Non-Invasive Ventilation (NIV) Learning Package

Department of Respiratory & Sleep Medicine
John Hunter Hospital

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NSW GOVERNMENT Health
Hunter New England
Local Health District

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Purpose:

For Registered and Enrolled Nurses, working within areas where Non-Invasive Ventilation (NIV) is a common part of treatment, to become competent in the delivery and management of NIV in both acute and chronic respiratory failure.

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Introduction

Non-invasive ventilation (NIV) is the delivery of respiratory support without the need for an invasive artificial airway (Sharma, 2006). Such ventilation has a role in the management of acute and chronic respiratory failure in many patients, and may have a role for some patients with heart failure. NIV can often eliminate the need for intubation or tracheostomy and preserve normal swallowing, speech, and cough mechanisms. Since the 1990s, the use of non-invasive positive pressure ventilation (NIPPV) has increased significantly in both the hospital and home setting.

The use of NIV for the management of acute hypercapnic respiratory failure is now seen as standard practice, and is associated with decrease patient morbidity, mortality and hospital length of stay.

(http://emedicine.medscape.com/article/304235-overview, 2014).

Disclaimer

This learning package has been developed by health professionals employed by the Hunter New England Local Health District's Respiratory and Sleep Medicine Department at John Hunter Hospital. While all care has been taken to ensure that the information is accurate at the time of development, the authors recommend that all information is thoroughly checked before use if utilised by another unit, context or organisation.

SDLP Naming Convention

Respiratory Medicine: Non-Invasive Ventilation

Aim

This package provides an introduction to the concepts and principles Non-Invasive Positive Pressure Ventilation (NIPPV). The aim of this package is to provide the theoretical knowledge required to become competent in the management of NIV in the ward setting.

Learning Outcomes

Completion of this learning package will enable the Registered/Enrolled Nurse to complete the related NIV competencies, and therefore demonstrate an understanding of the following:

- Type 1 & type 2 acute & chronic respiratory failure
- Indications & contraindications for NIV
- Venous & arterial blood gas interpretation
- Ventilator and mask management
- Nursing care of patients requiring NIV
- Complications of NIV
- Identification of NIV treatment failure

Pre-requisites

Although there are no prerequisites to undertake this self-directed learning package, it is expected that the participant will be working in an area where NIV is regularly utilised as part of treatment. This is essential to allow the knowledge to become embedded in practice.

Learning Package Outline

The package is designed to be a self-directed learning experience that will guide you through the literature and clinical issues related to NIV.

This package is developed within an adult learning framework so not all activities need to be documented but it is expected that you will complete them in order to facilitate your learning.

Once all parts of this learning package are completed successfully, including theoretical and practical assessments, a record will be added to your professional development record in HETI and you will be credited with 8 continuing professional development (CPD) hours.

Problem based learning

This program is based on a problem-based approach to learning. This approach has been chosen to enhance critical thinking, and to create a body of knowledge that the nurse can apply to practice. Problem based learning (PBL) is characterised by the use of patient specific problems or situations as a context for developing problem-solving skills and for acquiring clinical knowledge.

How to use this resource or Instructions for participants

- Completion of this package is equivalent to 8 CPD hours which is a requirement for National Registration. A certificate identifying CPD hours will be given on the successful completion of the package.
- This package can be used as an introduction for nurses wishing to further their knowledge and skills in this area.
- At the completion of this learning package you will be required to complete a problem based scenario related to NIV.
- There is a suggested reference list and it is by no means complete. Please read widely to facilitate your learning.
- This resource has been written from a Hunter New England Area Local Health District perspective so it is not specific to any one health facility. Throughout the package procedures from the John Hunter Hospital have been mentioned as an example of practice only.
- Throughout this self-directed learning package there are readings and activities that you will need to complete. You can access the readings online (journal articles) through CIAP. The online readings are not provided within this document due to copyright law restrictions. You will be provided with information on how to access the online readings. If you have any difficulty locating the readings please seek assistance from your hospital / health facility library.

Assessment process

1. Complete the theoretical assessment (pages 45 – 50) and return to your relevant CNE/CNC. A pass mark of at least 80% is required to move on to the practical assessment.
2. Complete the practical assessment. This is typically a 20 – 30 minutes session with your relevant CNE/CNC. See page 52.

Respiratory Physiology

A review of the physiology of the respiratory system.

Cells within the body require oxygen in order to access the energy they need from nutrients (during cellular metabolism). The body is unable to store oxygen for long periods of time; therefore it needs a continuous supply of oxygen. Metabolism produces carbon dioxide, which becomes an acid in the blood and must be removed from the cells. ***Respiration*** is the process of gas exchange between the atmospheric air and the blood and between the blood and the cells of the body to provide oxygen to and remove carbon dioxide from the cells. In order to work effectively it requires:

- Patent airway system to transport air to and from the lungs
- Effective alveolar system in the lungs to allow diffusion of gases into and out of the blood
- Effective cardiovascular system to carry nutrients and wastes to and from the body cells

The process of gas exchange has five components:

- Breathing
- External Respiration
- Internal Respiration
- Cellular Respiration
- Gas Transport

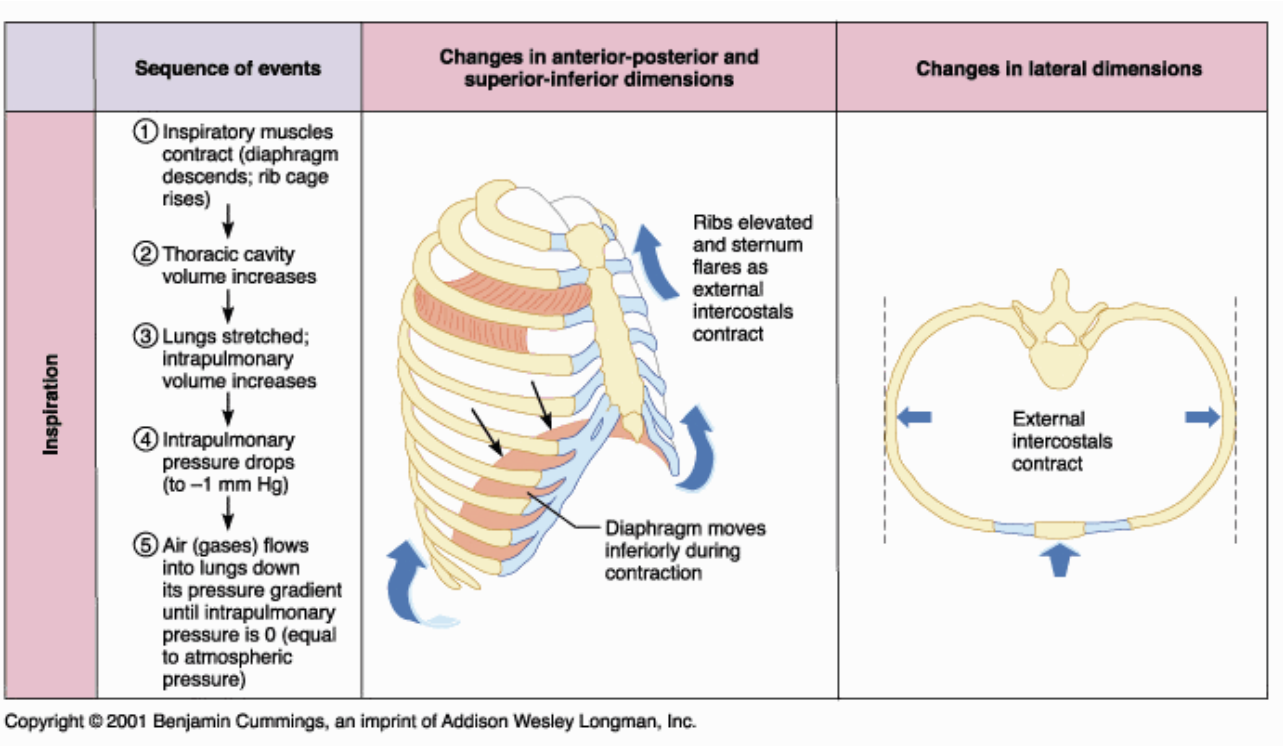
Breathing

Breathing, or ***ventilation***, is the movement of air through the airways between the atmosphere and the lungs. The air moves through the passages because of pressure differences between the atmosphere and the gases inside the lungs that are produced by

contraction and relaxation of the diaphragm and thoracic muscles. There are two phases of breathing; inspiration and expiration.

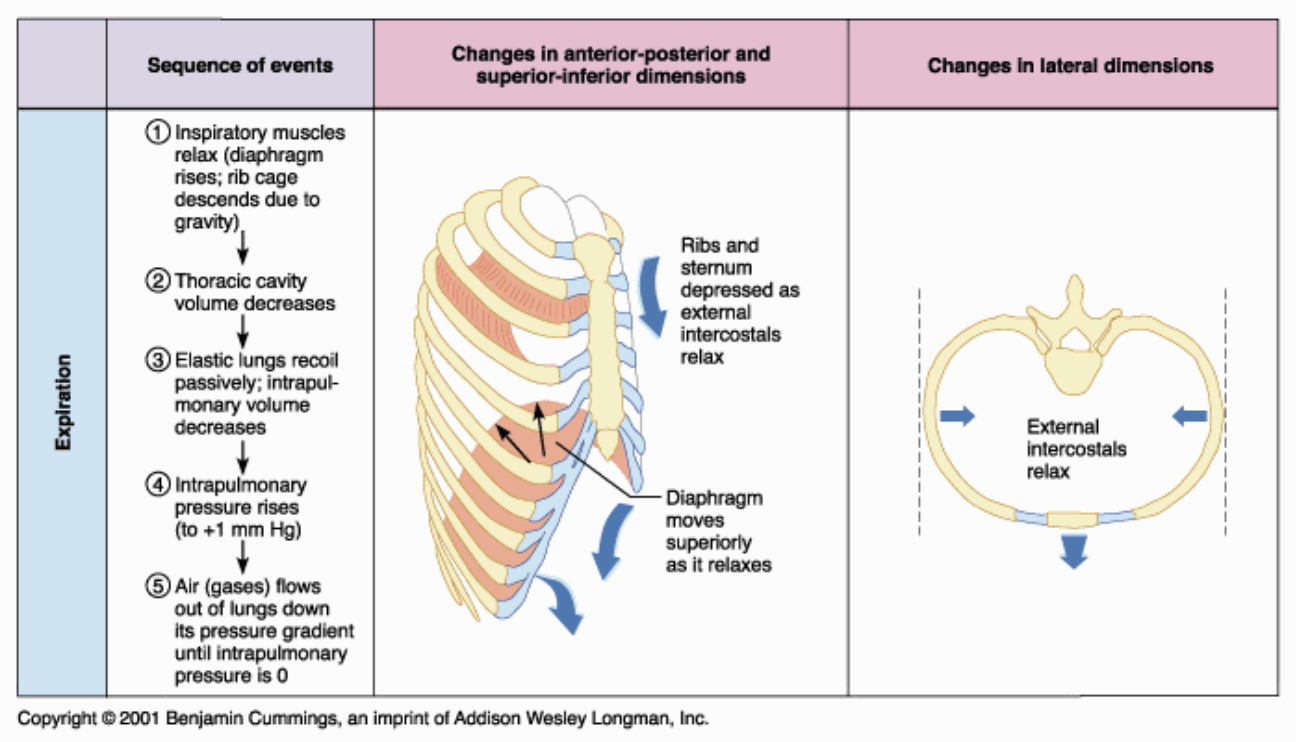
Inspiration

Inspiration is the process of taking air into the lungs. It is the active phase of ventilation because it is the result of muscle contraction. During inspiration, the diaphragm and intercostal muscles contract, enlarging the thoracic cavity. The diaphragm, doing most of the respiratory work during quiet breathing, moves downwards increasing the volume of the thoracic (chest) cavity, and the intercostal muscles pull the ribs up expanding the rib cage, further increasing this volume. This increased capacity lowers the air pressure in the alveoli to below atmospheric pressure. This decrease in intra-alveolar pressure draws air into the lungs as air, like other gases, flows from a higher pressure region to a lower pressure region.



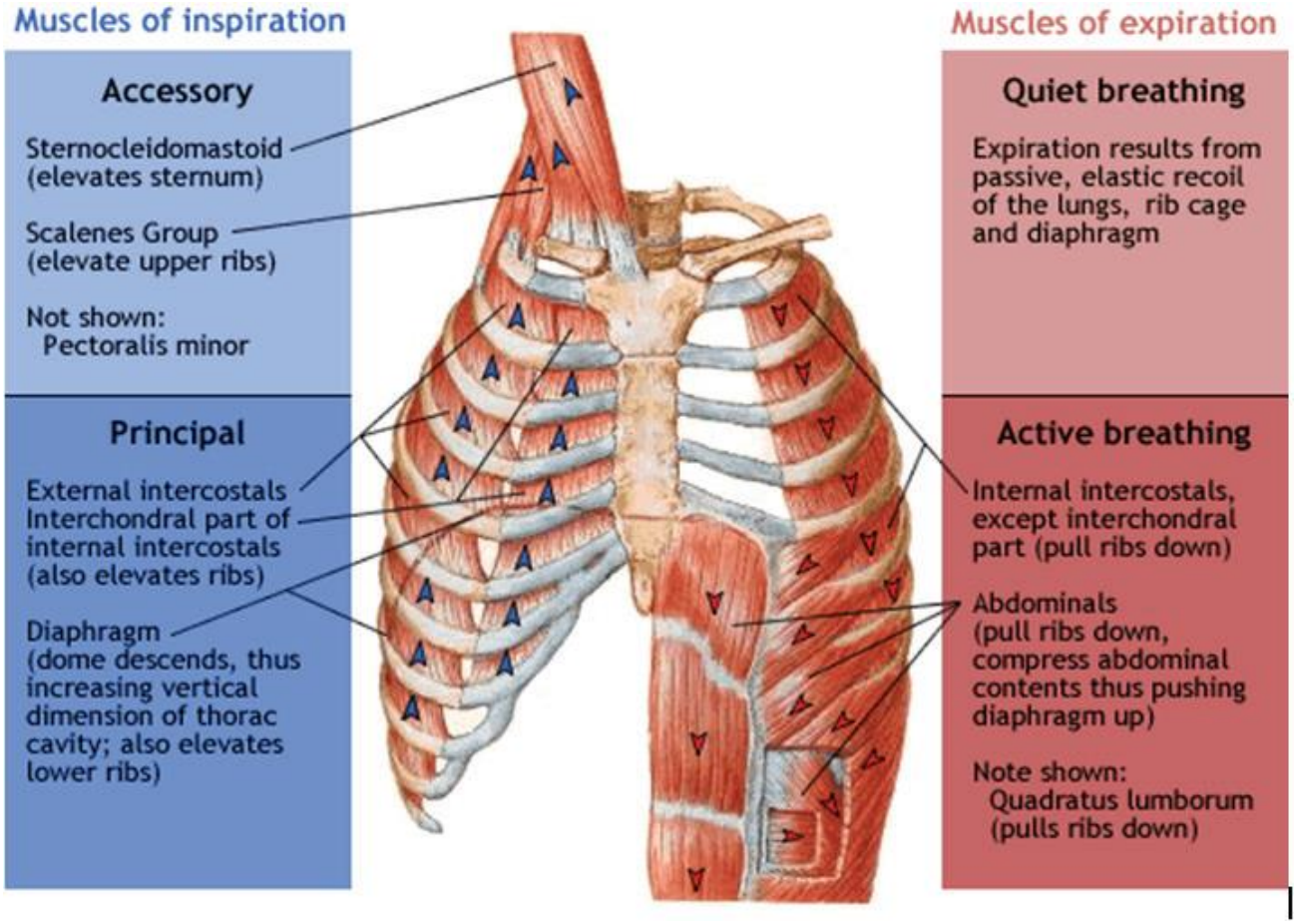
Expiration

Expiration is the process of letting air out of the lungs during the breathing cycle. During expiration the diaphragm and intercostal muscles relax. This returns the thoracic cavity to its original volume, increasing the air pressure in the lungs. The increase in intra-alveolar pressure pushes air out of the lungs. Expiration normally takes twice as long as inspiration.



Active Breathing

The body needs to be able to rapidly respond to changes in activity and therefore demand for energy. This means that when there is an increased demand for oxygen due to increased cellular metabolism, for example during exercise or illness, there also needs to be a corresponding increase in supply of oxygen. The increased metabolism will also result in increased production of carbon dioxide, which must also be removed. In order to increase gas exchange to meet the increased demand, extra muscles are used to increase the capacity of the respiratory system. The use of these extra muscles is often referred to as ***active breathing***. During active breathing accessory muscles are used during inspiration to lift the rib cage, creating a larger space within the thorax, further decreasing the pressure and causing a more rapid flow of air into the lungs. Expiration during active breathing becomes an active rather than passive action, with contraction of muscles to rapidly decrease the size of the thorax, thereby increasing the pressure and forcing air out of the lungs.



Lung volumes

Air movement in and out of the lungs is determined by the pressure gradient between the atmosphere and the alveoli. The volume of air inhaled and exhaled with each breath is called the ***tidal volume***. The pressure gradient, and therefore respiratory effort, required to obtain a particular tidal volume may be affected by the lung compliance and the resistance of the airways.

Lung compliance is the dispensability or “stretchability” of the lung and the elastic recoil back to its original shape. Lung compliance is affected by connective tissue and alveolar surface tension. A highly compliant lung will expand easily when pressure is applied; however a poorly compliant lung requires a greater than normal pressure, and therefore effort, to expand it.

The ***resistance*** of the airways refers to the opposition to gas flow through the airways. It is primarily determined by the radius of the airway, with a smaller bronchial diameter increasing the resistance or opposition to air flow, therefore slowing down the air flow for a particular pressure gradient. An airway with high resistance will therefore require greater than normal pressure gradient and effort to achieve normal levels of ventilation.

External Respiration

Once the air has reached the alveoli, gas exchange between the air and blood can occur, known as ***external respiration***. In order for effective gas exchange, there not only needs to be good ventilation but also good perfusion, or circulation, to the ventilated alveoli.

The pulmonary circulation is a low-pressure system, able to vary its resistance to accommodate the blood flow received and alter the direction of blood flow to well ventilated areas. Due to the low pressures however, the distribution of blood is greatly affected by gravity, with minimal perfusion to the lung apices when in an upright position. It is also affected by alveolar pressure, as high alveolar pressures will cause compression of the capillaries and therefore restrict pulmonary blood flow to the area.

The movement of oxygen and carbon dioxide between the alveoli and capillaries is controlled by diffusion, with gas moving across the alveolar membrane from areas of high concentration to areas of low concentration.

Gas diffusion is affected by:

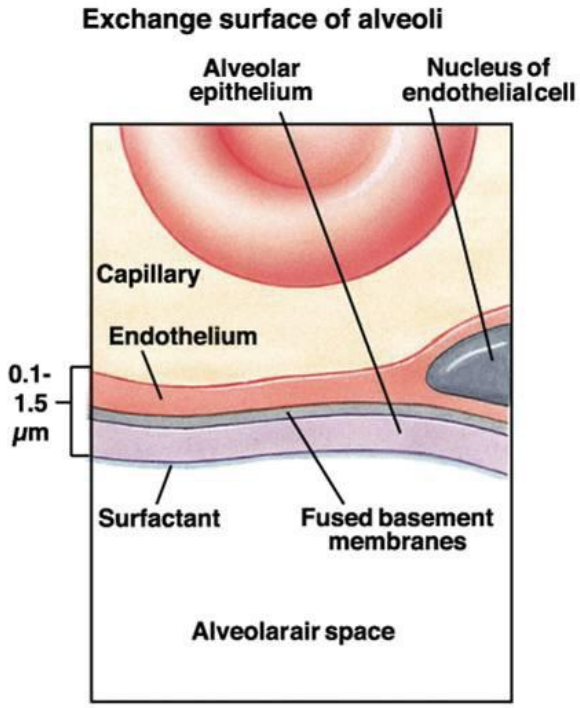
- Thickness of barrier
- Surface area available for exchange
- Concentration gradient
- Solubility of gas

The alveolar-capillary membrane is ideal for diffusion as it has a thin membrane, as thin as 0.3 micrometres in some areas, and a large surface area of 50-100 m2 in a normal lung. For gas to get between the air and the red blood cells it must diffuse through the following layers:

- Alveolar fluid
- Alveolar epithelium

- Alveolar basement membrane
- Interstitial space
- Capillary basement membrane
- Capillary endothelium
- Plasma

Any changes to the surface area or layers will affect the diffusion of gases.



Gas concentrations are expressed as partial pressures. In a mixture of gases, each gas contributes to the total pressure according to its concentration. For example if a gas is 50% of total, it produces 50% of the pressure. The pressure of room air, or atmospheric pressure, at sea level is 760 mmHg. This pressure is made of different concentrations of gases – with approximately 78% nitrogen, 21% oxygen, 0.03% carbon dioxide and 0.05% water vapour. The partial pressure of oxygen in room air is therefore 21% of 760 mmHg.

When air enters the trachea it is humidified, becoming fully saturated with water vapour. The water vapour, now taking up approximately 6% (47 mmHg) of the pressure, displaces other gases and reduces their concentrations (Smeltzer & Bare, 1992). In the alveoli there

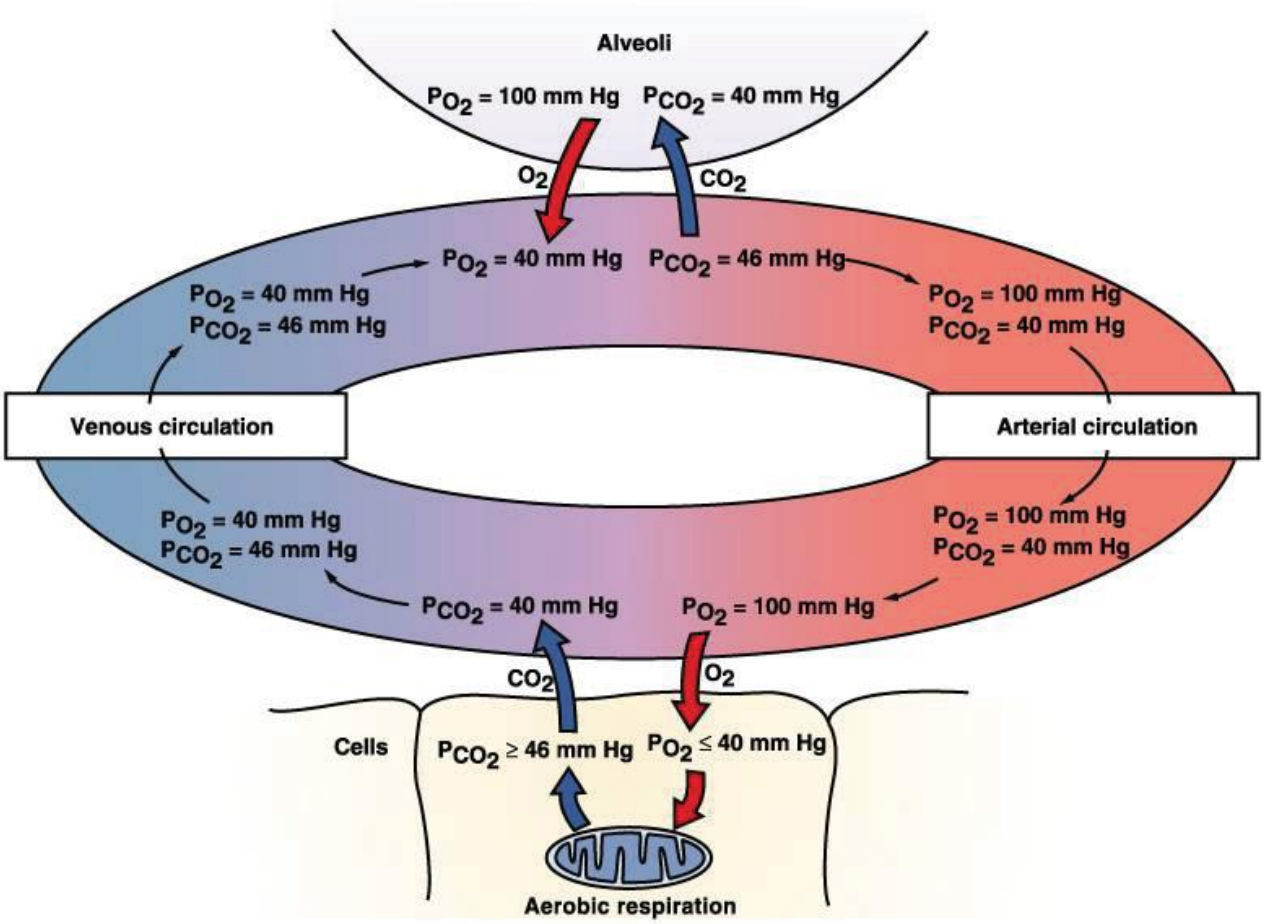
is some residual carbon dioxide, which further alters the balance of concentrations, with the resulting partial pressures within normal alveoli:

- Nitrogen, 569 mmHg (74.9%);
- Oxygen, 104 mmHg (13.6%);
- Carbon dioxide, 40 mmHg (5.3%);
- Water vapour, 47 mmHg (6.2%).

The speed of oxygen and carbon dioxide diffusion across the alveolar membrane is affected by the size of the ***concentration gradient***. The bigger the difference between concentrations on either side, the faster the gas will move. The biggest gradient, and therefore fastest diffusion of gas, occurs when fresh gas is brought in to the alveoli during inspiration.

In the alveolar capillaries the blood is returning, through the right side of the heart, from the tissues, where oxygen has been used and carbon dioxide produced during cellular metabolism. The capillary oxygen levels are therefore usually low, and the carbon dioxide levels high in comparison to the alveolar gases. The differences between the ***concentration gradients***, allow movement of oxygen from the alveoli into the blood, and carbon dioxide from the blood into the alveoli.

The diffusion of a gas is also affected by the gas's ***solubility***. Oxygen and carbon dioxide must be able to dissolve into the alveolar fluid and then the blood in order to be transported to and from the tissues. When gas is exposed to a liquid, the gas will dissolve in the liquid until the concentration, or partial pressure, of the gas is the same in the liquid and the gas. This means that oxygen and carbon dioxide are exchanged across the alveolar membrane until the partial pressure is the same in the alveoli and the blood. Some gases dissolve more quickly and easily than others. Carbon dioxide, for example, dissolves approximately 20 times faster than oxygen. It is therefore relatively unaffected by increased fluid in the alveoli or interstitial space, whereas oxygen diffusion may be affected. As carbon dioxide diffuses and equalises so quickly, the best way to remove more from the blood is to replace the air in the alveoli with new air to re-establish the concentration gradient and therefore restart the diffusion process.



Internal Respiration

The process of gas exchange between the blood and the cells, or ***internal respiration***, is the same as for external respiration. Movement of the gases is primarily affected by the concentration gradient between the blood and the cells. When oxygen-enriched blood comes in contact with tissue with a lower PaO2, oxygen will move from the blood into that tissue. Also, when the partial pressure of carbon dioxide (PaCO2) in the tissue exceeds that of the blood, carbon dioxide will move from the tissue into the blood to be transported to the lungs. Metabolic changes, as well as increases in interstitial fluids may affect the diffusion of oxygen into the cells, and therefore impair cell function.

Cellular Respiration

Cellular respiration, or cellular metabolism, is the process of deriving energy, in the form of Adenosine Triphosphate (ATP), from molecules such as glucose. The cells break down glucose either with or without oxygen. When a glucose molecule is broken down without oxygen (anaerobic metabolism) 2 ATP molecules are produced, however in the presence of

oxygen (aerobic metabolism) most cells can produce a further 34 ATP molecules. Oxygen is therefore essential for energy-efficient metabolism to produce enough energy to maintain normal cell function.

Gas Transport

Oxygen and carbon dioxide are transported between the lungs and the cells in the blood stream. Some of the gas is transported dissolved in the plasma; however the majority is transported combined with some of the elements of the blood. Gas transport is therefore reliant on the adequate functioning of the cardiovascular system. Changes to circulation (such as poor cardiac output) or components of the blood (such as anaemia) will affect the ability of gas to be transported to and from the lungs and cells.

Oxygen transport

The oxygen that is dissolved in the plasma of arterial blood, measured as a partial pressure or PaO2, is in a form that is readily available for diffusion to the tissues. The poor solubility of oxygen, however, limits the amount of oxygen that can be dissolved in the blood. The body therefore needs to have a reserve of oxygen that can be made available in periods of increased demand, such as exercise or illness.

Haemoglobin (Hb), found in the red blood cells, significantly enhances the oxygen carrying capacity of blood and providing a reserve supply. For every 100ml of blood, approximately 0.3mls of oxygen is physically dissolved in the plasma, however approximately 20mls of oxygen is present combined with haemoglobin (which becomes oxyhaemoglobin). At rest only 30% of the oxygen on the haemoglobin is normally used by the tissues. Haemoglobin is made up of iron-containing haem molecules combined with the protein globin. The iron in haem is able to reversibly bind an oxygen molecule. This means that oxygen can bind to Hb in the lungs and then be released at the tissues. There are four iron atoms in each Hb molecule comprising four haem groups. Each Hb molecule can therefore bind with four oxygen molecules. When oxygen is bound to all 4 haem groups, the Hb is said to be fully saturated.

In the loading and unloading of oxygen, there is cooperation between the four haem groups. When oxygen binds to one of the groups, the others change shape slightly and their attraction to oxygen increases. The loading of the first oxygen results in the rapid loading of the next three (forming oxyhaemoglobin). At the other end, when one haem group unloads its oxygen, the other three rapidly unload as their groups change shape

again having less attraction for oxygen. This method of cooperative binding and release can be seen in the dissociation curve for haemoglobin. Over the range of oxygen concentrations where the curve has a steep slope, the slightest change in concentration will cause haemoglobin to load or unload a substantial amount of oxygen.

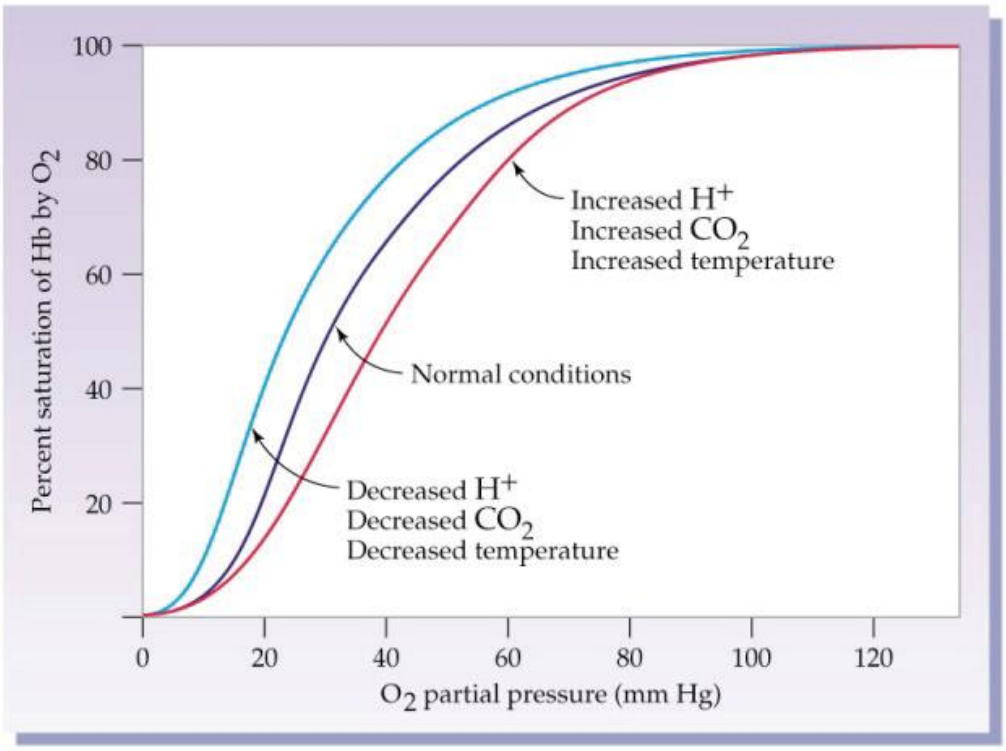
The major factor that determines the movement of oxygen onto the haemoglobin is the amount of oxygen dissolved in the plasma (PaO2). As the concentration of oxygen in the plasma increases, more oxygen combines with the haemoglobin, until it is fully saturated i.e. oxygen is bound to all 4 haem groups. Haemoglobin usually becomes 100% saturated, under normal conditions, at a PaO2 of 150mmHg. In healthy person breathing room air, the expected arterial oxygen levels would be a PaO2 100mmHg (achieving equalisation with alveolar oxygen concentration) and a corresponding SaO2 of 97%. Table below shows the relationship between PaO2 and SaO2 at various levels:

- At PaO2 150mmHg, even though the PaO2 has been increased by nearly 50%, there is minimal change in the SaO2 as the haemoglobin is almost completely saturated and cannot combine with any more oxygen.
- PaO2 60mmHg, when the symptoms of hypoxia normally begin, although there is a drop of nearly half the dissolved oxygen concentration, the haemoglobin is still 90% saturated at this level. Small changes in SaO2 at this level correspond with large changes in PaO2.

PaO2	SaO2
150 mmHg	100 %
100 mmHg	97 %
60 mmHg	90 %
40 mmHg	70 %

It is vital to the delivery system for the oxygen to bind and release from the haemoglobin at the right time and the right place. The oxyhaemoglobin dissociation system is designed to facilitate loading of oxygen onto the haemoglobin in the lungs, and offloading of oxygen in

the systemic capillaries to supply the tissues. Factors such as the temperature, pH and carbon dioxide differ from the systemic to the pulmonary capillaries. The systemic capillaries provide oxygen for and carry wastes from cellular metabolism. It is here that we need oxygen to easily leave the haemoglobin. The conditions in the systemic capillary are greatly affected by the cellular metabolism that is occurring around it. There are low levels of dissolved oxygen (PaO2) as it is consumed by the cells, but high levels of carbon dioxide (and therefore a low, or acidic, pH) and heat produced during the metabolism. These factors, low oxygen, high CO2 and high temperature all affect the binding of oxygen to the haemoglobin, helping it to release easily.

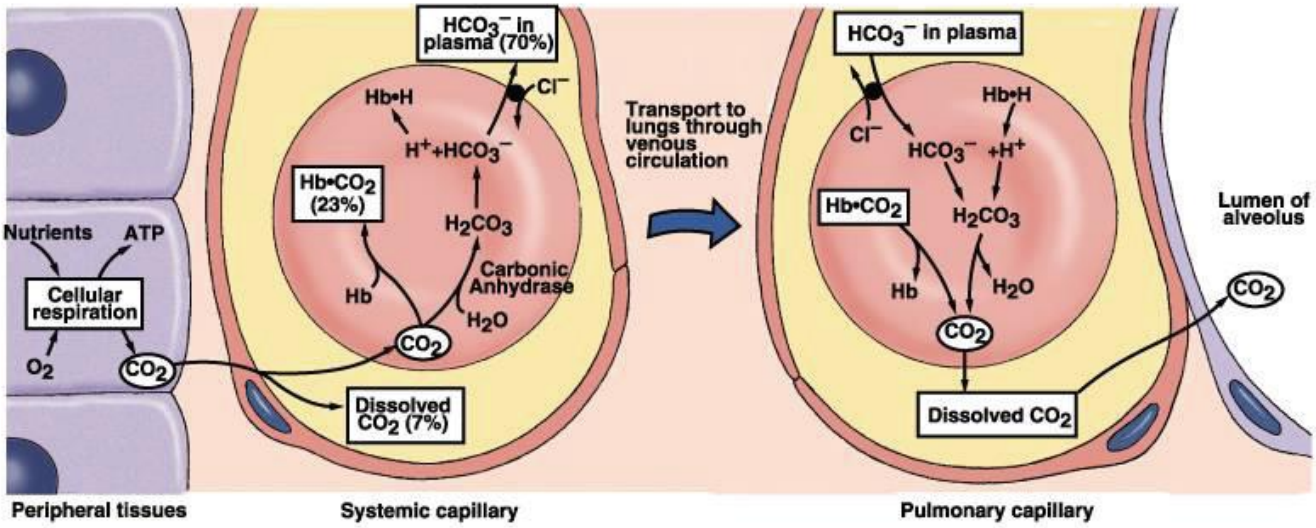


Oxyhaemoglobin dissociation curve

The pulmonary capillaries allow release of carbon dioxide into the atmosphere and bring oxygen into the circulation. The blood, having returned from the tissues, has low oxygen levels, however as carbon dioxide quickly and easily diffuses into the alveoli, the plasma CO2 level in the pulmonary capillary is low, as is the temperature as heat is lost over the membrane. In these conditions, oxygen binds more strongly to the haemoglobin, allowing the haemoglobin to be saturated with oxygen to then be transported back to the cells.

Carbon dioxide transport

Carbon dioxide created during cellular metabolism diffuses into the blood plasma with over 90% then entering the red blood cells. Once in the red blood cell approximately 23% binds to the multiple amino groups of haemoglobin to form carboxyhaemoglobin, whilst the majority (approximately 70%) is converted to bicarbonate ions and released into the plasma. The amount of carbon dioxide being transported in the blood is one of the major determinants of the acid-base balance of the body. When carbon dioxide enters the plasma, it reacts with water to form carbonic acid. Carbonic acid is a strong acid and readily donates its hydrogen ions. An increase in carbon dioxide levels within the blood will therefore cause an acidosis. As discussed previously, the carbon dioxide diffuses easily across the alveolar membrane, equalising quickly with the alveolar gas. The arterial carbon dioxide level is therefore usually equivalent to the partial pressure of carbon dioxide in the alveoli, i.e. 40 mmHg.

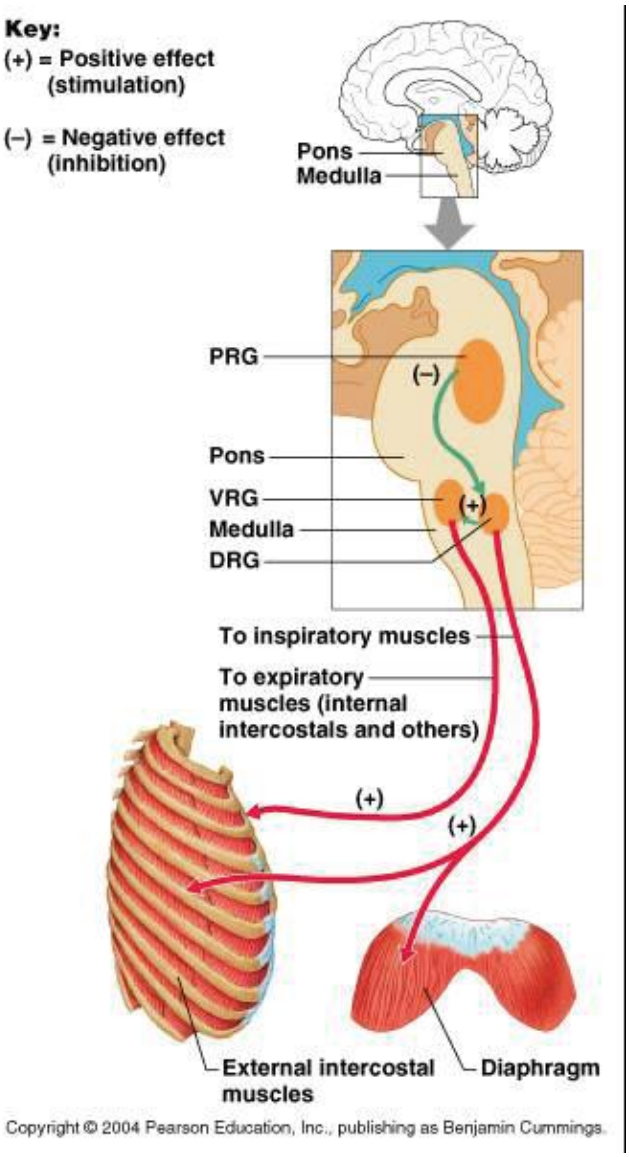


Control of Breathing

Central Control of Breathing

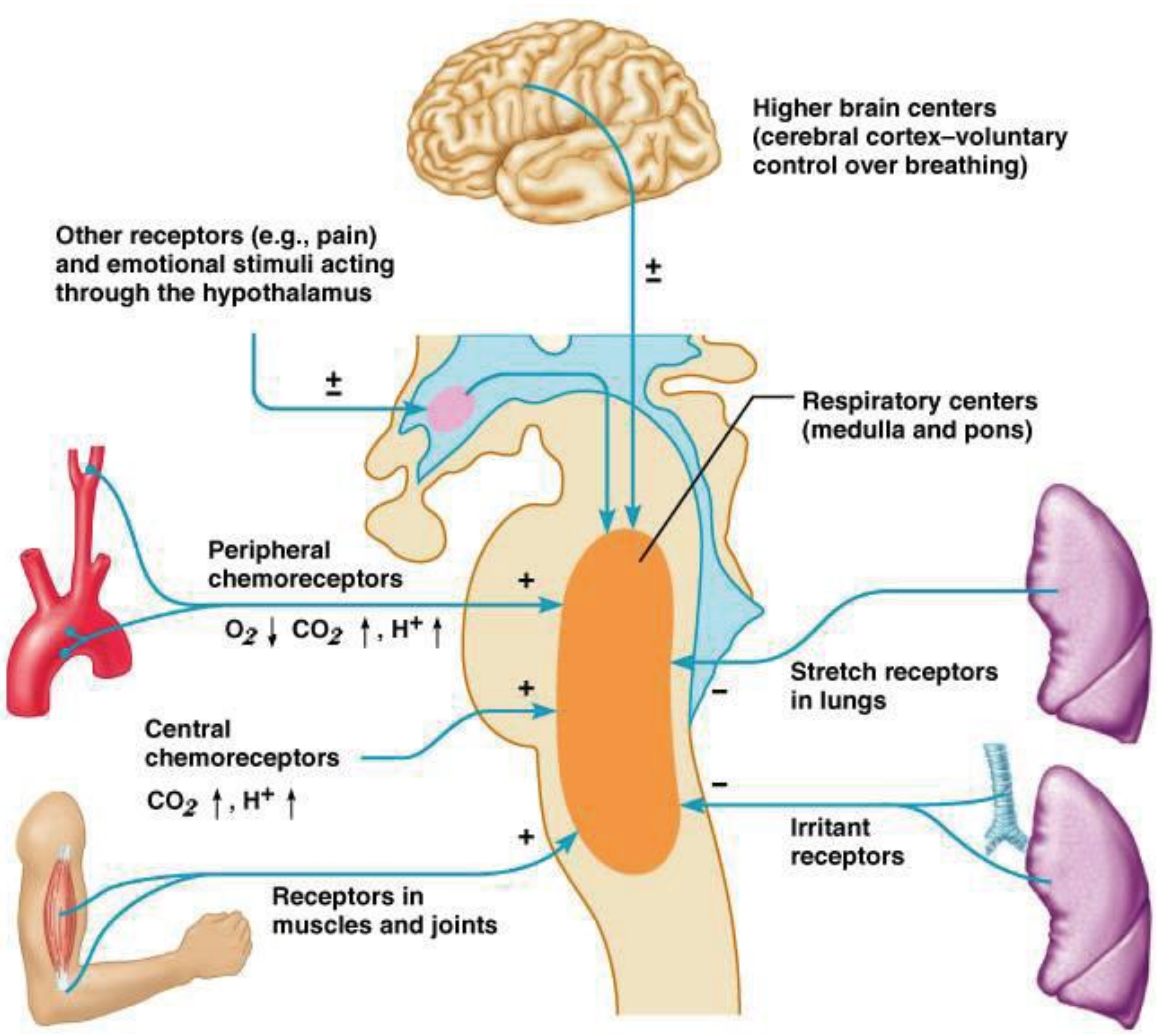
The respiratory centre is found in the brain stem. The pons and medulla oblongata are both integral to the control of breathing. The medulla oblongata rhythmically stimulates the intercostal muscles and diaphragm — making breathing possible. The pons also participates in the reflexes that regulate breathing. The brain stem receives signals from various organs in order to detect changes and respond to changes in physical demands of the body. It receives positive and negative stimuli to determine the respiratory rate and depth required. The rate of cellular respiration (and hence oxygen consumption and carbon dioxide production) varies with the level of physical activity. Vigorous exercise can increase tissue oxygen demand by 20-25 times that at rest. An increase in physical activity, and therefore cellular metabolism, will result in increased carbon dioxide levels and acidity, which is detected by peripheral and central chemoreceptors which provide a positive stimulus to the brainstem to cause an increase in ventilation, i.e. increase in rate and depth of breathing.

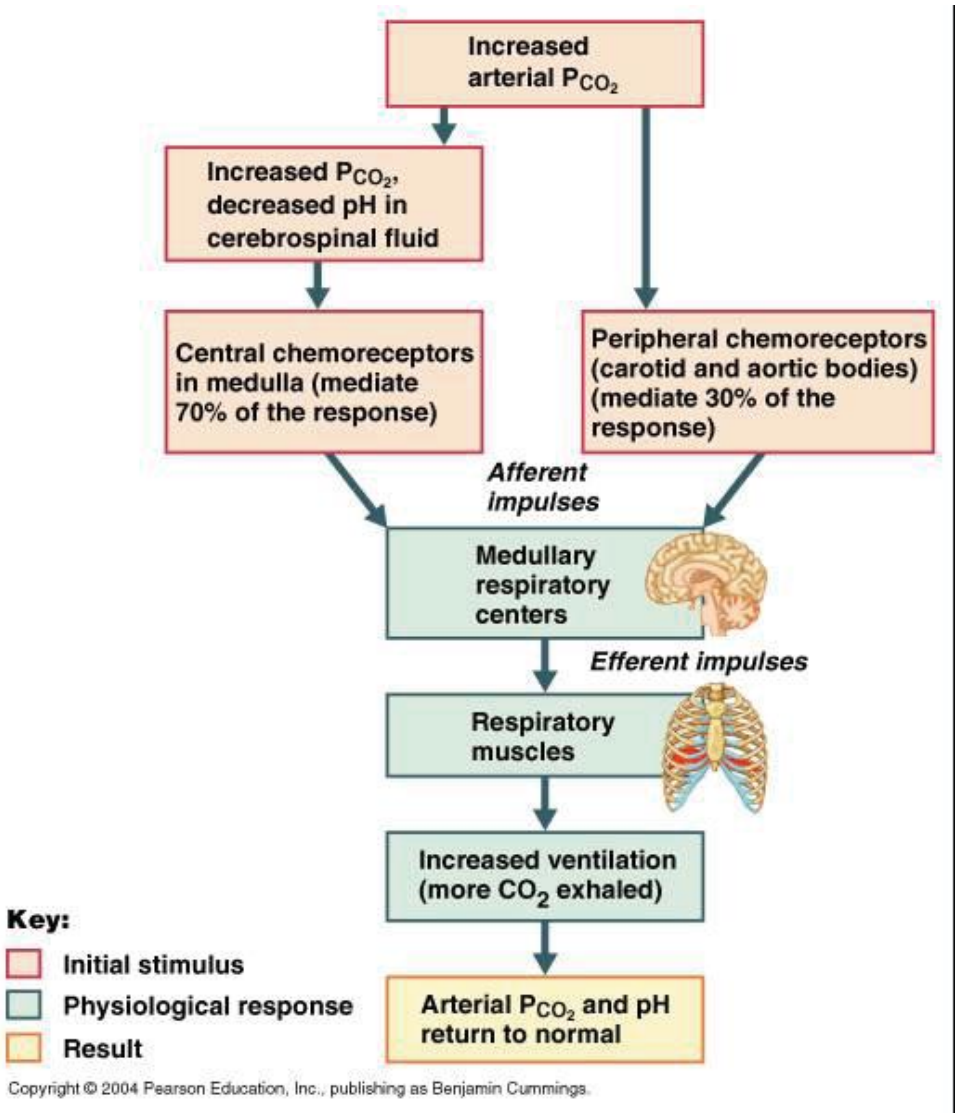
Stretch receptors in the lungs detect over distension, resulting in a negative stimulus to the brain stem, reducing the rate and depth of breathing, protecting against trauma to the airways. Irritant receptors in the bronchi and lungs will also cause a reduction in ventilation, to help prevent deep inhalation of irritants into the lower airways.



Breathing may be affected by emotional factors, such as fear, anxiety, or pain. Signals are transferred through the hypothalamus to the brain stem to affect ventilation. We are also capable of voluntary or conscious control over breathing. The brain stem receives signals from the higher brain centres to increase or decrease ventilation accordingly.

The most important factor in regulating ventilation is a rising concentration of carbon dioxide - not a declining concentration of oxygen. The concentration of carbon dioxide is detected by cells in the medulla by changes in the pH of the CSF. If the carbon dioxide level rises, the medulla responds by increasing the activity of the motor nerves that control the intercostal muscles and diaphragm. However, the carotid body in the carotid arteries does have receptors that respond to a drop in oxygen. Their activation is important in situations where oxygen supply is inadequate but there has been no increase in the production of CO2, for example at high altitude in the unpressurised cabin of an aircraft, or in situations of long term hypercapnia.





Local Control of Breathing

In addition to central control affecting the rate and depth of breathing, there is also a local control within the lungs. The smooth muscle in the walls of the bronchioles is very sensitive to the concentration of carbon dioxide. A rising level of CO2 causes the bronchioles to dilate. This lowers the resistance in the airways and thus increases the flow of air in and out.

Pathophysiology

An overview of respiratory conditions and their effect on gas exchange.

Effective gas exchange relies on each part of the respiratory process to be functioning. An interruption at any point will affect the ability of the body to supply oxygen to the tissues and remove carbon dioxide. We will focus on some of the respiratory conditions and their impact on gas exchange. Impaired pulmonary function can be classified into two main types:

- Oxygenation impairment – inadequate arterial oxygenation, or hypoxia.
- Ventilation impairment – inadequate carbon dioxide removal, or hypercapnia, in the presence of normal alveolar-arterial (A-a) gradient.

Oxygenation impairment

Diffusion of oxygen, as previously discussed, is greatly affected by any changes to the air-blood barrier in the alveoli. Due to its poor solubility, any changes in the surface area, thickness of the barrier or increased fluid can result in inadequate oxygenation of the arterial blood. Not all of the oxygen can diffuse into the capillaries, with a higher concentration still left in the alveoli (called an A-a gradient). Treatment of oxygenation impairment requires supplementing oxygen (which increases the concentration gradient, speeding up oxygen diffusion) to ensure tissue hypoxia doesn't occur, whilst treating the cause; e.g. increase surface area, reduce alveolar/interstitial fluid, improve ventilation-perfusion match.

Ventilation impairment

Ventilation impairment, or inadequate exchange of gas through the airways between the atmosphere and the lungs, is characterised by high arterial carbon dioxide levels, respiratory acidosis and increased work of breathing. Inadequate spontaneous ventilation may be caused by a reduced drive to breathe, as is found in CNS disorders; or a reduction in tidal volumes from neuromuscular, musculoskeletal, pleural, or conducting airways disorders. As carbon dioxide excretion is dependent on the amount of air it can exchange with, any changes to the respiratory rate or tidal volume (and therefore the minute ventilation) affects the arterial carbon dioxide level, resulting in hypercapnia. Treatment of ventilation impairment is focused on increasing the respiratory rate by minimising any

central respiratory depressants or improving tidal volumes by reducing the work of breathing and supporting the inspiratory effort.

*Minute Ventilation is the amount of air (normally expressed in litres) exchanged between the lungs and atmosphere over 1 minute.

Therefore:

$$\mathrm{VE} = \frac{(\mathrm{VT} \times \mathrm{RR})}{1000}$$

VE = Minute Ventilation (L/min)

VT = Tidal Volume (mL)

RR = Respiratory Rate (bpm)

Acute Respiratory Failure

Respiratory failure is a syndrome in which the respiratory system fails in one or both of its gas exchange functions: oxygenation and carbon dioxide elimination. The lungs cannot maintain adequate alveolar ventilation. Acute respiratory failure is the most common indication for mechanical ventilation in the Intensive Care Unit. Respiratory failure may be acute or chronic. Acute respiratory failure is characterised by life-threatening alterations in arterial blood gases and acid-base status. Respiratory failure can be defined as a PaO2 value of less than 60 mmHg (SpO2 < 90%) while breathing air, or a PaCO2 of more than 50 mmHg. The manifestations of chronic respiratory failure are less dramatic and may not be as readily apparent. Common causes of acute respiratory failure include pneumonia, apnoea, neuromuscular dysfunction, head trauma, cardiac arrest, or drug-induced central nervous system (CNS) depression.

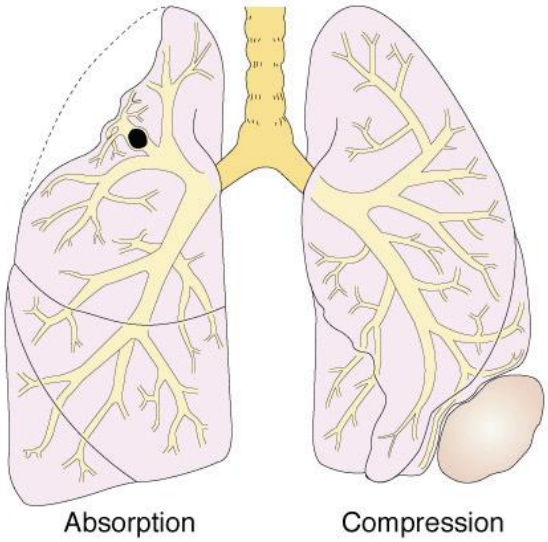
- Type I: Hypoxia PaO_2 <60mmHg (low levels of oxygen and normal levels of carbon dioxide)
- Type II: Hypoxia with Hypercapnia $PaCO_2$ >45mmHg (low levels of oxygen and high levels of carbon dioxide)

Common Respiratory Disorders

Atelectasis

Atelectasis refers to the collapse of an area of lung, which could be an alveoli, lobule or larger lung unit. Atelectasis may result from:

- Airway obstruction (e.g. from sputum blocking a bronchiole), preventing airflow into the alveoli. The gas remaining in the alveoli eventually gets absorbed into the capillary, and with no fresh air to replace it, the alveoli collapse. Failure to adequately remove secretions, due to neurological or respiratory disorders, commonly causes atelectasis.
- Compression of the lung tissue, limiting expansion and restricting air movement into the alveoli. The lung tissue may be compressed by air or fluid in the pleural space (i.e. pneumothorax or pleural effusion), enlarged heart, pericardial effusion, thoracic tumour, patient positioning preventing adequate lung expansion, or abdominal distension pushing the diaphragm upward.
- Failure of the normal splinting mechanisms
- Loss or dilution of surfactant in the alveoli will increase the surface tension, causing collapse of the alveoli.
- Loss of nitrogen in the alveoli due to inhaling high concentrations of oxygen. Nitrogen normally comprises approx. 75% of the gas in the alveoli. It is a large molecule that does not diffuse across the alveolar membrane, assisting in splinting the alveoli open. If high concentrations of oxygen are delivered, there will be a consequential drop in the percentage of nitrogen. If all the alveolar oxygen diffuses into the capillary before it is refreshed with new gas, the alveoli can collapse.
- Decrease in alveolar pressure, which may cause collapse of the terminal bronchioles, which do not have any cartilage to splint them open.



Acute Pulmonary Oedema

Acute pulmonary oedema (APO) is the abnormal accumulation of fluid in the lungs, either in the interstitial spaces or in the alveoli. An increase of fluid at the air-blood interface impairs the ability of oxygen to diffuse into the capillaries (with carbon dioxide being largely unaffected due to its high solubility). An increase in fluid within the alveoli causes a dilution of the alveolar surfactant, which may result in collapse of the alveoli. APO may be caused by left heart failure (cardiogenic APO) which results in increased pulmonary blood flow and pressure, causing fluid to leak out of the capillaries into the interstitial airways. Non-cardiac APO may also be caused by conditions that increase the pulmonary pressures, affect the colloid osmotic pressure (e.g. in nephritis), increase the permeability of the pulmonary capillaries (e.g. systemic inflammation) or damage to the capillary walls (e.g. inhalation of noxious gases, pneumonia and Acute Respiratory Distress Syndrome [ARDS]).

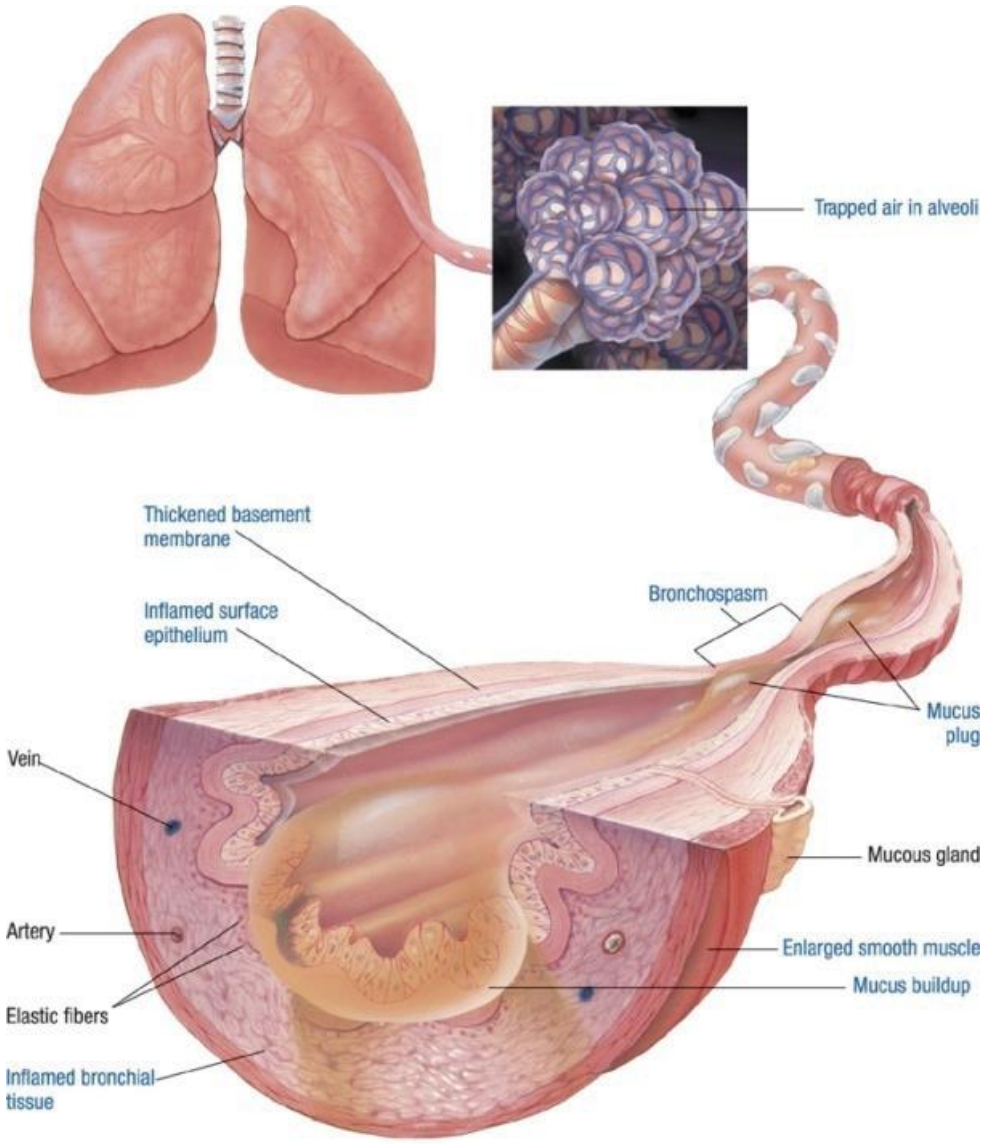
Pneumonia

Pneumonia is an infection of the alveoli. It can be caused by many kinds of bacteria (e.g. Streptococcus pneumoniae) and viruses. Fluid accumulates in the alveoli, reducing oxygen diffusion as well as diluting the surfactant causing collapse of the alveoli and therefore reducing the surface area exposed to air. Treatment includes clearance of secretions, and antibiotic treatment, if appropriate. If enough alveoli are affected, oxygenation is impaired and the patient may need supplemental oxygen.

Asthma

In asthma, periodic constriction of the bronchi and bronchioles makes it more difficult to breathe in and, especially, out. The swollen walls and increased secretions narrow the airways, increasing resistance and reducing air flow. During inspiration the airways are pulled open, however during expiration, the elastic recoil causes some obstruction of the airways, trapping air in the alveoli. The resultant ventilation impairment causes a rise in carbon dioxide levels. Attacks of asthma can be triggered by:

- Food and environmental allergies; e.g. dust mites, pollens, animal dander
- Air borne irritants; e.g. chemical fumes, pollution, cigarette smoke
- Exercise
- Drugs; e.g. aspirin, β-blockers, NSAIDs

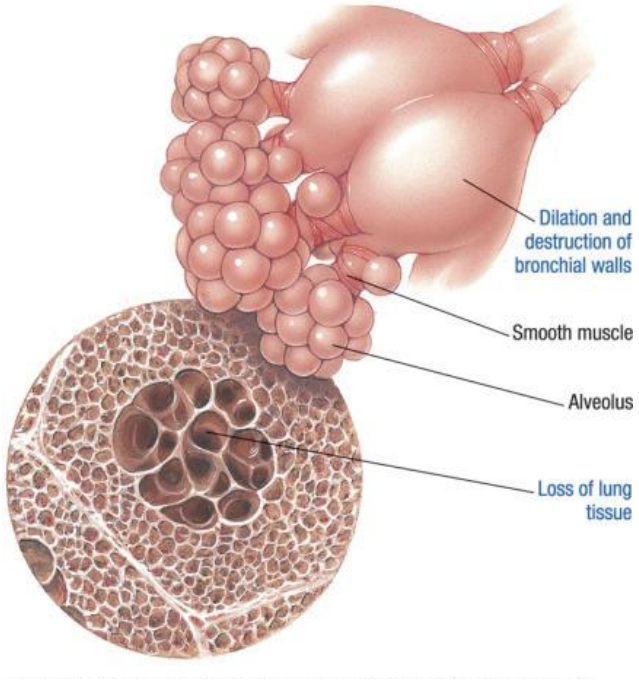


Chronic Bronchitis

Any irritant reaching the bronchi and bronchioles will stimulate an increased secretion of mucus. In chronic bronchitis inflammation and thickening cause narrowing of the airways, with increased mucous secretions leading to a persistent cough. Oxygenation is impaired as diffusion is difficult through the secretions and due to alveolar collapse. Chronic bronchitis is usually associated with cigarette smoking.

Emphysema

In emphysema, the delicate walls of the alveoli break down, reducing the gas exchange area of the lungs. They are grouped into three types according to where in the alveolar unit the tissue break down occurs. The condition develops slowly and is seldom a direct cause of death. However, the gradual loss of gas exchange area forces the heart to pump ever-larger volumes of blood to the lungs in order to satisfy the body's needs. The added strain can lead to heart failure.

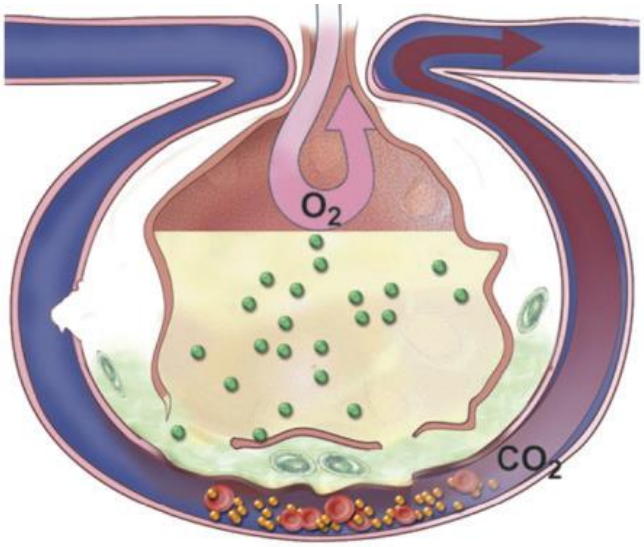


Chronic Obstructive Pulmonary Disease (COPD)

Irritation of the lungs can lead to asthma, emphysema, and chronic bronchitis. And, in fact, many people develop two or three of these together. This combination is known as (COPD).

Acute Respiratory Distress Syndrome (ARDS)

Acute respiratory distress syndrome is the clinical manifestation of severe, acute lung injury. It is characterized by the acute onset of diffuse, bilateral pulmonary infiltrates secondary to non-cardiogenic pulmonary oedema, refractory hypoxia, and decreased lung compliance. Acute respiratory distress syndrome can result from direct chest trauma, prolonged or profound shock, fat embolism, massive blood transfusion, cardiopulmonary bypass, oxygen toxicity, or acute haemorrhagic pancreatitis. Most of these patients have no previous lung disease. At the onset of ARDS, lung injury may first appear in one lung, but then quickly spreads to affect most of both lungs. When alveoli are damaged, some collapse and lose their ability to receive oxygen. With some alveoli collapsed and others filled by fluid, it becomes difficult for the lungs to absorb oxygen and get rid of carbon dioxide. Within one or two days, progressive interference with gas exchange can bring about respiratory failure requiring mechanical ventilation. As the injury continues over the next several days, the lungs fill with inflammatory cells derived from circulating blood and with regenerating lung tissue. Fibrosis (formation of scar tissue) begins after about 10 days and can become quite extensive by the third week after onset of injury. Excessive fibrosis further interferes with the exchange of oxygen and carbon dioxide.



Phase 6. Pulmonary edema worsens, inflammation leads to fibrosis, and gas exchange is further impeded.

Non-Invasive Ventilation in Acute Respiratory Failure

Acute respiratory failure is defined as an inability to maintain adequate gas exchange. This is divided into:

- Type I: Hypoxia PaO_2 <60mmHg (low levels of oxygen and normal levels of carbon dioxide)
- Type II: Hypoxia with Hypercapnia $PaCO_2$ ≥45mmHg (low levels of oxygen and high levels of carbon dioxide)

The presence of acute hypercapnic respiratory failure induces a respiratory acidosis, where arterial pH is <7.35 and serum HCO_3 is within the normal range. Over time, if CO_2 levels remain high, there will also be an increase in serum HCO_3 levels as the $PaCO_2$ is converted to HCO_3 in the blood stream. This is known as chronic or compensated type II respiratory failure.

Those at particular risk of acute hypercapnic respiratory failure are patients with:

1. COPD
2. Neuromuscular disease
3. Spinal cord injury (T12 and above)
4. Chest wall disorders
5. Morbid obesity
6. Central sleep apnoea
7. Cystic fibrosis
8. Chest or abdominal pain
9. Reduced level of consciousness

NIV versus CPAP

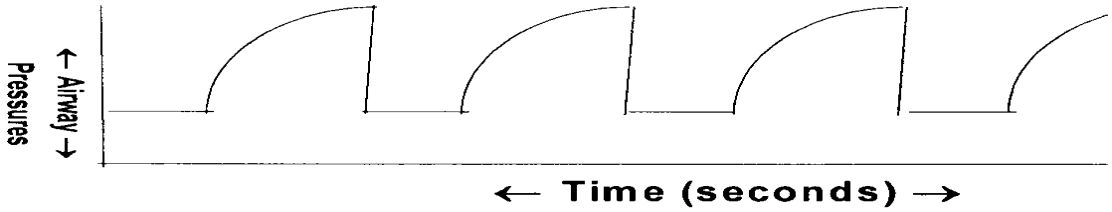
NIV refers to bi-level pressure support without an invasive artificial airway. Although often referred to as BiPAP or VPAP, NIV is now the term used worldwide to describe Non-Invasive Positive Pressure Ventilation (NIPPV).

With NIV, the patient receives both an inspiratory pressure (IPAP) and expiratory pressure (EPAP). The EPAP is constant, whereas the IPAP is a pressure delivered above the EPAP as either a synchronised or timed breath.

IPAP minus EPAP = Pressure Support

In other words, the difference between the two pressures is the amount of support the patient receives for inspiration.

NIV:



Benefits

- ↓ Work of Breathing (WOB) & sensation of breathlessness
- ↑ Tidal Volumes
- ↓ Respiratory Rate
- ↓ Supplemental O_2 requirements
- ↓ $PaCO_2$
- ↓ Incidence of intubation in chronic respiratory disease

Time out

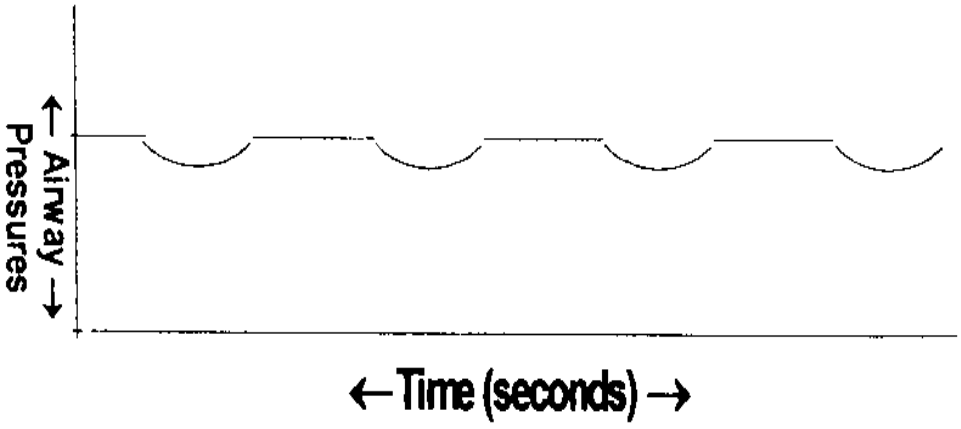
Imagine lifting your arm up and down, up and down to shoulder height. After 5 to 10 minutes you would tire, especially if your arm and shoulder and back muscles were not fit and regularly exercised. After 45 minutes you may find this exhausting. This is what it is like to have an increased work of breathing, where you are using all of your respiratory muscles to force air in and out of your lungs. Now imagine someone came and helped just

by lifting your arm for you. This would still be demanding because your arm would still be moving up and down up and down, however, that assisted lift would provide some much needed relief. That is similar to the support given by NIV to a patient in acute respiratory failure.

Continuous Positive Airway Pressure (CPAP)

This refers to spontaneous ventilation with a positive expiratory pressure being maintained throughout the whole respiratory cycle. CPAP provides positive pressure at the end of exhalation, thus preventing alveolar collapse, improving the functional residual capacity and enhancing oxygenation. The patient must have a reliable ventilatory drive and adequate tidal volume because no mandatory breaths or other ventilatory assistance is given to the patient. Furthermore, the patient performs all the work of breathing (Pierce, 2007)

CPAP:



CPAP Benefits

- Increases Functional Residual Capacity and alveolar recruitment, and reduces shunting, improving PaO_2
- Has a splinting effect for fractured ribs with a flail segment
- In acute cardiogenic pulmonary oedema, reduction of venous return (pre-load), decreases after-load and reduction in transmural LV pressure gradient

NB: CPAP is not recommended in acute type 2 respiratory failure as it increases the effort required to expire, and therefore can significantly worsen PCO_2 levels.

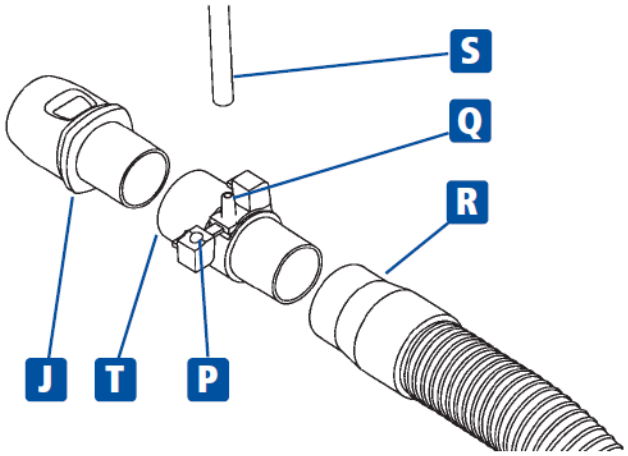
Time out

Think of the lung as a sponge "sucking" blood in (pre-load) and filling up and having to then push the blood out (after load). The lung and thoracic cavity usually has a negative pressure which assists venous blood return from the abdomen and lower limbs to the right side of the heart. The CPAP creates a positive pressure with the chest cavity instead of the usual negative pressure therefore "sucking in" less blood/venous return which leads to less lung filling. At the same time the positive pressure created by the CPAP inside the chest makes it easier for the heart to pump the blood back to the rest of the body (reducing after-load).

CPAP for Obstructive Sleep Apnoea

CPAP is also commonly used for the treatment of Obstructive Sleep Apnoea (OSA) often seen in the community. This works by splinting open the upper airway with continuous positive pressure, which would otherwise collapse, particularly in deep sleep when the muscles throughout the body have very little activity. If admitted to hospital, it is important for the patient to use their CPAP machine, just as they would at home. If required, supplemental oxygen can be added to the CPAP circuit either by using a port on the mask, or the addition of an oxygen connector (see below).

Oxygen connection to a home CPAP/NIV Machine:



Key:			
J	Mask Connection	R	Ventilator Circuit
P	Port Cap	S	Oxygen Tubing
Q	Oxygen Port	T	Oxygen Connector

Indications for NIV

Common:

- Acute Exacerbation of COPD
- Acute Cardiogenic Pulmonary Oedema

Selected patients:

- Post-extubation / weaning (in COPD pts.)
- Community Acquired Pneumonia (in COPD pts.)
- Asthma
- Neuromuscular respiratory failure
- Obesity Hypoventilation Syndrome
- Chest wall disorders
- Cor pulmonale
- Cystic fibrosis
- Immunosuppressed pts. with acute respiratory failure

Contraindications to NIV

- Inability to maintain own airway / GCS < 8
- Marked confusion
- Shock / cardiac instability
- Severe type 1 respiratory failure
    - PaO_2 < 60mmHg on FiO_2 100%
- Facial trauma / burns
- Uncontrolled vomiting
- GI bleeding / obstruction
- Copious respiratory secretions
- Cardio-respiratory arrest
- Any condition requiring immediate intubation
- Pneumothorax → requires ICC first

Initiation of NIV in Acute Hypercapnic Respiratory Failure

1. Initial settings: IPAP 8-12 cmH2O, EPAP 4-6 cmH2O, Rise Time 0.2 sec.

    For patients already established on home NIV: commence at the same pressures, and then titrate as clinically indicated.

2. Increase IPAP in 2 cmH2O increments, until maximum tolerance or targeted tidal volume is achieved.

    A target tidal volume of 6 to 8 mL/Kg (ideal body weight) is aimed for adult patients.

    A target tidal volume of 8 to 10 mL/Kg (ideal body weight) is aimed for COPD patients.

    The maximum IPAP should rarely exceed 23 cmH2O.

3. Minimise FiO2 to keep SpO2 85 - 92%.

4. Adjust mask to minimise leaks whilst being comfortable. A complete seal is not essential.

5. In patients with hypercapnia, increases to EPAP should be undertaken with caution.

NB: Pressure Support = IPAP minus EPAP.

6. Assess clinical response: within the first hour the patient's WOB and respiratory rate should begin to decrease.

7. Perform ABG 1 hour post commencement of NIV to assess physiological response.

NB: If both the respiratory rate and pH have not improved within 1 hour of initiating NIV, the patient will require an urgent ICU review.

Selection of Mask:

	Advantages	Disadvantages
Face	• Best suited for less cooperative patients • Better in patients with a higher severity of illness • Better for mouth-breathing patients • Generally more effective ventilation	• Claustrophobic • Hinder speaking and coughing • Risk of aspiration with emesis

Nasal	• Better in patients with a lower severity of illness • Less claustrophobic • Allows speaking, drinking, coughing, and secretion clearance • Less aspiration risk with emesis • Generally better tolerated	• More leaks possible (e.g. mouth-breathing) • Effectiveness limited in patients with nasal deformities or blocked nasal passages

Assess the clinical response:

- After checking the circuit, gently hold the mask over the patient's face and encourage the patient to breathe using the mask. Secure the mask using the head straps checking for air leaks (alternatively if the patient is able, allow them to hold the mask over their face so they become familiar with it – this often leads to better adherence of the treatment by the patient). Small leaks are acceptable.
- Position the patient at 45º
- Ensure patient is triggering the ventilator and respiratory effort is synchronous with the machine. Adjusting the rise time may be necessary: in general, patients with a high respiratory rate will prefer a shorter rise time (e.g. 0.2 seconds).
- Ensure there is adequate chest wall excursion with each breath and an adequate tidal volume (TV) is delivered.
- Within the first hour the patient should feel less breathless and the respiratory rate should begin to decline.

Assess the physiological response:

- Continuous SpO_2 monitoring needs to be available.
- ABGs should be repeated within 1-2 hours of commencing and stabilising NIV. At this point there should be an improvement in respiratory acidosis (pH rising, even if slightly)
- If ABGs improving they should be repeated at a minimum of 4 hourly intervals until pH is within the normal range. This may vary according to clinical judgement.

Complications:

- Facial/nasal pressure injury
  - Small leaks are acceptable
  - Space breaks at regular intervals

- Gastric inflation
  - Can occur when IPAP > 20 – 25 cmH_2O
  - May require wide bore NGT

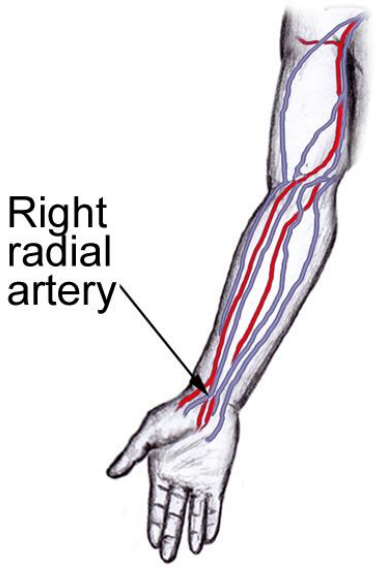
- Dry mucous membranes & thick secretions
  - Humidify circuits in prolonged NIV
  - Regular oral care

- Aspirate pneumonia
  - Anti-emetic therapy
  - Do not use NIV in emesis

Arterial Blood Gases

Arterial blood gas sampling and analysis is a commonly performed procedure within the hospital setting. It is used to assess respiratory, metabolic and renal function.

Sampling typically occurs from the radial artery, although in certain situations it may also be obtained from the femoral or brachial arteries. Intensive care units often use an arterial catheter, which can remain in the artery for a period of up to 7 days and allows for more regular sampling of arterial blood, as-well-as continuous blood pressure monitoring in critically ill patients.



When performed by an appropriately trained clinician, arterial blood sampling is a relatively safe procedure. However, complications can include: pain, syncope, bleeding, haematoma, altered blood flow to the distal limb, and infection. Once the sample has been obtained, it is important to expel any air bubbles within the syringe to minimise gas exchange with atmospheric air prior to analysis.

Measured Values:

Arterial pH, $PaCO_2$, PaO_2 and bicarbonate levels are very sensitive indicators of lung perfusion and ventilation. Normal values are:

Value	Normal Range
pH	7.35 – 7.45
PaO_2	75 – 100 mmHg
$PaCO_2$	35 – 45 mmHg
HCO_3	22 – 28 mmol/L
Base excess	-2 – +2 mEq/L

A pH below normal is called an acidosis (or acidaemia), whereas a pH above normal is termed an alkalosis (or alkalaemia). Derangement of pH can be due to either abnormal metabolic or respiratory function.

As discussed previously, type 2 respiratory failure (T2RF) is defined as a $PaCO_2$ above normal (hypercapnia). This can then be split further into acute, chronic, or acute on chronic type 2 respiratory failure, depending on pH and bicarbonate levels. Here are some typical ABG results for you to compare:

Acute T2RF:	pH	7.31		Low
	PaCO2	55	mmHg	High
	HCO3	26.8	mmHg	Normal
	Base excess	+ 0.9		Normal

Chronic T2RF:	pH	7.38		Normal
	PaCO2	63	mmHg	High
	HCO3	36	mmHg	High
	Base excess	+ 10.5		High
Acute on Chronic T2RF:	pH	7.26		Low
	PaCO2	72	mmHg	High
	HCO3	31.2	mmHg	High
	Base excess	+ 4.4		High

Increased carbon dioxide within the blood is dissolved as carbonic acid in the plasma, which pushes down the pH. As you can see above, acute hypercapnia is displayed with a low pH and normal bicarbonate levels. This is because the body's haemostasis mechanisms have not had time to correct the blood pH yet.

Chronic hypercapnia is displayed with a normal pH, but high bicarbonate levels. This is known as compensated respiratory acidosis, where the body has converted excess levels of CO_2 to bicarbonate, which maintains a normal pH.

In acute on chronic type 2 respiratory failure you will see high bicarbonate levels but low pH. The high bicarbonate indicates chronically raised CO_2, and the acidosis indicates an acute episode.

Venous Blood Gases

ABGs are the gold standard for assessing severity respiratory failure, however obtaining the sample can at times be difficult, as well as painful and distressing for the patient. Venous blood is an acceptable alternative when wanting to assess pH* and trends in PCO_2.

*Venous blood pH is consistently 0.03 below arterial pH (Byrne et al, 2014).

NB: Venous blood is <u>not</u> an accurate indicator of oxygenation.

Nursing Considerations

Historically, NIV was perceived as an intervention implemented by respiratory physicians. (Bolton & Bleetman, 2008). The use of NIV for the clinical management of respiratory failure has been identified as one of the major technological advances in respiratory management in the last decade. (Credland, 2013). As a result, nursing clinicians have been required to substantially increase their knowledge and responsibility of this intervention (Rose & Gerdtz, 2008), and further their understanding of specific nursing considerations that need to be observed.

The delivery of nursing care is determined by the development of an individualised nursing care plan. Underpinning any nursing care plan is the nursing process of assessment, diagnosis, planning, implementation and evaluation. (Alfaro-LeFevre, 2014). The utilisation of the nursing process permits the use of a critical thinking model to support actual and potential needs and risks of any patient. (Gulanick & Myers, 2014).

Whilst it is recognised all nursing care plans are individualised and continually evolving, the following information outlines the specific nursing considerations that are required when providing clinical nursing care to the patient requiring non-invasive ventilation support. This list is not exhaustive; the delivery of holistic nursing care should not be limited by the identified considerations, rather, it should enhance the delivery of nursing care. Supplemental reading is recommended to support the delivery of nursing care and can be accessed here.

1. GNAH_0522_Management_NIV_non_critical_care_units (JHH local guidelines)
2. HNELHD_Pol_14_06_Minimum_Standards_of_Patient_Care_for_Adult_Inpatients

Potential Complications of NIV	Nursing Consideration
Pressure ulceration to bridge of nose or above ears due to tight fitting mask and strapping.	• Consider alternative mask styles and securing devices • Use of customised foam on the bridge of the nose • Pressure area care is to be initiated as per Pressure Injuries: Prediction, Prevention and Management PD 2005_257:PCP1

Corneal irritation and conjunctivitis to eyes due to leakage of high flow medical gas	• Readjustment of mask to decrease leakage of medical gas to eyes • Eye care is to be performed by nursing staff for all inpatients as per HNELHD_Pol_14_06_Minimum_Standards_of_Patient_Care_for_Adult_Inpatients in conjunction with medical officer review and adherence to HNELHD_CP_14_37_Administration_of_Eye_drops_Ointmentand_Eye_Cleansing
Nasal congestion/dryness/ soreness due to ill-fitting mask	• Nasal care to be performed by nursing staff as per HNELHD_Pol_14_06_Minimum_Standards_of_Patient_Care_for_Adult_Inpatients. • Consider change of mask to achieve a small leak of medical gas. • Consider nasal pharmacological therapy to manage nasal symptoms • Use of humidification of the NIV circuit
Dry mouth/irritation of the buccal mucosa related to ill-fitting masks and/or high flow medical gas	• Oral hygiene to be performed by nursing staff as per HNELHD_Pol_14_06_Minimum_Standards_of_Patient_Care_for_Adult_Inpatients. • Promote an adequate fluid intake to stimulate the flow of saliva. Intravenous fluids may be considered. • Promote a nutritious diet that provides the tissues with the nutrients necessary for growth and repair • Consider humidification
Gastric Distention/air insufflation into the stomach related to high flow levels of medical gas	• Monitoring abdominal distention when NIV insitu • Assessment of the patients pain using subjective and objective data and recording on Standard Adult General Observation (SAGO) Chart • In consultation with Respiratory physician, analysis of arterial blood gases (ABG), consider decreasing inspiratory pressure, as to not compromise the patient • Consider a wide bore nasogastric tube

Claustrophobia associated with NIV interface, potentially causing agitation	• Nursing with 1:1 ratio whilst patient agitation is present, to promote efficacy and coordination of respiratory cycling. Refer to GNAH_0038_Models_of_Care_Pts_requiring_additional_supervision • Provide patient emotional support with mask placement and tolerance to promote positive patient treatment outcome
Poor inflation of the chest	• Reposition the patient, sit upright and promote lung expansion. Consider the use of a soft collar or towel to provide support to the head, as to not allow the chin to fall to the chest. • Consider bronchospasm, mucus plugging, pneumothorax, atelectasis/collapse , consolidation, pulmonary oedema or kinking/obstruction of NIV circuit tubing • Confirm diagnosis through x-ray in consultation with respiratory physician • Referral to physiotherapist for chest percussion
Inability to call for help	• Nursing with 1:1 ratio in the acute phase of implementation. Refer to GNAH_0038_Models_of_Care_Pts_requiring_additional_supervision • Ensure the nurse call bell is with patient and instruction has been given on use as per HNELHD_Pol_14_06_Minimum_Standards_of_Patient_Care_for_Adult_Inpatients. • Provide education to the co-operative and conscious patient on release of mask in the event of vomiting or expectoration of secretions

Theoretical Assessment

Name:	Employee Number:
Role:	Place of Work:

Section 1:

1. List the 5 components of gas exchange:

_____ _____

_____ _____

_____

2. In normal breathing, what is the ratio of inspiration to expiration?

_____

3. Explain what “tidal volume” means:

_____

_____

4. In order for effective gas exchange to occur in the alveoli, there needs to be effective: (circle one)

Temperature & Humidity Gas Mixing Ventilation & Perfusion

5. What affects gas diffusion in the alveoli? (List 4):

_____ _____

_____ _____

6. The speed of oxygen and carbon dioxide diffusion across the alveolar membrane is affected by what?

_____

_____

7. At what SaO2% do symptoms of hypoxia normally begin?

_____

8. Name 3 factors which affect the dissociation of oxygen from haemoglobin?

_____ _____

_____

9. How is the majority of carbon dioxide transported in the venous circulation from the tissues to the lungs?

_____

_____

10. Where is the respiratory centre found?

_____

11. What is the main stimulus to increase a person's ventilation? (Circle one)

↓Oxygen ↓Carbon Dioxide ↑Carbon Dioxide ↑Anxiety

12. In situations of chronic hypercapnia, what then becomes the main stimulus to increase a person's ventilation? (Circle one)

↓Oxygen ↓Carbon Dioxide ↑Carbon Dioxide ↑Anxiety

13. In your own words, describe "*ventilation impairment",* including what you would expect to see in regards to arterial carbon dioxide levels:

_____

_____

_____

_____

14. Name 3 common respiratory disorders which can cause hypoxia:

_____

_____

15. Name 3 common respiratory disorders which can cause hypercapnia:

_____

_____

Section 2:

1. Type 1 Respiratory Failure is defined as:

_____

_____

2. Type 2 Respiratory Failure is defined as:

_____

_____

3. In patients at risk of hypercapnic respiratory failure, what SaO2 is typically aimed for?

_____

4. What is the main difference between CPAP and NIV? (Circle one)

CPAP gives only EPAP

NIV gives Pressure Support

NIV can deliver a Timed Breath

All of the above

5. List 5 benefits of NIV:

_____ _____

_____ _____

_____

6. List 5 indications for NIV:

_____ _____

_____ _____

_____

7. List 5 contraindications to NIV:

_____ _____

_____ _____

_____

8. What would you expect to see within 1-2 hrs of initiating NIV? (Circle one):

↓Respiratory Rate ↑pH ↓$PaCO_2$ All of these

9. If a patient receiving NIV becomes drowsy and has reduced oxygen saturations, what would you do?

_____

_____

_____

10. If a non-invasive ventilator has settings of:

IPAP 20 cmH_2O, EPAP 5 cmH_2O, Rate 14 bpm, FiO2 .28

What Pressure Support will the patient receive?

_____

11. If a patient has tidal volumes (VT) of 600 mL and a respiratory rate (RR) of 18 breaths per minute, what is their minute ventilation (VE)?

_____

12. What type of NIV mask would you select in a patient who has a history of vomiting despite anti-emetics?

_____

13. In acute hypercapnic respiratory failure, would you expect to see a high or low pH?

_____

14. In chronic (compensated) respiratory failure, would you expect to see high or low bicarbonate levels?

_____

15. True or False: Venous PO_2 is an accurate indicator of arterial oxygenation.

_____

Total Score:

Practical Assessment

All of the following elements must be achieved in order to pass the practical assessment:

Essential Elements	Yes	No
1. Discusses the indications for NIV		
2. Discusses the contraindications and limitations to NIV		
3. Identifies when active humidification of the NIV circuit is required		
4. Selects mask appropriate to identified patient		
5. Installs the NIV circuit & mask ready for patient use		
6. Enters settings appropriate for identified patient		
7. Places patient on NIV, with particular focus to mask fitting		
8. Adjusts alarm parameters to appropriate limits		
9. Discusses level and frequency of observations required		
10. Discusses nursing responsibilities of patients on continuous NIV		
11. Identifies adjuncts to NIV (patient position etc.)		
12. Identifies signs and symptoms of acute deterioration of patient on NIV or when an escalation in attention is required		

To be signed once both the theoretical and practical assessments have been completed, and the learner is deemed competent:

Name & Signature of Assessor	Designation	Name & Signature of Learner	Date

References

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Reflection tool

At the completion of the SDLP we have added a reflection form that will assist staff in reflecting on the package and how it meets their professional development needs and can be added to your professional portfolio.

Evaluation

An evaluation form is included at the conclusion of this SDLP for completion by the learner. All forms of feedback are appreciated and assist in further development of the program. Completed evaluation forms should be returned to the author, Nick Yates, CNC Respiratory Failure & Non-Invasive Ventilation, Department of Respiratory and Sleep medicine, John Hunter Hospital.

REFLECTION ON LEARNING

Date: Self-directed learning package:

Amount of time spent completing SDLP:

What are the RN, EN or speciality competency standard (s) this SDLP meets?

_____

_____

_____

_____

Please outline the key learning you obtained:

_____

_____

_____

_____

Was the learning useful for your practice? Please explain?

_____

_____

_____

_____

How will the learning influence your practice?

_____

_____

_____

_____

Is there further information you need to obtain?

_____

_____

_____

_____

Signature Date

Learning Package Evaluation Form

Return to: Nick Yates, CNC Respiratory Failure & Non-Invasive Ventilation

Department of Respiratory and Sleep Medicine, John Hunter Hospital

Please circle your response to the following questions:

1. The aims and objectives of the learning package were clear and appropriate to your learning needs and goals? Yes No

2. I have achieved my learning goals? Yes No

3. As a result of completing this package I now have a better understanding of NIV Yes No

4. The activities and case scenarios were helpful? Yes No

5. The package was easy to follow? Yes No

6. The workload was reasonable? Yes No

7. The information and skills I can use from the package are:

_____

_____

_____

_____

8. Some suggestions I would like to make to improve the package are:

_____

_____

_____

_____

9. Further comments I would like to make are:

_____

_____

_____

_____