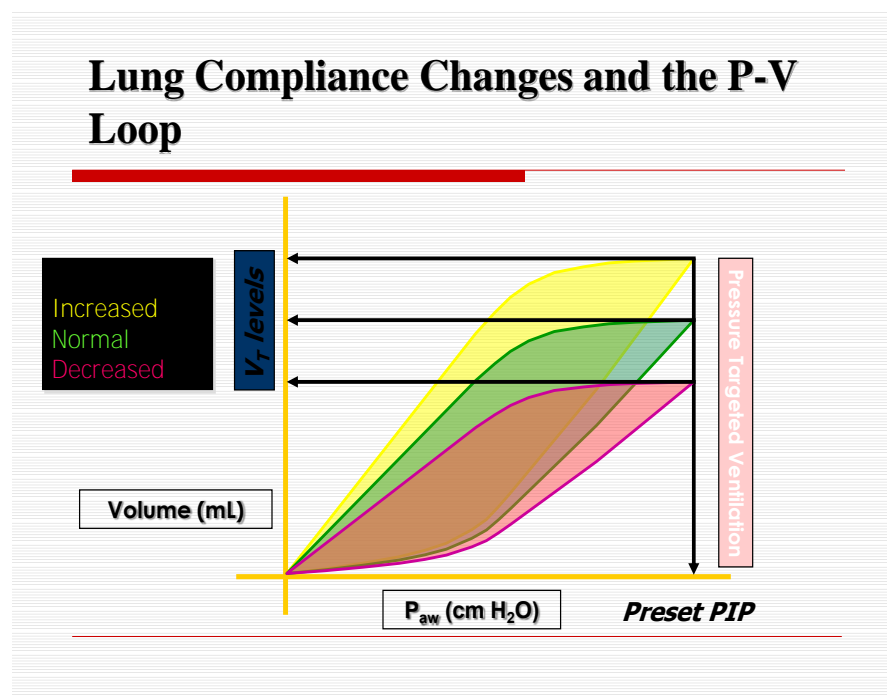
PCV is useful in non-compliant lungs which exhibit high airway pressures and poor oxygenation while on volume controlled ventilation. The clinician is able to gain control over airway pressures and prevent complications by predetermining the inspiratory pressure required by the patient, to achieve ideal volumes and improve oxygenation.

With PCV, pressure is sustained throughout the inspiratory phase, splinting the airways and allowing

for improved gas distribution. In volume controlled ventilation, flow is constant while the airway pressure increases during inspiration until a maximum PIP is reached, thereby preventing noncompliant alveoli from adequately filling.
 (Henderson, 1999;Pierce, 1995; Pilbeam, 1993; Tobin,1994 and Woodruff,1999)



<http://www.ccmtutorials.com/rs/mv/page2.htm>



CMV- Controlled Mandatory Ventilation – does not support spontaneous breathing. ^{14,17, 33,43}
 This mode of ventilation is rarely used because the patient has to be heavily sedated and paralysed to tolerate not being able to trigger a breath. It is sometimes used in patients requiring set sigh breaths as part of lung recruitment strategies. These are often patients with ARDS or ALI injury who are deeply sedated and paralysed.
 CMV is available in both pressure control or volume control ventilation depending on the type and

model of the ventilator available. The ventilator delivers a preset tidal volume or inspiratory pressure, at a preset rate. The patient is unable to trigger the ventilator to take a spontaneous breath.

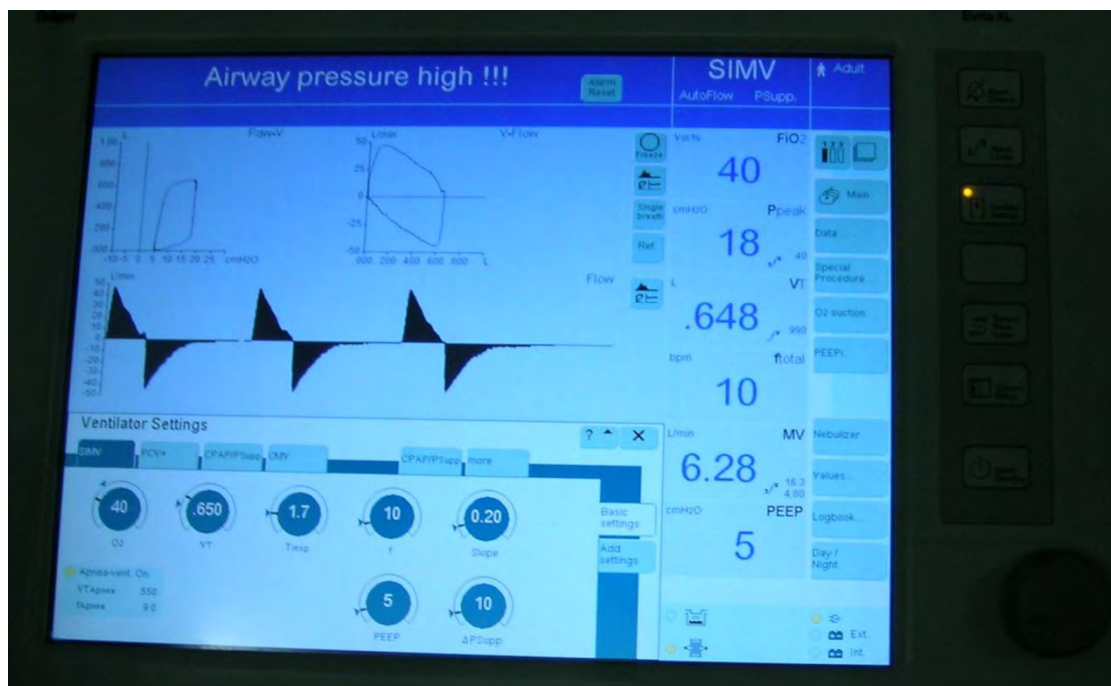
The ventilator performs all the work of breathing, respiratory muscle weakness and atrophy results with prolonged use of CMV, making the task of weaning the patient from the ventilator so much harder.

The patient is unable to cough and is more likely to develop pneumonia.

(Henderson, 1999, Pierce, 1995 and Sassoon, 1990)

- Preset Mandatory Rate x Tidal volume or Inspiratory Pressure
- No spontaneous breathing
- An inspiratory effort by the patient will not produce a machine delivered breath
- The patient cannot trigger the ventilator to take a spontaneous breath
- The patient should be sedated and paralysed

Ventilator Parameters^{14,17, 33,43}



FiO₂ – Fraction of inspired oxygen (the blend of oxygen and air to give the desired inspired oxygen tension from 21% to 100%). Setting will be dependent on patients oxygenation as determined by SpO₂, SvO₂, SaO₂ and PaO₂.

V_T - Tidal Volume (volume delivered for each breath). **Set at 8ml/kg (for patients with Acute lung injury or ARDS set at 6ml /kg)**

f – Frequency – (mandatory respiratory rate per minute). **Set initial rate at 12-14bpm.**

The total Frequency of breaths includes the mandatory, assisted and spontaneous breaths.

MV – This is not set, but measured. It is the Tidal volume x the respiratory rate. Utilised to set lower and upper minute volume alarm limits.

Adjusting either tidal volume or respiratory rate will affect PaCO₂. For example, decreasing the VT or ↓RR will ↓ the expired tidal volume and result in an increased PaCO₂.

PEEP – Positive End Expiratory Pressure (Pressure applied at the end of expiration to maintain alveolar recruitment). Initially this is set at **5 to 10 cmH₂O**.

PS – Pressure Support (The preset pressure limit to which the ventilator delivers a flow when triggered by the patient. The flow cycles off when a certain percentage of peak inspiratory flow (usually 25%) has been reached. It is used to assist spontaneous breathing of the ventilated patient). Initially set at **10 -15 cmH₂O**.

Insp. Press – Inspiratory pressure (Preset pressure limit in PCV for each breath. Tidal volume is variable and determined by this preset pressure limit as well as lung compliance). **Set initially at 20 to 25 cmH₂O (adjust to achieve V_T of 6 to 8ml/kg)**

Insp. time / T.insp – (This is the time allowed for inspiration – the aim is to adjust the inspiratory time and respiratory rate to achieve an **I:E ratio of 1:2**). **Set at 1.7sec for a set rate of 12bpm**.

I:E Ratio – the ratio of time for inspiration : expiration for each breath, **normally 1:2**. It is determined by the set Insp.time and the respiratory rate.

Inverting the I:E ratio is a strategy sometimes utilised in an attempt to improve gas distribution and increase mean airway pressure for improvement of oxygenation. Remember that you are altering the normal ventilatory pattern and keep in mind that shortening the expiratory time results in gas trapping in the lung, creating **auto PEEP**.

Lengthening the inspiratory time to give an I:E ratio of 1:1 is a strategy that can be utilised to improve VT delivery in Acute lung injury

^_ - Pressure rise time – (the time allowed by the ventilator for the preset pressure limit to be achieved). Initially set at **0.2sec**.

Peak Flow / Autoflow – The peak flow rate determines the speed of gas delivery from the ventilator through the circuit to the patient. It may be manually set or automatically titrated to the patients inspiratory demand (auto flow). The easiest rule of thumb to follow is that a patient requires a peak flow of roughly four times the minute ventilation. It should be set slightly higher when using a decelerating waveform pattern and in those with airflow obstruction. It is set at **30-40 l/min (roughly 4 times the MV)**.

Autoflow is the peak flow automatically set by the ventilator. When this option is available it is generally chosen instead of selecting a peak flow.

Sensitivity - The sensitivity can be a flow or pressure trigger. It should be set as close to zero as possible, without allowing the ventilator to cycle spontaneously (auto-cycling). Auto-cycling occurs when the ventilator interprets the following scenarios as patient effort and continues to cycle with a detected respiratory rate of 40 to 60 breaths per minute:

- **Flow Trigger** – this is the set flow signal that triggers the opening of the inspiratory valve on the ventilator. (Some ventilators may use a pressure trigger / sensitivity instead of a flow trigger). This is what triggers the delivery of the set pressure support. . As the patient inhales there is a drop in flow in the circuit and the ventilator recognises this and PS delivery commences. Normally set at **1L/min**.
- **Pressure trigger** - A pressure device monitors a decline in pressure. As the patient initiates a breath, the resulting sucking effort reduces the airway pressure in the circuit. During this time, there is no flow from the ventilator increasing the patient's work of breathing further.

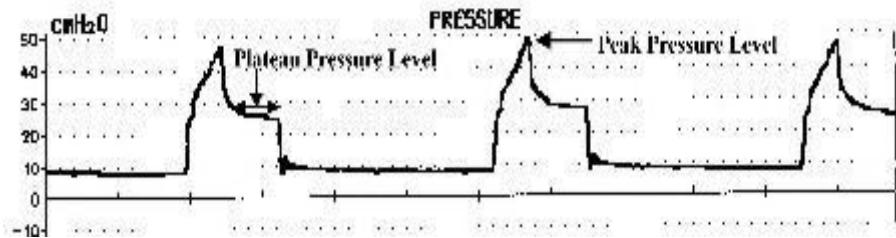
Apnoea Ventilation – ensure this is turned on and set at Tv– 400ml and rate – 10bpm. When turned on this allows for automatic switch over to volume controlled ventilation if the patients stops breathing for the preset apnoea alarm time (normally 15-20sec). Volume controlled ventilation in the apnoea mode is with the parameters (V_T and rate) set in apnoea ventilation. To terminate apnoea ventilation it is necessary to press the reset key.

Inspiratory Hold – this is an inspiratory pause delivered at end inspiration. It prolongs the inspiratory time and maintains air in the lungs at the end of inspiration. It can be used as part of recruitment manoeuvres or to measure the plateau pressure for mandatory breaths.

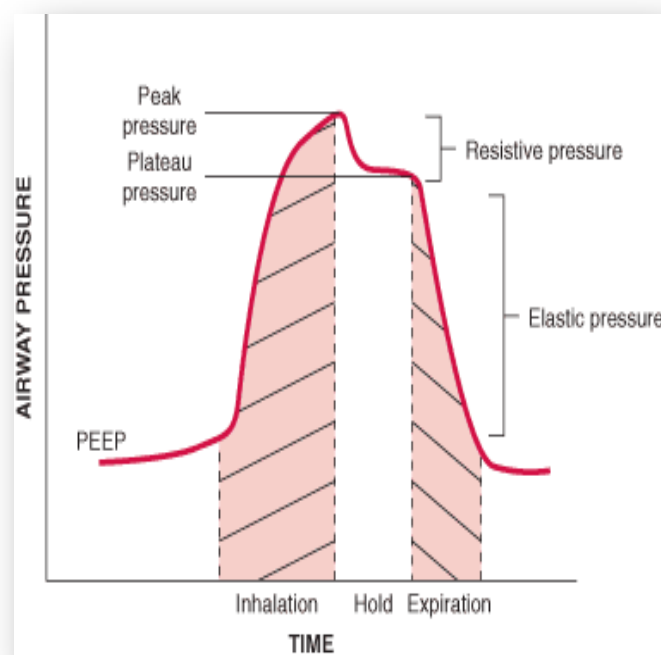
Airway Pressures:

- ◆ **Peak Airway Pressure** – This is the pressure measured by the ventilator in major airways and it strongly reflects airway resistance. It is the sum of resistive pressure and plateau pressure.

- ◆ **Plateau Pressure** – This is the pressure applied to the small airways and alveoli (in positive pressure ventilation). This pressure reflects lung and chest wall compliance. It is measured after a 0.5sec inspiratory hold manoeuvre. Normal plateau pressure range is 15 -25cmH₂O. **With lung protective ventilation strategy aim is to maintain plateau pressure < 30cmH₂O.**



Source: <http://www.ccmtutorials.com/rs/mv/page2.htm>



Alarm settings^{14,17, 33,43}

MV – Minute ventilation.

High MV Alarm - Usually set 5L above mandatory and or spontaneous minute ventilation rate. This alarm results from an increased rate and or V_T.

Causes of this alarm:

- Patient wakes up and commences spontaneous breathing
- Patient is apprehensive
- Patient is in pain/uncomfortable
- Rate or V_T adjusted or high minute volume alarm setting not reset
- Wall oxygen, when used for nebuliser, can increase minute volume and trigger the alarm
- Patient not coping with present ventilatory mode (tachypnoeic)
- Lung compliance improves in PCV and greater V_T is delivered for set inspiratory pressure

Management

- If patient wakes and is ready to commence weaning, decrease ventilator rate.
- If spontaneous V_T are large decrease pressure support
- Treat pain, reposition and reassure your patient
- Adjust alarm setting in accordance with current minute volume value if the value is in an acceptable range

- Increase ventilatory assistance if patient not coping with present ventilatory mode
- Low MV Alarm** - Usually set 2 to 3 l/min below mandatory and or spontaneous minute ventilation rate, minimum setting 3 to 4 l/min. Alarm violation occurs when the monitored minute volume value falls below set alarm value. This alarm is often called the **apnoea or disconnection alarm**

Causes of this alarm:

- Disconnection somewhere in the ventilator circuit.
- ETT cuff leak, dislodgement of ETT.
- Hypoventilation or Apnoea caused by bolus of sedation or excessive infusion rate, fluctuating level of consciousness due to neurological insult, cardio-pulmonary event, patient tiring during the weaning process decreasing rate and or VT.
- Rate or VT adjusted but low minute volume alarm not reset.
- Lung compliance decreases in PCV and less VT is delivered for set inspiratory pressure

Management

- Reconnect patient to circuit if disconnected. Check circuit for leak, particularly at connection sites. Replace circuit if necessary.
- Check cuff pressure, reinflate cuff if leaking. Seal cuff with a 3-way tap or change ETT if unable to prevent leak.
- Auscultate chest and confirm correct tube placement.
- Review patient's analgesia/sedation requirements, achieve control over pain and discomfort using the least possible dose and the least drugs. Remember, drugs are not metabolised or excreted as efficiently for patients suffering liver or renal failure.
- Increase ventilatory assistance if patient not coping with present ventilatory mode.
- Adjust alarm setting in accordance with current minute volume value if the value is in an acceptable range

High V_T – tidal volume - set high limit at 800 ml

Alarm violation occurs when the monitored tidal volume value is higher than set alarm value. This alarm is an extra safety feature on some ventilators to protect against barotrauma.

Causes of this alarm:

- Incorrectly set mandatory VT.
- Incorrectly set mandatory inspiratory pressure.
- Pressure support level too high.
- VT adjusted but alarm setting not reset.

Management

- Adjust mandatory VT \approx 8mls/kg.
- Adjust mandatory inspiratory pressure to achieve desired VT.
- Adjust pressure support level to achieve desired VT.
- Adjust alarm setting to appropriate value

f (Frequency) – Respiratory rate – set high limit at 25-30 bpm. Alarm violation occurs if patient becomes tachypnoeic with a high respiratory rate.

Causes of this alarm:

- Patient wakes up and commences spontaneous breathing
- Patient is coughing and requires suctioning.
- Patient is apprehensive, in pain/uncomfortable
- Patient not coping with present ventilatory mode (tachypnoeic)
- Patient has increased work of breathing and requires alteration to mode or level of support

Management

- If patient wakes and is ready to commence weaning, decrease ventilator rate.
- Suction patient if required
- Treat pain, reposition and reassure your patient
- Increase ventilatory assistance if patient not coping with present ventilatory mode

Airway Pressure Alarms

This alarm continuously compares the monitored value for peak inspiratory pressure with its high and low settings. When either of these settings is violated, an alarm sounds and the alarm violation will be indicated.

Low Airway Pressure

- Usually set at **10 cm H₂O**
- Alarm violation occurs when the monitored peak inspiratory pressure value falls below set alarm value
- This alarm is often called the **disconnection alarm**

Causes of this alarm:

- Disconnection somewhere in the ventilator circuit or circuit leak.
- ETT cuff leak.
- Low pressure limit set above the peak inspiratory pressure monitored.

Management

- Reconnect patient to circuit if disconnected. Check circuit for leak, particularly at connection sites. Replace circuit if necessary.
- Check cuff pressure, reinflate cuff if leaking. Seal cuff with a 3-way tap or change ETT if unable to prevent leak.
- Adjust low airway pressure alarm setting in accordance with current peak inspiratory pressure value if the value is in an acceptable range.

High Airway Pressure

- Usually set 10 cm H₂O above peak inspiratory pressure monitored.
- Alarm violation occurs when the monitored peak inspiratory pressure value increases above set alarm value
- Keep the peak inspiratory pressures 30-35 cm H₂O if possible
- Though peak pressure alarms are set, if peak pressure is raised always check the plateau pressure under values measured and this should be kept < 30cm H₂O

Apnoea alarm

- Apnoea alarm will be activated if a mandatory or spontaneous breath is not registered within set time period (apnoea time period).
- If apnoea ventilation is an option on your ventilator and is set correctly, the ventilator will then switch over to volume controlled ventilation. The patient will receive a guaranteed tidal volume and rate that has been set under the apnoea settings.
- Apnoea ventilation settings, V_T 400 x rate (6). The respiratory rate is set below a normal rate to encourage the patient to continue to breathe spontaneously. If a rate of 10 or 12 breaths per minute is set, the patient will have no desire to take spontaneous breaths and will remain in a mandatory volume controlled mode.
- Apnoea time period is usually set 15 to 20 seconds.

Causes of this alarm: (Hypoventilation or Apnoea)

- Bolus of sedation or excessive infusion rate.
- Fluctuating level of consciousness due to neurological insult or cardio-pulmonary event.
- Neuromuscular event.
- Patient exhausted by the weaning process, late stage as patient initially tachypneic.
- Inappropriate ventilator settings, i.e. sensitivity set too high for patient to trigger, insufficient pressure support level

Management

- Review patient's analgesia/sedation requirements, and check the RASS sedation score. Achieve control over pain and discomfort using the minimal dose and infusion rate to achieve the desired sedation score and ensure patient is comfortable. Remember, drugs are not metabolised or excreted as efficiently for patients suffering liver or renal failure.
- Increase ventilatory assistance if patient is not coping with present ventilatory mode. Treat the

cause.

- Adjust sensitivity level (flow trigger). The sensitivity should be set as close to zero as possible, without allowing the ventilator to cycle spontaneously (auto-cycling).
- If Patient not coping on PSV, discuss with medical team may need to return to mandatory ventilation mode.

Ventilation Emergency Drill

If the ventilator is continuously alarming and simple troubleshooting measures of the alarms have not worked, CALL FOR HELP and activate the VENTILATION EMERGENCY DRILL.

The problem can only exist in one of three places:

1. The Machine
2. The Tube
3. The Patient

- CALL FOR HELP
- DISCONNECT THE PATIENT FROM THE VENTILATOR, and gently hand ventilate with 100 % O₂ by Laerdal bag.
- If the patient is easy to ventilate, then the problem is usually with the machine
- Perform the ventilator check procedure
- Re-evaluate the ventilator mode and settings, especially if the patient is breathing spontaneously
- Pass a suction catheter down the ETT or Tracheostomy tube
- Perform an assessment of airway, breathing, circulation, auscultate the chest, check vital signs. Perform an ABG, obtain a chest X-ray

Refer to the table below for individual causes and treatment.

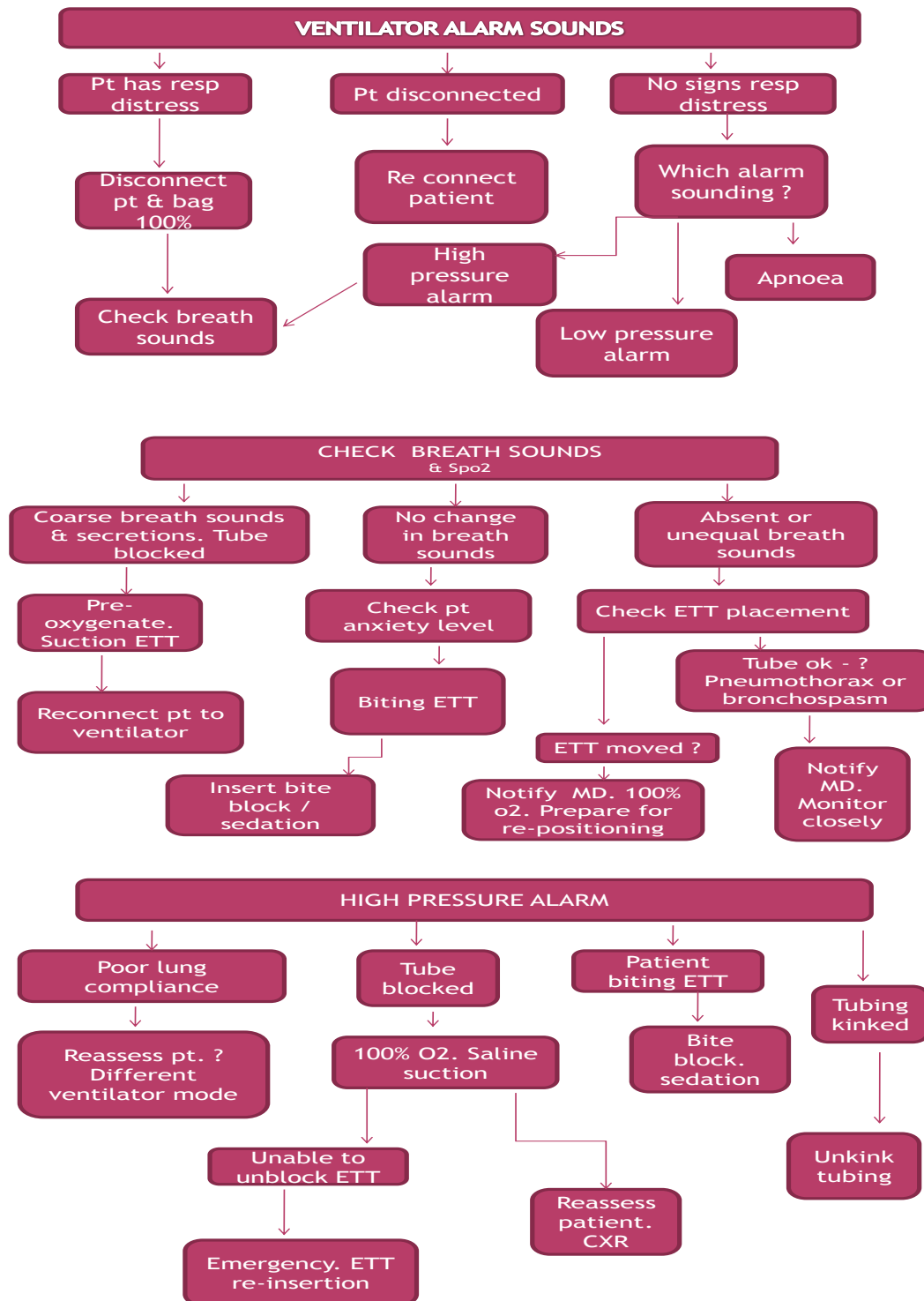
Causes and management of situations that constitute a Ventilation Emergency.^{7,43,44}

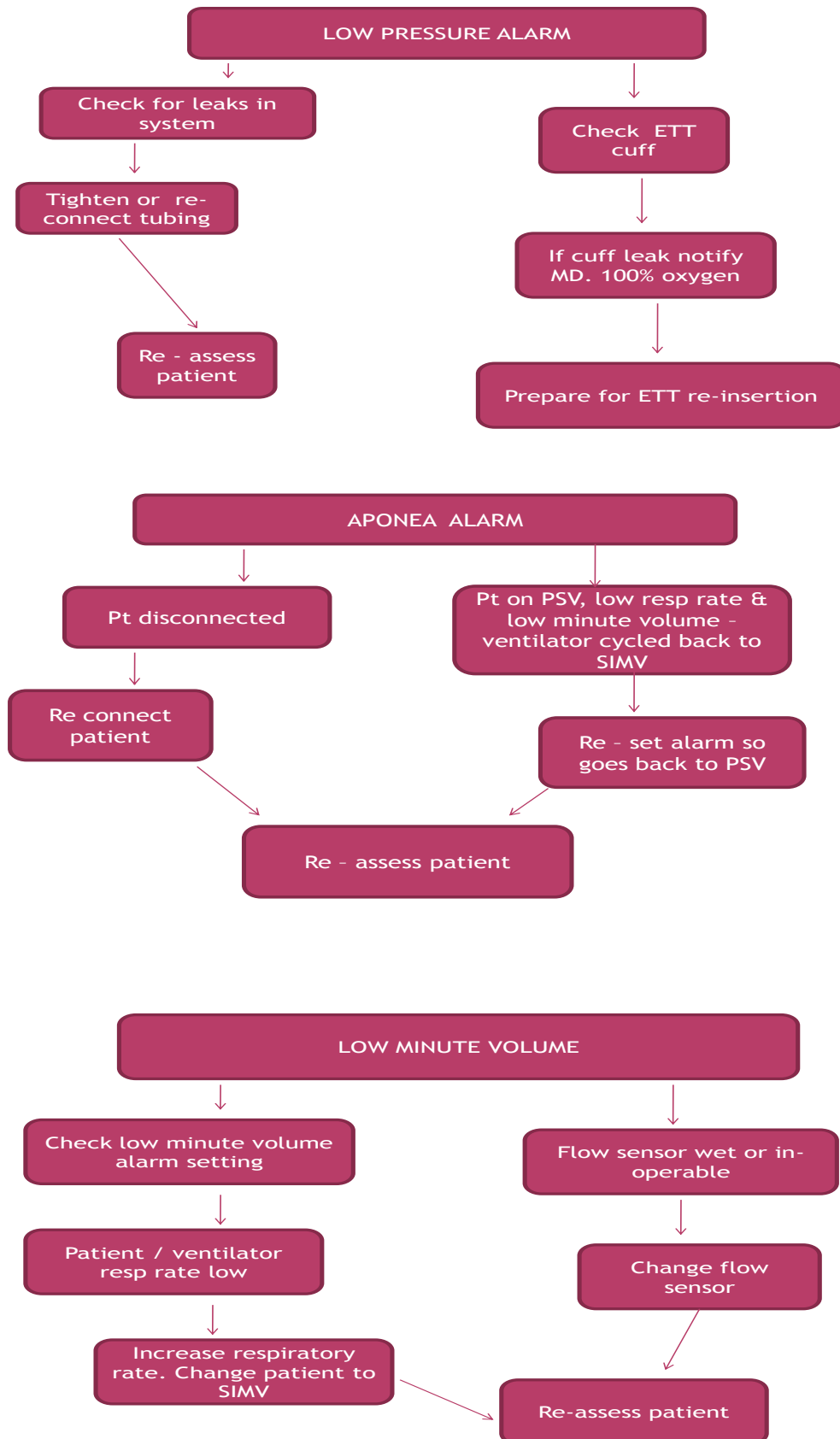
CAUSES	MANAGEMENT
<p>The ETT is obstructed</p> <ul style="list-style-type: none"> • Patient biting the tube • Blood or sputum in the ETT • Water in circuit tubing • Kinked ETT or kinked ventilator tubing <p>Always check for presence of EtCO₂ Waveform – if there is slopping up (ascending) waveform or no waveform and unable to ventilate patient ETT Tube is obstructed or misplaced.</p>	<ul style="list-style-type: none"> • Assess sedation score, explain why ETT insitu, if patient non compliant - Insert a bite guard , assess need to increase sedation. • Attempt to pass a suction catheter. If sputum cannot be cleared, CALL FOR HELP, if ICU medical officer not present call a MET. • Empty water into humidification chamber. Ensure humidifier on and temperature set appropriately. • Reposition the patient's head and neck. Unkink tubing • IF THE ETT IS COMPLETELY OBSTRUCTED AND CANNOT BE CLEARED, REMOVE IT IMMEDIATELY AND GIVE 100 O₂ BY BAG- MASK VENTILATION.
<p>Coughing</p>	<ul style="list-style-type: none"> • Suction the ETT in case of sputum • If continues, check position of ETT • ↑ Sedation if patient won't settle evidence of ventilator – patient dysynchrony & no identifiable problem present.

<p>Patient – Ventilator Dysynchrony (Patient is “ fighting the ventilator”)</p>	<ul style="list-style-type: none"> • Review ventilator settings with resource person • Assess patient – level of sedation, breathing pattern, mode, settings, auscultate the chest. Observe the flow waveforms and pressure volume loops. Adjust ventilation settings or change mode to facilitate patient-ventilator synchrony. • Having ruled out all other problems, it may be necessary to give a bolus dose of sedation, +/- neuromuscular blocking agents.
<p>Some of the possible complications of intubation and mechanical ventilation are:</p> <ul style="list-style-type: none"> • Pneumothorax, • Haemothorax, • Tension pneumothorax or pleural effusion • Pulmonary oedema • Bronchospasm • ETT down the right main Bronchus • Sputum plug, collapsed lung 	<ul style="list-style-type: none"> • Pleural decompression by ICU medical officer with insertion of ICC. • ↑ PEEP/CPAP, ↑ FiO₂, Drug therapy (may include diuretic, vasodilator, inotropes) • Bronchodilators • Treat by pulling the tube back & confirm with a CXR • Suction, chest physiotherapy, ensure effective humidification and possibly bronchoscopy
<p>Pressure limit set inappropriately</p>	<ul style="list-style-type: none"> • Adjust high airway pressure alarm setting in accordance with current peak inspiratory pressure value if the value is in an acceptable range.
<p>Inappropriate ventilator settings</p>	<p>Review ventilator settings with resource person</p> <ul style="list-style-type: none"> • Assess if flow rate set too high, adjust flow rate to achieve lowest possible peak inspiratory pressures whilst still delivering target VT and patient synchrony. Turn Auto flow on if option available. • Check I:E ratio and respiratory rate, is adequate time allowed for inspiration and expiration? • VT or Inspiratory pressure set too high for patient's individual compliance and resistance • Check flow trigger setting.

Troubleshooting Alarms

TROUBLESHOOTING VENTILATOR ALARMS





Humidification.

Humidity is the amount of vaporised water contained within a gas. There are two measures of humidity – absolute and relative humidity.

Absolute humidity – This refers to the total mass of water contained in a given volume of gas at a given temperature. Stated another way, absolute humidity refers to the actual amount of moisture present in a volume of gas, at a given temperature.

Relative humidity – This is a ratio of actual (absolute) humidity to the amount of water vapour that gas could potentially contain at a given temperature. This means that although the actual amount of moisture in a volume of gas, at 37 °C, may be 30 g/L (absolute humidity), the gas could potentially hold 43.4 g/L of water vapour. The relative humidity of the gas, in this instance, would be approximately 69% ($30/43.4 \times 100$). If the actual or absolute humidity equals the potential humidity that a volume of gas can hold at a given temperature then the relative humidity equals 100%. From the previous example, 100% relative humidity at 37 °C would be achieved if the absolute humidity were 43.4 g/L.¹⁸

Need for humidification:^{18,28,51}

In normal respiration, inspired gases entering the upper trachea are warmed and humidified, by the naso-oropharynx, to a temperature of 32–36 °C with a relative humidity of about 90%. As the gas travels through the respiratory tract, humidification and warming continues through to the alveoli until the gas is fully saturated (ie absolute humidity 43.4 g/m³) at 37 °C. Inspired gases delivered to the patient via an endotracheal / tracheostomy tube completely bypass these normal mechanisms of humidification and warming supplied by the naso-oropharynx. To compound this problem, the supplies of medical air and oxygen delivered to the patient via the endotracheal / tracheostomy are cool and dry, with an absolute humidity of 0.00. Cold and dry gases presented to the lower respiratory tract can cause the following complications:

- Mucosal ulceration, inflammation and increased mucus viscosity.
- Depressed ciliary function.
- Microatelectasis.
- Airway obstruction caused by tenacious sputum.

To overcome this problem, inspired gases delivered through the endotracheal tube should be heated to body temperature (ie 37 °C) with a minimum relative humidity of 75%.

Two types of humidification are available:

- HMEF's (Heat and moisture exchange filters) – Dry Circuit
- Water bath humidification – Wet Circuit

HMEF's (Heat & Moisture Exchange Filters)^{18,28}

HMEFs combine a filter (designed to trap virus, bacteria and other organisms before they enter the unprotected airways) and a hydrophobic paper that heats and moisturises gas. Commercial heat-moisture exchange unit combined with a filter that provides filtration at 99.99% or greater efficiency according to ISO standards and Medical Devices Directive and a minimum standard of absolute humidification of > 23 – 30mgH₂O/Litre, attached at the patient end.

The heat and moisture exchanger retains condensed exhaled water, which vaporises on the next inspiration, providing the patient with humidification and heat conservation. This hydrophobic filter reaches peak efficiency in 3–4 breaths. With this device, as the gas is exhaled it cools and the moisture condenses on the filter providing a moist surface area for the following inspiratory flow.

Active (Water Bath) Humidification^{18,28,51}

This involves using a heater base to heat the water bath. The inspiratory and expiratory limbs of the ventilation circuit are also heated. The temperature of the base is 39°C and this ensures the delivery of 37°C heated gas at the patient end.

- Water-bath humidifiers must not be left to run dry due to the risk of airway burn.
- Active humidification circuits should be changed at least weekly or if soiled.
- Only sterile water-for-irrigation can be used in water-bath humidifiers.
- Humidification circuit must be lower than the level of the ETT /TT at all times to prevent aspiration of condensation from the tube (rain out).

Weaning From Mechanical Ventilation^{11,16,42}

- Weaning is the process of gradual reduction of mechanical ventilation support. The goal is to enable spontaneous breathing by the patient with the aim to achieve extubation / decannulation
- Successful weaning depends on several factors. These may include the weaning method applied, the patients clinical status and respiratory function.
- Objectives of weaning are.
 - To commence early weaning as soon as the patient fulfils weaning criteria.
 - To minimise the duration of ventilation.
 - To reduce length of stay in ICU
 - To reduce the incidence of ventilation associated complications.
 - To promote spontaneous breathing trials.
 - To maintain continuity in the weaning process.
 - To improve patient outcomes.

Criteria for weaning^{11,42}

- **Respiratory:**
 - Spontaneous mode of ventilation such as PSV or BiPAP
 - $FiO_2 \leq 40\%$ (assess the $PaO_2 : FiO_2$ ratio to be at least 200)
 - $PEEP \leq 8$
 - Pressure Support ≤ 10
 - $SaO_2 \geq 92\%$
 - Arterial blood gas - $pH \geq 7.30$, $PaO_2 \geq 80$, $PaCO_2 \leq 45$ (PaO_2 and $PaCO_2$ parameters for weaning will differ with individual patients)
 - Negative inspiratory force (maximum inspiratory pressure) – Refer to Appendix 2 for explanation on the manoeuvre.
 - Airway-occlusion pressure at 0.1s of occlusion (this is a measure of neuromuscular respiratory drive) – Refer to Appendix 2 for explanation on the manoeuvre.
 - Rapid shallow breathing index (RSBI [f/VT]). The likelihood of successful liberation from mechanical ventilation appears enhanced if the RSBI is less than 105 per minute per liter, providing another objective means of analysis. (*Note: While this is a sensitive test, its specificity is much less*).
 - The patient is capable of maintaining an airway with good cough reflex and minimal secretions to suction.
- **Cardiovascular:** hemodynamically stable, inotrope /vasopressor free or low dose inotropes / vasopressors, afebrile, optimise afterload reduction
- **Neurological:** Sedation off or minimal sedation (sedation score of +1 to -1), pain well controlled, intact respiratory drive. It is also desirable but not essential to have the patient appropriate and compliant. Neurosurgical patients weaning parameters may differ and will be individualised.
- **Psychological:** It is desirable to have the patient well rested with no delirium / psychosis.
- **Adequate Nutrition:** Carefully assess and exclude overfeeding with excess CO_2 load.
- **Fluid status.** Carefully assess and exclude signs of fluid excess. Patients with cardiac failure and ARDS should have a conservative fluid strategy and consider the use of diuretics to achieve a negative fluid balance prior to extubation². A negative fluid balance is significantly associated with successful weaning.
- Able to tolerate head of bed elevation $> 30^\circ$.
- Evidence of reversal of underlying cause of respiratory failure.
- No new potentially serious clinical conditions.

Contraindications to Weaning^{11,42}

- Respiratory rate ≥ 35 breaths /min
- $SpO_2 < 90\%$
- Heart Rate > 130 beat/min or change in rhythm.
- Systolic BP > 180 mmHg or < 90 mmHg
- ABG abnormal for the patient.
- Excessive anxiety, agitation or delirium.

Procedure / Process for Weaning^{11,42}

Assess the patients who are able to breathe spontaneously. Assess the patients pulmonary function status and ensure that patient meets the criteria for weaning. Position patient in an upright position with head of bed elevated to 45° - 60°. Monitor patient for any signs of fatigue or respiratory distress. There should be a document weaning plan for the patient.

Method of weaning.^{11,16,42}

Weaning can be achieved in two ways:

- By reducing the level of pressure support provided by the mechanical ventilator during Pressure Support Ventilation (PSV) or Bi-level Positive Airway Pressure (BiPAP).
- By using a T-piece spontaneous breathing trial (SBT). This is used mainly for weaning tracheostomy patients.

The two goals of a weaning trial are:

- The early detection of patients who are able to breathe without a ventilator, in order to avoid complications of prolonged mechanical ventilation
- The identification of patients who are not able to breathe spontaneously to avoid extubation failure and its potential complications.

Pressure support ventilation (PSV)

- In pressure support ventilation the patient has control over all aspects of breathing such as rate and tidal volume. The breath is assisted by a preset pressure.
- The patient controls all parts of the breath except the pressure limit. A set flow trigger (usually 1-2 L/min) allows the patient to trigger the ventilator to deliver a flow up to a preset pressure limit (e.g. Pressure Support of 10cm H₂O). The patient continues the breath for as long as they wish and the flow cycles off when a certain percentage of the peak inspiratory flow (usually 25%) has been reached. The tidal volume varies for each breath just as it does in normal spontaneous breathing.
- Weaning from pressure support occurs by gradual reduction of the Pressure Support to the minimal level needed to overcome dead space and resistance in the ventilator tubing (eg: 5 to 10cm H₂O). The patient is closely observed for any signs of fatigue or respiratory distress. Minute ventilation and ABG's are also monitored. If there are no signs of distress the weaning process is continued and the patient will be extubated. If there are signs of distress the patient is returned to previous ventilation settings, the reasons for failure are reviewed and documented and the weaning process recommenced at a later time.
- Weaning may occur rapidly or as a slower process depending on the patient's condition and duration of time spent on mechanical ventilation.
- Depending on the patients medical history and duration of time spent on the ventilator it may be necessary to implement the weaning process over a period of days, whereby there are short durations of reduction in pressure support for 2-4 of hours each day. The aim is to increase the length of time spent with low levels of pressure support everyday.
- If the patient tolerates the weaning of pressure support, consider extubation.

Bi-level Positive Airway Pressure (BiPAP)

- BiPAP provides 2 levels of positive pressure support.
- A high pressure for inspiration, inspiratory positive airway pressure (IPAP) to facilitate a larger tidal volume.
- A lower pressure for expiration, expiratory positive airway pressure (EPAP), to prevent alveolar collapse and maintain functional residual capacity.
- The same principles of weaning pressure support in PSV apply to BiPAP. The level of pressure support is reduced by decreasing the amount of IPAP, as this will reduce the difference between IPAP and EPAP.

Continuous Positive Airway Pressure (CPAP)

- CPAP and Positive End Expiratory Pressure (PEEP) are two terms that are used interchangeably. The concept of PEEP is that a pressure is applied at the end of expiration to maintain alveolar recruitment and functional residual capacity. Airway pressure is kept continuously positive during the entire respiratory cycle.

- CPAP can be used as a primary ventilatory mode to maintain alveolar recruitment and in the management of acute pulmonary edema or as a weaning mode.
- It increases functional residual capacity (FRC) and PaO₂ while decreasing intra pulmonary shunt, work of breathing and oxygen consumption⁶.

Spontaneous breathing trial / T- Piece weaning⁴²

- This involves a spontaneous breathing trial using a T-piece Circuit. It is mainly used in Liverpool ICU for weaning of tracheostomy patients.
- The patient is taken off the ventilator and the tracheostomy is connected to a humidified O₂ T-piece circuit (see picture below).
- The patient is then allowed to initiate spontaneous breaths for 1 to 2 minute. If the patient tolerates the trial and shows no signs of fatigue or distress, they may be left on the T-Piece for an initial duration of 15 – 30 minutes.
- The duration of the T-piece trial may be extended to 30-120 minutes as per the algorithm on the next page.
- If the spontaneous breathing trial is successful the patient may be extubated. If the patient shows any signs of fatigue or distress, return the patient to the previous ventilation settings, review and document the reasons for failure and recommence the weaning process at a later time.



- Multiple daily SBTs are reasonable provided there is no clinical evidence of respiratory muscle fatigue. A spontaneous T piece trial (30 min duration) is a rapid way to assess potential to wean.
- Failure of repeated SBTs should prompt consideration of:
 - Whether there is a benefit to daily SBT trials for that patient (if ventilator muscles are allowed to fatigue they may require >24 hrs to recover)
 - Identifying other reversible causes (e.g. a missed phrenic nerve injury)
 - Introduction of a weaning protocol (e.g. gradually increasing duration of spontaneous breathing trials for short periods progressing to long periods)
 - A more gradual reduction of pressure support (which avoids fatigue and lessens anxiety)

Signs of Fatigue^{11,42}

- Increased respiratory rate ≥ 35 breaths/min.
- Decreased Tidal volume ≤ 5 ml/kg
- Decreased PaO₂, SaO₂, SpO₂,
- Increased PaCO₂
- Patient looks and feels distressed - tachycardic, hypertensive, diaphoretic, anxious.

Factors that may prevent weaning^{11,42}

- Cardiac dysfunction
- Respiratory muscle weakness. Diaphragmatic weakness rather than fatigue is a significant reason for failure to wean.

- Abdominal distension that displaces the diaphragm upward and limits ventilation.
- Impaired oxygen delivery to the tissues from low cardiac output states. Signs of cardiac failure.
- Signs of systemic sepsis or metabolic disturbances.
- Lack of motivation to wean and anxiety on the part of the patient.
- Inappropriate positioning of the patient that will prevent adequate inflation of the lungs.
- Poor upper airway function, absence of good cough and swallowing, increased secretions.
- Poor pain control.
- Delirium
- Prolonged sedation and ICU acquired weakness – polyneuromyopathy.
- Large cumulative positive fluid balance and generalised oedema.

Extubation

Once it is determined that a patient no longer requires mechanical ventilation to maintain sufficient ventilation and oxygenation, the patient's ability to protect the airway and airway patency should be evaluated in preparation for possible extubation.

Precautions for Extubation^{3,42}

- History of difficult intubation, previous failed extubation
- Any degree of respiratory distress, increased work of breathing on the ventilator
- Low SpO₂ in the absence of pre-existing lung disease or abnormal arterial blood gas (ABG) values
- Agitation/delirium
- No evidence of a cuff leak with the cuff deflated (consider use of a glucocorticoid)³
- Poor cough³
- Large volume of secretions

Criteria for Extubation^{3,42}

This involves assessment of cough, level of consciousness, amount of secretions, and looking for a cuff leak.

- Verbal or written request made by the ICU Senior Registrar/Consultant
- The patient is awake and cooperative (able to open eyes, follow with eyes, grasp hand, and stick out tongue)^{5,6} and the original reason for intubation has resolved. N.B. *Extubation may at times be for withdrawal of therapy and end of life care.*
- Haemodynamically stable, pain well controlled³
- The patient on mechanical ventilation is weaned (exception may be the patient who will proceed to non-invasive ventilation):
 - to minimal support of not more than 5-8 cm H₂O PEEP, and
 - pressure support <10 cm H₂O, and
 - FiO₂ ≤ 40% (assess the PaO₂ : FiO₂ ratio to be at least 200)
 - achieving tidal volumes > 5 mL/Kg
- Assessed as likely to be able to protect their own airway from secretions (effective cough on suctioning).
- Assess for presence of cuff leak. It is reasonable to proceed with extubation if a cuff leak exists, but to delay extubation if a cuff leak is reduced and there are risk factors for laryngeal oedema (e.g. prolonged intubation). Extubation should be considered on an individual basis in patients who have an absent cuff leak, but no risk factors for laryngeal oedema and otherwise appear ready for extubation.
- Minimal secretions

Note: Patients who are at known risk of aspiration (e.g. those with ineffective cough, previous neurological injury affecting swallow/cough), may be extubated but should remain nil by mouth (NBM) until reviewed by the Speech Pathologist; and may require Dietician review.

Clinical Considerations for Extubation^{3,42}

Document the weaning process in the patient's health care record and flow chart. The patient should be assessed for weaning from the time they are commenced on mechanical ventilation and routinely at each morning shift review. The weaning process should occur by using a collaborative approach between the ICU medical, nursing and allied health team. Minimal sedation and adequate analgesia should be considered for patient's deemed ready for weaning. Patients may benefit from night sedation with drugs that promote sleep and rest during the night. Patients with cardiac failure and

ADRS should have a conservative fluid strategy and consider the use of diuretics to achieve a negative fluid balance prior to extubation. Afterload reduction is important for patients with cardiac disease to aid in weaning from ventilation. Assess and optimise nutrition status as malnutrition is associated with loss of muscle mass and difficulty to wean. Document complications, interventions, management plan and outcomes.

Complications of Positive Pressure Ventilation^{14,32,43,53}

Many changes occur when a patient is converted from spontaneous to mechanical ventilation. The major goal when placing someone on mechanical ventilatory support should be to minimise the work of breathing while effectively promoting gas exchange. The nurse has a key role in achieving this goal. He or she must be aware of the negative effects of ventilation at all times, in order to provide the best care possible for the patient under his/her care.

Respiratory

- Intrathoracic Pressure Changes During spontaneous breathing, air enters the lungs because intrapulmonary (intraalveolar) pressure, drops from 760 to 758mm Hg .ie pulmonary pressures are negative During mechanical ventilation air is forced into the lung by applying positive pressure, which increases intrathoracic pressure. High inflation pressures can result in barotrauma. High tidal volumes can result in volutrauma. Both of these situations can cause acute lung injury, and worsen respiratory failure.
- Atelectasis related to patient position and unvaried tidal volumes. When the patient is supine, the abdominal contents push the diaphragm up causing a decrease in the functional residual capacity and a resultant increase in airway resistance.
- Alveolar and bronchial collapse occurs when the lung volume decreases significantly, further complicating mechanical ventilation. The response may vary with patients but in general the pulmonary and respiratory system compliance will decrease
- V/Q mismatch related to air trapping and patient position
- Oxygen toxicity
- Increased work of breathing related to the ventilator circuit and patient interaction with the ventilator

Cardiovascular

- Increased intrathoracic pressure, decreases venous return to the heart (preload). This may cause a decrease in cardiac output, as evidenced by increased pulse rate and decreased blood pressure

Renal

- A decrease in blood pressure can alter renal perfusion
- Redistribution of blood in the kidneys from cortical to juxtamedullary nephrons

Gastrointestinal

- Stress ulceration

Neurological

- Increased intrathoracic pressure, impedes venous drainage which may increase intracranial pressure

Infection

- Related to immunocompromised critically ill patient
- Related to endotracheal tube or tracheostomy tube

Nursing Care Of the Mechanically Ventilated Patient.^{12,49}

- Ensure the safety / emergency equipment needed is available in the bed area of the mechanically ventilated patient.
 - Resus bag with PEEP valve.
 - Resus Masks – Size 3 (small adult), 4 (regular adult) & 5 (large adult)
 - Guedel airway – size 2 (green 8cm), 3 (yellow 9cm) and 4 (red 10cm).

- Nasopharyngeal airway – size 6 & 7.
- Suction catheters – size 12 & 14 and Yankauer sucker.
- O₂ cylinder > ½ full.
- Ensure that the ventilator is plugged into isolated power supply.
- Ensure that ventilator brakes are on and tubing is supported in tubing holder to reduce drag on patients endotracheal or tracheostomy tube.
- Ensure the airway (endotracheal tube or tracheostomy tube) is secured and that the ventilator circuit is correctly connected.
- Ensure the ventilator is switched on (and is off standby mode) prior to connecting patient.
- Identify the need to ensure correct placement of the airway by ETCO₂ assessment and by checking the position on the chest x-ray.
- Auscultate the patient's chest for the presence of breath sounds.
- Never leave a ventilated patient unattended.
- Select the most appropriate mode of ventilation.
- Ensure that the ventilator and monitor alarms are turned on and set appropriately.
- Ensure there is working humidification in the ventilation circuit (HMEF or wet circuit).
- Perform arterial blood gases as clinically indicated or when there is a change in patient's ventilatory status. Interpret the arterial blood gases results and adjust ventilation accordingly.
- Ensure the use of capnography monitoring in all mechanically ventilated patients.
- Obtain chest x-rays as clinically indicated and every day for all mechanically ventilated patients.
- Perform ongoing clinical assessment of the patients respiratory function.
- Identifies clinical situations or ventilator faults that may cause the ventilator to alarm and troubleshoot accordingly.
- Consider the use of sedation and analgesia to improve patient comfort and tolerance of and compliance with mechanical ventilation.
- Monitor for complications of mechanical ventilation, such as:
 - Complications associated with the endotracheal or tracheostomy tube such as tube malposition, laryngeal injury, tracheal stenosis, tracheomalacia and oral trauma.
 - Volutrauma leading to overdistension of the alveoli and biophysical damage to the lungs.
 - Barotrauma from high distending pressures leading to pneumothorax, subcutaneous emphysema or pneumomediastinum.
 - Shear injury from cyclical alveolar inflation and deflation.
 - Airway obstruction and mucus plugging may result from inadequate humidification.
 - Ventilation associated pneumonia
 - Potential for oxygen toxicity when on prolonged period of high FiO₂.
 - Indirect complications such as immobility, potential for thromboembolism, pressure areas, malnutrition, pain, stress, disturbed sleep patterns and communication difficulties.
- Be aware of the requirements for and procedure involved in transferring the mechanically ventilated patient to another area / department:
 - Assess patients clinical status to ensure they are safe to transport and that benefits outweigh risks of transport.
 - Select an appropriate ventilator for transport. Set ventilator mode and parameters and assess patients breathing on the transport ventilator prior to transfer.
 - Connect patient to the transport monitor.
 - Ensure all lines and tubes are secure.
 - Ensure all infusions have adequate volume for the duration of transport. Minimising /rationalising fluids and infusions required during transport.
 - Ensure that there is a full oxygen cylinder with Twin o vac and suction.
 - Check transport box for emergency drugs and airway equipment including the laerdal resus bag.
 - For tracheostomy patients take box with emergency tracheostomy equipment.
 - All ventilated patients must be accompanied by an ICU medical officer, ICU registered nurse and wardsperson (patients for OT will be transferred by OT staff).
- Assess patients readiness to wean and suitable weaning strategies.

M_FASTHUG – Acronym for a daily checklist that can be utilised in ICU to prevent ventilation associated pneumonia⁴⁹

Check these:	Use this guide:
M Mouthcare?	<ul style="list-style-type: none"> • Attended every 6 hours, or • If unable – document why not
F Feeding?	<ul style="list-style-type: none"> • Commence by day 1 in ICU, follow guide, or • If unable – document why not
A Analgesia? Pain assessed?	<ul style="list-style-type: none"> • Review medication chart and flowchart (remember: IV analgesia can be changed to oral or NG liquid - paracetamol etc.) • To assess level of pain, use: <ul style="list-style-type: none"> → Visual pain scale (awake patients) or → Behavioural Scale (ventilated & sedated/ coma)
S Sedation? Sedation prescribed? Document desired sedation score and weaning goals.	<ul style="list-style-type: none"> • Review ventilation, level of consciousness, lung function and disease process/reason for tube. • Specify weaning goals eg: keep MAP > 70mmHg, RR < 40 bpm. • Specify what sedation score should be attained/maintained. • A '<u>sedation vacation</u>' must be prescribed or the sedation is reduced
T s.c. heparin? prescribed? TEDs insitu	<ul style="list-style-type: none"> • Prescribe heparin or alternative • Contraindications? Specify these. • Review legs, any Contraindications for TEDs?
H Head up ≥ 30°?	<ul style="list-style-type: none"> • Specify desired patient position • Document the position attained on flowchart • Measure from the angle of the patient's <u>hip</u> up to tip of shoulder
U Ulcer prophylaxis?	<ul style="list-style-type: none"> • Review drug therapy, any contraindications? • If feeding goal rate attained, state why ulcer prophylaxis is being ceased.
G Glucose? GUT: Bowels last opened?	<ul style="list-style-type: none"> • Maintain glucose control • Date for bowels last opened is specified • When Bowels opened - charted, type of bowel movement...diarrhoea +++, etc.
S Skin care?	<ul style="list-style-type: none"> • Assess pressure ulcer risk using Waterlow Scale: document score and then document how often PAC is required • Consider need for pressure relieving mattress

14. LEGEND FOR SEDATION SCORE

Instructions

- Obtain a sedation score goal at handover/ward round; document this in the health care record (either in the clinical notes or on the ICU flowchart CR145).
- Assess a sedation score and a Glasgow Coma Score (GCS) at least every 4 hours and as clinically indicated. Note that if a stable patient is prescribed a sedative to assist with sleeping (e.g. temazepam), it is reasonable to omit one episode of assessment.
- Conduct a sedation score even if there is no apparent drug in use that would contribute to sedation; if sedation is present and not a goal of therapy – report this to the M.O. and document findings, action plan and outcome in the health care record.
- A 'sedation – vacation' from sedative drugs must be prescribed when the sedation score is deemed 'moderate sedation: '- 3', and this degree of sedation is not the goal of therapy.

Assessment

The use of a sedative aims to:

- enable the patient to cooperate with ventilation and treatments, and
- produce a desired amnesia to the Intensive Care environment.
- document which drugs the patient is taking to produce a sedative effect

Richmond Agitation-Sedation Score (RASS)^{6,16}

Score	Term	Description	Stimulus
+ 4	Combative	Overtly combative, violent, immediate danger to self, staff, others	-
+ 3	Very agitated	Pulls or removes tube(s) or catheter(s); aggressive	-
+ 2	Agitated	Frequent non-purposeful movement, fights ventilator	-
+ 1	Restless	Anxious but movements are not aggressive/vigorous	-
0	Alert and calm		-
- 1	Drowsy	Not fully alert, has sustained awakening (eye-opening/eye contact) to voice (≥ 10 seconds)	Verbal
- 2	Light sedation	Briefly awakens with eye contact to voice (< 10 seconds)	Verbal
- 3	Moderate sedation	Movement or eye opening to voice (but no eye contact)	Verbal
- 4	Deep sedation	No response to voice but movement or eye opening to physical stimulation	Physical
- 5	Unrousable	No response to voice or physical stimulation	Physical

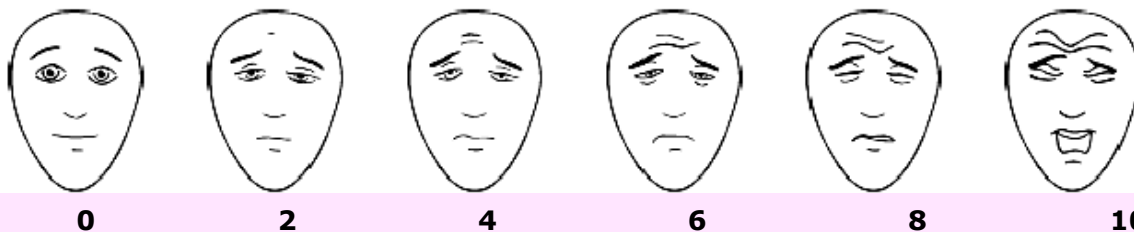
Procedure

- Observe patient
 - Patient is alert, restless or agitated (score 0 to + 4)
- If not alert, state patient's name and say to open eyes and look at speaker
 - Patient awakens with sustained eye opening and eye contact (score - 1)
 - Patient awakens with eye opening and eye contact, but not sustained (score - 2)
 - Patient has any movement in response to voice but no eye contact (score - 3)
- When no response to verbal stimulation, physically stimulate the patient by shaking shoulder and / or using the trapezius pinch or applying supra-orbital pressure, as appropriate
 - Patient has any movement to physical stimulation (score - 4)
 - Patient has no response to any stimulation (score - 5)

15. LEGEND FOR PAIN SCORE

Awake and responsive:^{6, 16}

Use "Faces Pain Scale - Revised"⁵ adapted for ICU - get the patient to point to the face that matches their pain level or ask the patient: 0 = none, 5 = worst pain.



- 0 No pain
- 2 Mild pain, discomfort only with moving
- 4 Continuous mild pain
- 6 Continuous moderate pain
- 8 Continuous severe pain
- 10 Excruciating pain

Assess for pain at least every 4 hours:

- If pain score < 4, consider analgesia effective, reassess frequently as ongoing analgesia may need to continue.
- If pain score \geq 4, increase analgesia to relieve pain
- Maintain prescribed sedation score, report any issues to the M.O. and document.
- Document score on the flowchart.
- If the patient has no pain and they are able to cough easily, deep breathe and move easily, the ongoing need for analgesia is assessed.

Patients who are sedated, mechanically ventilated and unresponsive

Use the "Behavioural Pain Scale"⁷ or the Critical-Care Pain Observation Tool (CPOT)^{6,16}

Behavioural Pain Scale

Behavioural Pain Scale		
ITEM	DESCRIPTION	SCORE
Facial Expression	Relaxed	1
	Partially tightened (eg, brow lowering)	2
	Fully tightened (eg, eyelid closing)	3
	Grimacing	4
Upper Limb Movements	No movement	1
	Partially bent	2
	Fully bent with finger flexion	3
	Permanently retracted	4
Compliance with mechanical ventilation	Tolerating movement	1
	Coughing but tolerating ventilation for most of the time	2
	Fighting ventilator	3
	Unable to control ventilation	4
TOTAL SCORE		3 TO 12
Score ranges from 3 (no pain) to 12 (maximum pain).		

The Critical-Care Pain Observation Tool (CPOT)^{6,16}.**Directives of use of the CPOT**

1. The patient must be observed at rest for one minute to obtain a baseline value of the CPOT.
2. Then, the patient should be observed during nociceptive procedures (e.g. turning, wound care) to detect any changes in the patient's behaviours to pain.
3. The patient should be evaluated before and at the peak effect of an analgesic agent to assess whether the treatment was effective or not in relieving pain.
4. For the rating of the CPOT, the patient should be attributed the highest score observed during the observation period.
5. The patient should be attributed a score for each behaviour included in the CPOT and muscle tension should be evaluated last, especially when the patient is at rest because the stimulation of touch alone (when performing passive flexion and extension of the arm) may lead to behavioural reactions.

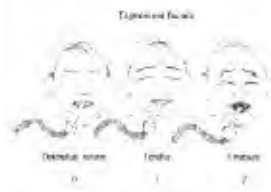
Observation of patient at rest (baseline).

The nurse looks at the patient's face and body to note any visible reactions for an observation period of one minute. She gives a score for all items except for muscle tension. At the end of the one-minute period, the nurse holds the patient's arm in both hands – one at the elbow, and uses the other one to hold the patient's hand. Then, she performs a passive flexion and extension of the upper limb, and feels any resistance the patient may exhibit. If the movements are performed easily, the patient is found to be relaxed with no resistance (score 0). If the movements can still be performed but with more strength, then it is concluded that the patient is showing resistance to movements (score 1). Finally, if the nurse cannot complete the movements, strong resistance is felt (score 2). This can be observed in patients who are spastic.

Observation of patient during turning.

Even during the turning procedure, the nurse can still assess the patient's pain. While she is turning the patient on one side, she looks at the patient's face to note any reactions such as frowning or grimacing. These reactions may be brief or can last longer. The nurse also looks out for body movements. For instance, she looks for protective movements like the patient trying to reach or touching the pain site (e.g. surgical incision, injury site). In the mechanically ventilated patient, she pays attention to alarms and if they stop spontaneously or require that she intervenes (e.g. reassurance, administering medication). According to muscle tension, the nurse can feel if the patient is resisting to the movement or not. A score 2 is given when the patient is resisting against the movement and attempts to get on his/her back.

The Critical-Care Pain Observation Tool (CPOT)
 (Gélinas et al., 2006)

Indicator	Score	Description	
Facial expression  Caroline Arbour, RN, B.Sc., PhD(student) School of Nursing, McGill University	Relaxed, neutral	0	No muscle tension observed
	Tense	1	Presence of frowning, brow lowering, orbit tightening and levator contraction or any other change (e.g. opening eyes or tearing during nociceptive procedures)
	Grimacing	2	All previous facial movements plus eyelid tightly closed (the patient may present with mouth open or biting the endotracheal tube)
Body movement:	Absence of movement or normal position	0	Does not move at all (doesn't necessarily mean absence of pain) or normal position (movements not aimed toward the pain site or not made for the purpose of protection)
	Protection	1	Slow, cautious movements, touching or rubbing the pain site, seeking attention through movements
	Restlessness/Agitation	2	Pulling tube, attempting to sit up, moving limbs/thrashing, not following commands, striking at staff, trying to climb out of bed
Compliance with the ventilator (intubated patients)	Tolerating ventilator or movement	0	Alarms not activated, easy ventilation
	Coughing but tolerating	1	Coughing, alarms may be activated but stop spontaneously
	Fighting ventilator	2	Asynchrony: blocking ventilation, alarms frequently activated
OR Vocalization (extubated patient):	Talking in normal tone or no sound	0	Talking in normal tone or no sound
	Sighing, moaning	1	Sighing, moaning
	Crying out, sobbing	2	Crying out, sobbing
Muscle tension Evaluation by passive flexion and extension of upper limbs when patient is at rest or evaluation when patient is being turned	Relaxed	0	No resistance to passive movements
	Tense, rigid	1	Resistance to passive movements
	Very tense or rigid	2	Strong resistance to passive movements or incapacity to complete them
TOTAL		___ / 8	

Assess Pain Scale every 4 hours. Self-reporting of pain should be used whenever appropriate.

Patient is in significant pain if BPS > 5; CPOT > 3

16. LEARNING ACTIVITIES

1. What are the four main functions of the respiratory system?
2. When the patient is in the upright position, in which of the 3 lung zones is V/Q normal?
3. What is anatomic dead space? What is alveolar dead space? Do they impact on ventilation and gas exchange?
4. How does lung compliance and resistance impact on tidal volume and airway pressures during mechanical ventilation?
5. How would you assess a patient's airway? Describe two artificial airway devices?

Activity: In the unit that you work in practice insertion of an artificial airway on the mannequin.

6. What are the three processes involved in oxygenation?
7. Explain how and why you need to measure DO_2 ? How would you improve DO_2 ?
8. List the causes that shift the oxyhemoglobin curve to the right. What happens to O_2 & Hb with a shift to the right?
9. What are the components of an Arterial blood gas. Interpret the following ABG.

The patient is on Non rebreather mask with 15L oxygen.

pH	7.339	Na	138.7
pO ₂	111	K	3.7
pCO ₂	29.6	Lactate	4.2
HCO ₃	15.5		
Base	- 8.9		
SaO ₂	98.4%		

10. **Activity:** Prepare and set up the equipment and drugs needed for Intubation.
11. How and why do you apply Cricoid pressure? Practice this on the mannequin.
12. What are the two main indications for positive pressure ventilation?
13. List at least three differences between CPAP & BiPAP.
Activity: Practice setting up a CPAP and BiPAP Circuit.
14. Draw a diagram and explain the four phases of ventilation.
15. List three types of triggers.
16. What three types of breaths can you have in a SIMV mode of ventilation?
17. Answer true or false: In volume controlled ventilation the tidal volume is constant and the airway pressure is variable.
18. Answer true or false: In pressure controlled ventilation the tidal volume is not determined by the set inspiratory pressure.
19. Draw a flow diagram of the ventilation emergency drill
20. What are the available methods of humidification?
Activity: Practice setting up an active humidified "Wet" water bath ventilation circuit.
21. What are the criteria for weaning mechanical ventilation?
22. List some of the complications of intubation and mechanical ventilation.

17. REFERENCE LIST

1. American Thoracic Society International Consensus Conferences in Intensive Care Medicine 2001, 'Non-invasive positive pressure ventilation in acute respiratory failure', *American Journal of Critical Care Medicine*, 163(1):283-291.
2. Antonelli M, Conti G, Moro ML, Esquinas A, Gonzalez-Diaz G, Confalonieri M, Pelaia P, Principi T, Gregoretti C, Beltrame F, Pennisi MA, Arcangeli A, Proietti R, Passariello M and Meduri GU 2001, 'Predictors of failure of non-invasive positive pressure ventilation in patients with acute hypoxemic respiratory failure: a multicenter study', *Intensive Care Medicine* (27): 1718 – 1728.
3. Bauman KA and Hyzy RC. 2015. Extubation management. 2015 UpToDate. www.uptodate.com Accessed 20th August 2015.
4. Blackwell N, Foot C & Thomas C 2007, *Examination Intensive Care & Anaesthesia*, Churchill Livingstone, Elsevier, Sydney.
5. Brochard L 2002, 'Non-invasive ventilation for acute respiratory failure', *JAMA*, 288(8):932-935.
6. Barr J. And et al 2013. Clinical Practice Guidelines for the Management of Pain, Agitation, and Delirium in Adult Patients in the Intensive Care Unit. *Critical Care medicine* 41:263-295.
7. Cairo, J.M. 2012 Pilbeam's Mechanical ventilation, 5th Edition, Mosby, St Louis
8. Cook, T. Woodall, TN. & Frerk, C. (2011) NAP4. 4th National Audit Project of the Royal College of Anaesthetists & The Difficult Airway Society. Major complications of airway management in the UK. Report and findings.
9. Critical Care Medicine Tutorials, Mechanical Ventilation Tutorial, viewed on 10th September 2015, www.ccmtutorials.com/rs/mv/index.htm
10. Elliot, D., Aitken, L. & Chaboyer, W. 2012. ACCCN,s Critical Care Nursing, 2nd edition, Mosby, Elsevier, Sydney.
11. Goodman, S. 2006. Implementing a Protocol for Weaning Patients Off Mechanical Ventilation. *British Association of Critical Care Nurses. Nursing in Critical Care*. 11 (1), 23-32.
12. Grossbach, I. Chlan, L. Tracy, M.F. 2011. Overview of Mechanical Ventilatory Support and Management of Patient- and Ventilator-Related Responses. *Critical Care Nurse*, vol.31 (3): 30-45
13. Guyton, AC & Hall, JE 2010, *Textbook of Medical Physiology*, 12th edition, W.B. Saunders Company, Philadelphia
14. Henderson, N 1999, 'Mechanical Ventilation', *Nursing Standard*, 13(44), 49-54.
15. Henderson JJ, Popat MT, Latto IP and Pearce AC. 2004 Difficult Airway Society guidelines for management of the unanticipated difficult intubation. *Anaesthesia* 59:675-694.
16. Heunks, L.M. & Van Der Hoeven, J. 2010. Clinical Review: The ABC of weaning failure – a structured approach. *Critical Care*. 14:245.
17. Hillman, K. & Bishop, G. 2004. *Clinical Intensive Care*. 2nd edition, Cambridge University Press, Melbourne.
18. ICCMU, 2013. Non- Invasive Ventilation Guidelines for patients with acute respiratory failure, <http://intensivecare.hsnet.nsw.gov.au/icwiki/index.php/Guidelines>
19. ICCMU, 2013, Suction of the artificial airway guideline, <http://intensivecare.hsnet.nsw.gov.au/icwiki/index.php/Suction>

20. Kidd, P & Wagner, K 1997, *High Acuity Nursing*, Appleton & Lange, Stamford.
21. Layfield C. Non-invasive BiPAP – implementation of a new service. *Intensive and Critical Care Nursing*. 2002. 18:310-319.
22. Lerman, J. 2009 On cricoid pressure: “may the force be with you”. *Anaesthesia & Analgesia*. 109(5):1363-1366.
23. Liesching T, Kwok H and Hill NS. Acute applications of non-invasive positive pressure ventilation. *Chest*. 2003. 124(2):699-713.
24. Lynn-McHale DJ & Carlson KK 2001, *AACN procedure Manual for Critical Care*, 4th Ed, WB Saunders Co, Philadelphia.
25. McCance, KL & Huerther, SE 2010, *Pathophysiology*, 6th ed, Mosby & Saunders, Philadelphia
26. Netting, S 2001, *Lippincott Manual of Nursing Practice*, 7th Ed, Lippincott, Williams and Wilkins, Baltimore
27. NSW Agency for Clinical Innovation. 2013. Care of adult patients in an acute facility with a tracheostomy. Clinical Practice Guideline. <http://www.aci.health.nsw.gov.au/>
28. NSW Health 2010. Tracheostomy Clinical Management (ADULT) PD2010_066 http://www0.health.nsw.gov.au/policies/pd/2010/PD2010_066.html
29. Oh, TE 2013, *Intensive care manual*, 7th edition, Butterworths, Sydney.
30. PATTS 2000, *Significant Body Systems, Pulmonary respiratory System*, viewed 19th September 2011, <http://webschoolsolutions.com/patts/systems/lungs.htm>
31. Percussionaire Corporation 2009, *Cardiopulmonary Ventilatory Management Contributions*, viewed on 19th September 2011, <http://www.percussionaire.com/historyreview.asp>
32. Pierce, L.N.B. 1995, *Guide to mechanical ventilation and intensive respiratory care*, W.B. Saunders, Philadelphia.
33. Pierce, L. 2010, *Management of the mechanically ventilated patient*, 2nd ed, W.B. Saunders, Philadelphia.
34. Poponick J.M., Renston J.P., Bennett R.P. and Emerman C.L. 1999. Use of a ventilatory support system (BiPAP) for acute respiratory failure in the emergency department. *Chest*. 116(1):166-171.
35. Rana. S., Jenad. H., Gay. P., Buck. C., Hubmayr. R. and Gajic. O. 2006. Failure of non-invasive ventilation in patients with acute lung injury: observational cohort study, *Critical Care* (10): R79.
36. Respirationics 2000, BiPAP Vision Ventilatory Support System, Clinical Manual.
37. Rittner & Doring 2005, *Curves and Loops in Mechanical Ventilation*, Draeger Medical Australia
38. Royal North Shore Hospital. 2013. Humidified High Flow Nasal Cannula Guideline, NHLHD.
39. Lynn-McHale, D.J. and Carlson K.K. (Eds.). 2001. *AACN procedure Manual for Critical Care*. (4th Ed.). WB Saunders Co. Philadelphia.
40. St. John, R.E. 2004. Airway Management. *Critical Care Nurse*, 24:93-97.
41. Temple, J.S. & Johnson, J.Y. 2010. *Nurses guide to clinical procedures*, Lippincott Williams & Wilkins Philadelphia, available via ciap, <http://www.ciap.health.nsw.gov.au>

42. Thille, A.W., Cortes-Puch, I. & Esteban, A. 2013. Weaning from the ventilator and extubation in ICU. *Current Opinion in Critical Care*, 19:57-64.
43. Tobin, MJ 1994, 'Current Concepts: Mechanical Ventilation' *The New England Journal Of Medicine*, 330,15, 1056-1061.
44. Tobin, M.J. 1991. 'What Should the Clinicians Do When a Patient "Fights the Ventilator"?' *Respiratory Care*, vol 36 (5):395-404.
45. Tortora, G. & Grabowski, S.R., 2003, *Principles of Anatomy and Physiology*, 10th ed, John Wiley & Sons, Inc, New York.
46. Truwit, E.D. & Epstein, S.K. 2011. Practical guide to mechanical ventilation, John Wiley & Sons, Inc, New York, <http://onlinelibrary.wiley.com/book/10.1002/9780470976609>
47. Urden, L. D., Stacy, K.M., & Lough, M.E. 2009. Critical Care Nursing: Diagnosis and Management, 6th edition, Chapter 24 – Pulmonary disorders. Mosby, Elsevier, St. Louise. Available via ciap <http://www.ciap.health.nsw.gov.au/>
48. West, J 1994, *Respiratory Physiology, the Essentials*, 5th ed, Williams & Wilkins Co. Baltimore
49. Vincent, J.L. 2005. Give your patient a fast hug (at least) once a day, *Critical Care Medicine*, vol. 33(6): 1225-1230.
50. West, J 2011, *Respiratory Physiology, the Essentials*, 9th edition, Williams & Wilkins Co. Baltimore
51. Williams, R., Rankin, N., Smith, T., Galler, D., Seakins, P. 1996. 'Relationship between the humidity and temperature of inspired gas and the function of the airway mucosa', *Critical Care Medicine*, 24,11:1920-1928.
52. Wikipedia, viewed on 20th September 2011, http://en.wikipedia.org/wiki/File:Oxyhaemoglobin_dissociation_curve.png
53. Woodruff, D.W. 1999. 'How to ward off complications of mechanical ventilation', *Nursing* 99, 29, 11: