Learning Package

Health
Hunter New England
Local Health District

Non-Invasive Ventilation (NIV): Learning Package

Sites where Learning package applies: All inpatient facilities where patients receive Non Invasive Ventilation (NIV)

Target audience: Nurses caring for patients receiving NIV

Description: This package provides an introduction to the concepts and principles Non-Invasive Positive Pressure Ventilation (NIPPV).

Learning Outcomes, On completion of this package you will be better able to:

Completion of this package is the first step towards developing competence in managing NIV. Competence refers to the combination of skills, knowledge, attitude, values and abilities that underpin effective and/or superior performance related in this case to management of NIV of Registered and Enrolled Nurses.

Following completion of this learning package the participant will be able to:

- *Define what is Non Invasive Ventilation (NIV)*
- *Identify indications and contraindications for NIV*
- *Define types of acute & chronic respiratory failure*
    - *Type 1 and Type 2*
- *Describe the benefits of NIV for acute and chronic respiratory failure*
- *Describe the nursing role in providing care to a patient with NIV including ventilator and mask management*
- *Outline complications of NIV and nursing considerations*
- *Identify indications of NIV treatment failure*

Keywords: Non Invasive ventilation (NIV), Continuous Positive Airway Pressure (CPAP), Oxygenation, Ventilation, Acute Respiratory Failure, Chronic Respiratory Failure, Chronic Obstructive Pulmonary Disease (COPD), Acute Respiratory Distress Syndrome (ARDS), Nursing Care

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Replaces existing document? No

Related Legislation, Australian Standard, NSW Ministry of Health Policy Directive or Guideline, National Safety and Quality Health Service Standard (NSQHSS) and/or other, HNE Health Document, Professional Guideline, Code of Practice or Ethics:

- List related documents here OR, if there are too many to fit on the front page:
- "See Reference Section on page …." And add list to the Reference section

Learning package contact person

Contact details: Nick Yates nick.yates@hnehealth.nsw.gov.au

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Non-Invasive Ventilation (NIV) Learning Package

Department of Respiratory & Sleep Medicine
John Hunter Hospital

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Hunter New England Local Health District

Author/s:

Nick Yates CNC Respiratory Failure & Non-Invasive Ventilation
John Hunter Hospital

Peer reviewed by:

Lia Heffernan: CNE Division of Medicine
John Hunter Hospital

External review by:

Leila Kuzmiuk: NE ICU, John Hunter Hospital

Instructional design and editing by

Felicity Williams: Nurse Manager Professional Development, HNELHD

Catherine Rutledge: HSM OCL eLearning and Curriculum Governance HNELHD

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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.

Acknowledgements:

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Kim Wild - NUM Respiratory Medicine
John Hunter Hospital (2010)

Prof Peter Wark - Senior Staff Specialist Respiratory & Sleep Medicine
John Hunter Hospital

Traci Wellington - Clinical Educator Mayo Healthcare Education Service
Mayo Healthcare Australia

Contact details:

Nick Yates - John Hunter Hospital
nick.yates@hnehealth.nsw.gov.au

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Introduction

This package provides an introduction to the concepts and principles Non-Invasive Positive Pressure Ventilation (NIPPV).

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 hypercapnic respiratory failure in many patients, and may have a role for some patients with heart failure. Compared with invasive ventilation, 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 exacerbations of chronic type II respiratory failure is now viewed as standard practice, and is associated with decrease patient morbidity, mortality and hospital length of stay.

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

HNELHD_Pol_14_06_Minimum_Standards_of_Patient_Care_for_Adult_Inpatients

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. Whilst 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 applying the content into practice, if being utilised by another unit, context or organisation.

Naming Convention

Respiratory Medicine: Non-Invasive Ventilation

Aim

The aim of this package is to provide the theoretical knowledge that underpins the achievement of competence related to the management of NIV.

Learning Outcomes

Completion of this package is the first step towards developing competence in managing NIV. Competence refers to the combination of skills, knowledge, attitude, values and abilities that underpin effective and/or superior performance related in this case to management of NIV of Registered and Enrolled Nurses.

Following completion of this learning package the participant will be able to:

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Pre-requisites

There is no prior learning required to undertake this package. It is assumed the participant will have current knowledge of the respiratory system. Full benefits of completing the package will be attained by working in a relevant clinical area.

Learning Package Outline

The package is designed to guide you through the literature and clinical issues related to NIV.

On completion and submission of this learning package a record of your completion will be added to your learning record in HETI online. You will be credited with 2 CPD hours.

To be assessed as competent (Step 2) you will be required to undertake a skills assessment performed by a clinician skilled and knowledgeable in NIV, using a relevant assessment tool. This enables knowledge to become translated to practice at an agreed standard.

How to use this resource or Instructions for participants

- 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, these may need to be contextualised in your local clinical setting.
- The readings enhance the learning and are therefore recommended. These can be accessed online (journal articles) through CIAP. If you experience difficulty locating these readings please seek assistance from your hospital / health facility library.

Assessment process

- You are required to complete the assessment of knowledge to finalise Step 1 of the competency process. Completion of this section will be recorded in your HETI online learning history under the title
- The package can be returned to a Nurse Educator/CNE/CNC with the relevant knowledge and skills in NIV/ respiratory nursing who will mark it and provide you with feedback.
- After the package has been marked you will need to have a practical assessment of competence. This finalises the assessment process through translation of knowledge to skills. Completion of the practical assessment will be separately recorded in your HETI online learning record, with Step 1 as a pre-requisite.

Reflection tool

At the completion of the learning package there is a tool for you to reflect on the learning. This guides your reflection on how the package meets your professional development needs, and how might you apply the learnings into practice. It is a useful document to include in your professional portfolio.

Evaluation

A form is included at the end of the Learning Package for completion by the learner. All feedback is appreciated and assists in development of a quality program. Return to: Nick Yates CNC Respiratory Failure & Non-Invasive Ventilation, Department of Respiratory and Sleep Medicine, John Hunter Hospital or nick.yates@hnehealth.nsw.gov.au

SECTION 1:

Learning outcomes related to this section

- Define what is Non Invasive Ventilation (NIV)
- Identify indications and contraindications for NIV

What is NIV?

NIV refers to bi-level pressure support without an invasive artificial airway.

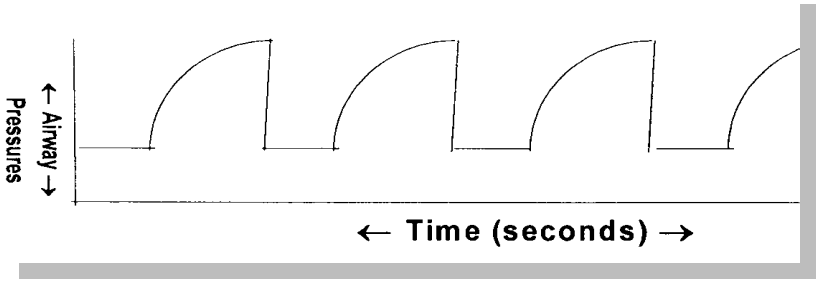
NIV is now used as the term to describe Non-Invasive Positive Pressure Ventilation (NIPPV) or BiPAP.

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.

How NIV pressure works over time



How does NIV support the patient?

- ↓ Work of Breathing (WOB) & sensation of breathlessness
- ↑ Tidal Volumes
- ↓ Respiratory Rate
- ↓ Supplemental O_2 requirements
- ↓ PCO_2
- ↓ incidence of intubation in chronic respiratory disease

Activity 1:

Imagine someone was paying you to lift your arm up and down, up and down just lifting it 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.

Use the following diagram to reflect on how muscle fatigue impacts on the respiratory system, and associated factors.

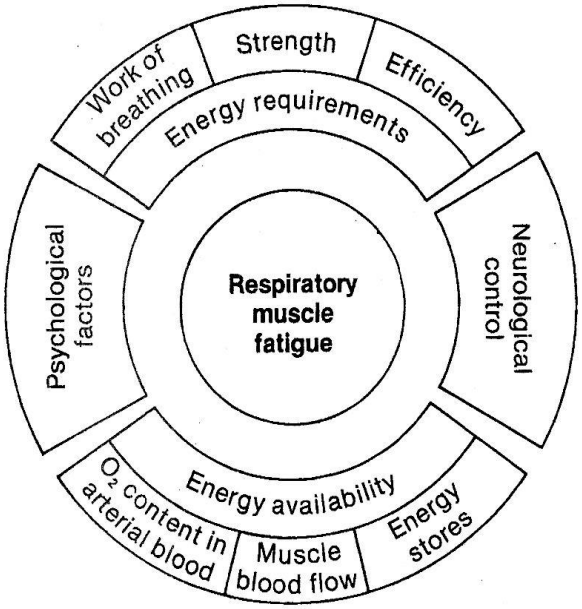


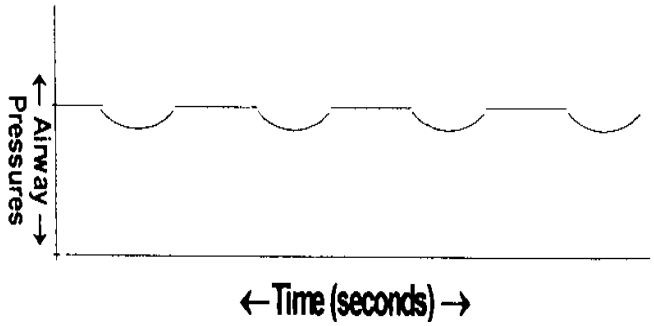
Figure 2-23 Factors associated with respiratory muscle fatigue.
(From Shaffer TH, Wolfson MR, Bhutani VK: Phys Ther 61:12, 1981.)

Continuous Positive Airway Pressure (CPAP)

This refers to spontaneous ventilation with a *positive airway pressure* being maintained throughout the whole respiratory cycle. CPAP provides positive pressure at end-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).

How CPAP pressure works over time



CPAP Benefits

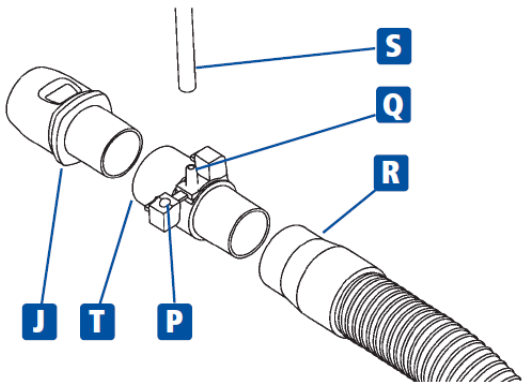
- Increases Functional Residual Capacity and alveolar recruitment, and reduces shunting, improving PaO_2
- Has a splinting effect for fractured ribs with a flail segment
- In cardiogenic acute pulmonary oedema, reduction of venous return (pre-load), decreases after-load and reduction in transmural LV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

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

CPAP is 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 when the individual approaches the relaxed state of deep sleep.

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

Activity 2:

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).

INDICATIONS AND CONTRAINDICATIONS FOR NIV	
Indications	**Contraindications**
• Acute exacerbation of COPD with respiratory acidosis (pH<7.35 & $PaCO_2$ >45mmHg) • Acute Cardiogenic Pulmonary Oedema • Post-extubation in patients with chronic type 2 respiratory failure • Neuromuscular disorder with ventilatory failure • Spinal cord injury (C5 and above) • Diaphragmatic paralysis/dysfunction • Obesity hypoventilation syndrome • Severe central sleep apnoea • Obstructive sleep apnoea with severe COPD • Chest wall disorder • Cystic fibrosis • Immunosuppressed patients with acute respiratory failure • Acute respiratory failure in selected "not for intubation" patients	• Life Threatening type1 respiratory failure ▪ PaO_2 < 60mmHg on FiO_2 100% • Condition requiring immediate intubation • Cardio-respiratory arrest • Copious respiratory secretions • Inability to maintain own airway • GCS <8 / marked confusion • Shock / cardiac instability • Facial trauma / burns / surgery • Uncontrolled vomiting • GI bleeding / obstruction • Pneumothorax (untreated)

SECTION 2:

Learning outcomes related to this section:

- *Define types of acute & chronic respiratory failure*
  - *Type 1 and Type 2*

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 oxygen's 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 supplemental oxygen to increase the concentration gradient, and therefore increase oxygen diffusion. The aim is to prevent tissue hypoxia whilst treating the cause, which maybe by increasing alveolar surface area, reducing alveolar/interstitial fluid or improving ventilation-perfusion mismatch.

Ventilation impairment

Ventilation impairment, or inadequate exchange of gas through the airways between the atmosphere and the alveoli, is characterised by high arterial carbon dioxide levels, respiratory acidosis (acute and/or compensated) and increased work of breathing. Inadequate spontaneous ventilation may be caused by:

- a reduced drive to breathe, for example CNS depression
- a reduction in tidal volumes, for example neuromuscular disorders
- a conducting airways disease, for example COPD

Carbon dioxide excretion is dependent on the amount of air it can exchange with. Any change to the respiratory rate or tidal volume, and therefore the minute ventilation, will affect the arterial carbon dioxide levels. Treatment of ventilation impairment focuses on increasing the minute ventilation (tidal volume X respiratory rate) by either: improving tidal volumes by supporting the inspiratory effort, and/or increasing the respiratory rate by minimising any central respiratory depressants.

Acute and Chronic Respiratory Failure

Respiratory failure is a syndrome in which the respiratory system fails in one or both of its gas exchange functions: oxygen uptake and carbon dioxide elimination.

Respiratory failure may be acute or chronic, with acute respiratory failure being a common indication for invasive or non-invasive ventilation. Acute respiratory failure is characterised by life-threatening alterations in arterial blood gases and acid-base status and is defined as an inability to maintain adequate gas exchange.

Common causes of acute hypoxic respiratory failure include pneumonia, pulmonary oedema, pulmonary embolism, cardiac failure, CNS depression, chest trauma and pneumothorax.

Manifestations of chronic respiratory failure may not be as readily apparent.

Respiratory failure is defined as:

- **Type I:** Hypoxia PaO_2 <60mmHg
  (low levels of oxygen without raised carbon dioxide)

- **Type 2:** Hypoxia with hypercapnia $PaCO_2$ ≥45mmHg
  (low levels of oxygen with increased levels of carbon dioxide)

Some texts use a definition of CO_2 >50mmHg as the defining number for a diagnosis of type two respiratory failure. However, here hypercapnia is defined as CO_2 >45mmHg, as patients with a CO2 > 45mmHg may deteriorate rapidly in the acute situation.

Risk Factors
Those at particular risk of acute hypercapnic respiratory failure
• COPD • Chest wall deformity • Morbid obesity • Neuromuscular disease • Cystic Fibrosis

Common Respiratory Disorders

Atelectasis

Atelectasis refers to the collapse of an area of lung.
Atelectasis may result from:

- Airway obstruction (for example 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 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 (from a 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 approximately 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.

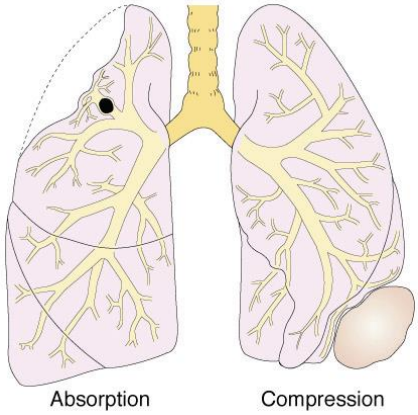


Diagram 2.1: provides an example of how atelectasis affects the lungs.

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.

Increased 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 in water.
- 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) which results in an increased amount of pulmonary blood and pressure, causing fluid to leak out of the capillaries into the interstitial airways.

Non-cardiogenic APO can be caused by sepsis, renal failure and acute respiratory distress syndrome (ARDS).

Pneumonia:

This is an inflammatory process of the alveoli, often caused by bacteria or viral infection. As a result, fluid accumulates in the alveoli, reducing oxygen diffusion as well as diluting the surfactant causing collapse of the alveoli. Management includes treating the cause of infection, clearance of secretions, and supportive therapies such as supplemental oxygen to maintain adequate blood oxygen levels.

In severe pneumonia, where adequate oxygenation of the blood is not achieved with supplemental oxygen alone, intubation and mechanical ventilation may be required.

Chronic Obstructive Pulmonary Disease (COPD)

Prolonged and repeated irritation of the airways and alveoli within the lungs by noxious stimuli can lead to emphysema or chronic bronchitis. A combination of the two is known as COPD. Tobacco smoke is the leading cause of this within Australia, however, in lesser developed countries COPD can be caused by indoor stoves and heating without adequate ventilation.

The following information provides an overview of these conditions and how they affect the lungs and oxygenation.

- **Chronic Bronchitis**

  Irritants reaching the bronchi and bronchioles will stimulate an inflammatory response leading to increased secretion of mucus. In chronic bronchitis, the airways narrow with inflammation, thickening and increased mucous secretions leading to a persistent cough. Reduced oxygenation is caused by impaired ventilation and reduced alveolar gas exchange due to secretion filled airways and alveoli.

- **Emphysema**

  Emphysema is where the delicate walls of the alveoli to 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.

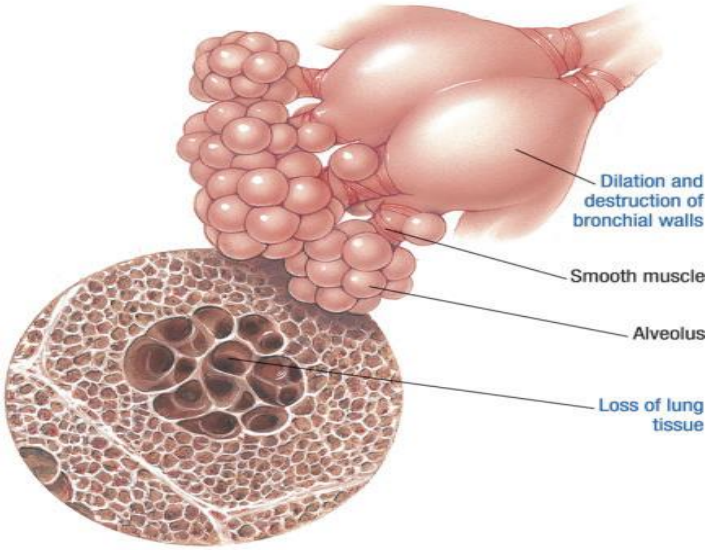


Diagram 2.3: Emphysema

The condition develops slowly and is seldom a direct cause of death. Gradual loss of gas exchange area forces the heart to pump ever-larger volumes of blood to the lungs to compensate for the hypoxia.

This can lead to right sided heart failure.

Acute Respiratory Distress Syndrome (ARDS)

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.

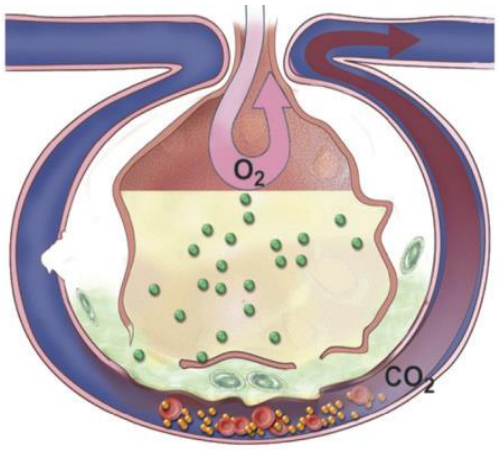
Acute respiratory distress syndrome can result from direct chest trauma, prolonged or profound shock, fat embolism, massive blood transfusion, cardiopulmonary bypass, oxygen toxicity, or acute haemorrhagic pancreatitis. Most of these patients have no previous lung disease.
At the onset of ARDS, lung 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.

Illustration 2.4: an example of ARDS

As the injury continues, the lungs fill with inflammatory cells 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.



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

SECTION 3:

Learning outcomes for this section are:

- *Describe the benefits of NIV for acute and chronic respiratory failure*

NIV for Type 2 Respiratory Failure

- **Type 2:** Hypoxia with hypercapnia $PaCO_2$ ≥45mmHg
  (Low levels of oxygen and high levels of carbon dioxide)

Some texts use a definition of CO_2 >50mmHg as the defining number for a diagnosis of type two respiratory failure. However here hypercapnia is defined as CO_2 >45mmHg, as patients with a CO2 > 45mmHg may deteriorate rapidly in the acute situation.

Hypercapnic respiratory failure is an independent risk factor for death and the need for intubation and mechanical ventilation. Individuals who require invasive ventilation may have prolonged ICU admissions and a mortality rate of 20-33% (Breen et al, 2002).

The presence of acutely raised carbon dioxide drops the pH of the blood. This is termed a respiratory acidosis, where arterial pH is <7.35 and serum bicarbonate is within the normal range.

In the presence of a functioning renal system, if the blood CO_2 levels remain high, the kidneys will hold on to bicarbonate, and over approximately 48 hours the blood pH will return to normal with serum bicarbonate levels increasing. This is known as compensated type II respiratory failure: raised PCO_2, normal pH, raised bicarbonate.

All individuals who present with acute dyspnoea and chronic lung disease should be assessed for the presence of respiratory failure using arterial blood gas (ABG) analysis.

ABGs need to be assessed by a clinician skilled in the interpreting of blood gases or discussed with an on call respiratory physician or equivalent.

Initial treatment of acute hypercapnic respiratory failure includes:

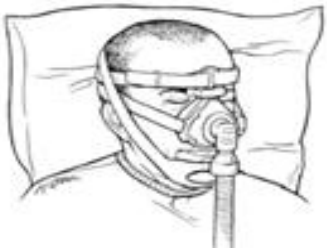
1. Rational use of oxygen. Aim to correct hypoxaemia but not induce CO_2 retention. In those at risk of hypercapnic respiratory failure aim for SpO_2 88 – 92%.
2. Administer inhaled bronchodilators and systemic corticosteroids in airways disease.
3. Position the patient to assist with chest wall expansion.
4. Reverse the effects of CNS depressants.

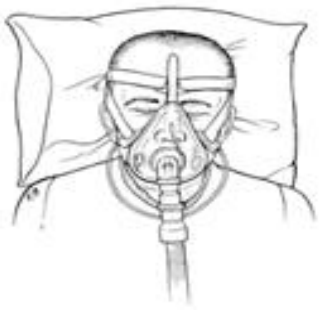
An ABG should be sampled and reassessed 1 hour after implementing the above treatment. If there is no improvement, NIV or invasive ventilation should be considered.

The decision to initiate NIV is made using a combination of the clinical assessment and ABGs. In the case of severe acute exacerbation of COPD, a pH of <7.35 and $PaCO_2$ >45mmHg is a clinical indication for NIV. Prior to commencement the contraindications for NIV will need to be reviewed.
All patients receiving NIV are to have a documented plan of care. This is developed at the commencement and reviewed at least every 24hours or on change of condition.
The plan is to be developed by a critical care or respiratory physician or a qualified proxy.

Mask selection:

	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 (mouth-breathing) • Effectiveness limited in patients with nasal deformities or blocked nasal passages





Wearing the mask
Fit and comfort

Initiation and Titration of NIV in Acute Hypercapnic Respiratory Failure

- Commence at: IPAP 10 cmH_2O, EPAP 5 cmH_2O.
  If on outpatient NIV, commence at same pressures as home device.
- Increase IPAP upwards as tolerated (max 23 cmH_2O).
  A target tidal volume of 8 - 10 mL/Kg (ideal body weight) is aimed for COPD & OHS patients.
  NB:IPAP minus EPAP = Pressure Support
- Adjust rise time to syncronise with patient's inspiratory demand.
- Minimise FiO2 to maintain SpO_2 of 88 - 92%.
  Initially there may be a fall in SpO_2 following commencement of NIV.
- Minimise mask leak whilst preserving comfort.
- Within the first hour, the patient's respiratory rate and PCO_2 should begin to decline.
- Consider intubation if respiratory acidosis worsens despite optimal NIV.

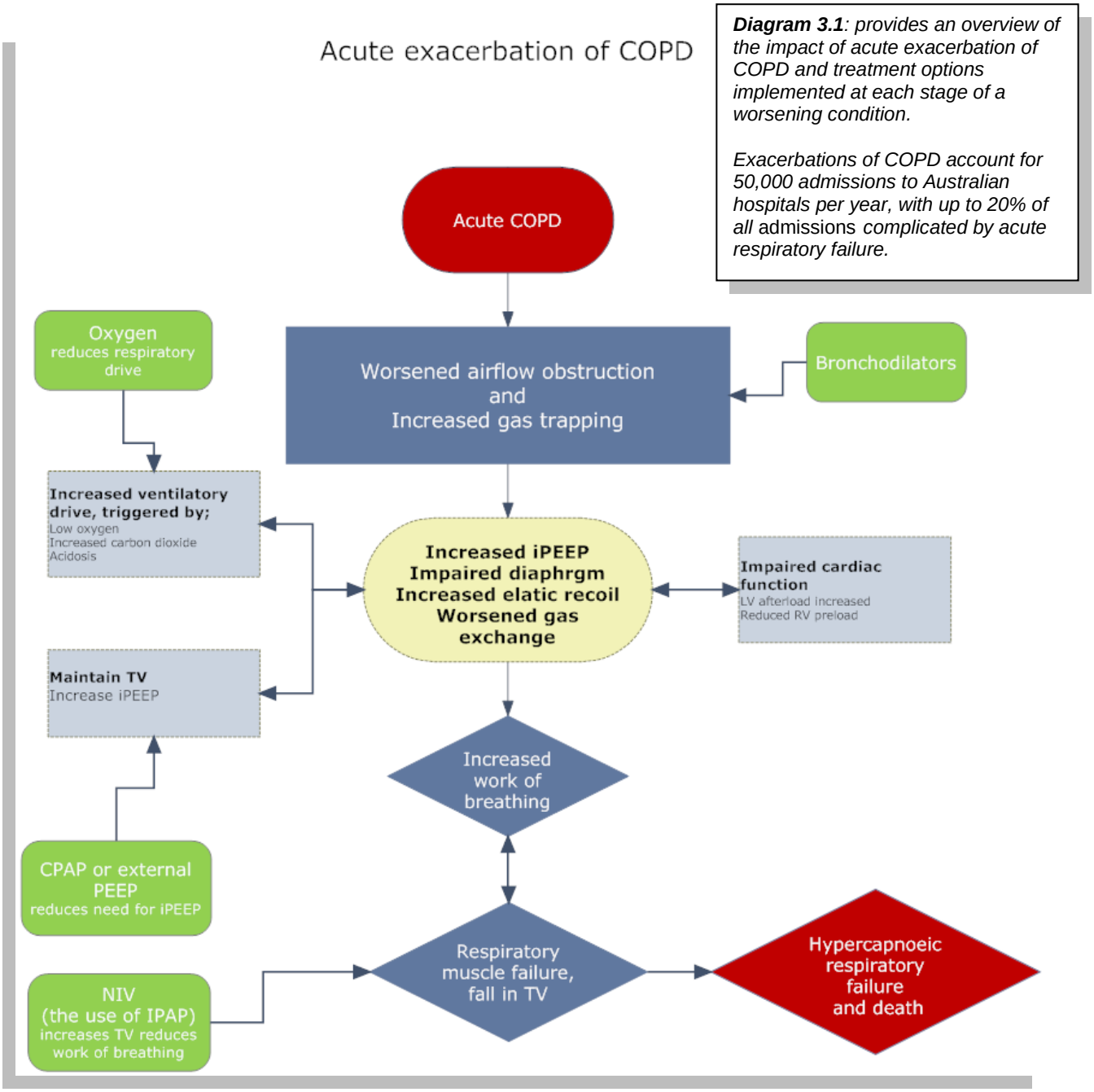


***Diagram 3.1**: provides an overview of the impact of acute exacerbation of COPD and treatment options implemented at each stage of a worsening condition.*

Exacerbations of COPD account for 50,000 admissions to Australian hospitals per year, with up to 20% of all admissions *complicated by acute respiratory failure.*

Reflection on your learning:
Given the following benefits of using NIV for acute exacerbation of COPD:

- They are the patient group most likely to benefit from NIV
- NIV is most effective in moderate to severe disease
- NIV is recommended as 1st line therapy in COPD patients with acute hypercapnic respiratory failure (pH < 7.35)
- Reduces:
  - Need for intubation
  - Mortality rates
  - Complications
  - Length of Stay

Think about the personal impact of an admission to ICU for a person with an acute exacerbation of COPD and their family?
How might NIV prevent this?

SECTION 4:

Learning outcome for this section:

- *Describe the nursing role in providing care to a patient with NIV including ventilator and mask management*
- *Outline complications of NIV and nursing considerations*
- *Identify indications of NIV treatment failure*

Assess the clinical response

- Checking the circuit and gently hold the mask over the patients face (if the patient can hold the mask over their face– it can lead to better adherence with the treatment).
- Encourage the patient to breathe using the mask. Secure the mask using the head straps checking for air leaks
- Position the patient at ≥45º
- Ensure patient is triggering the ventilator and respiratory effort is synchronous with the machine
- Ensure there is adequate chest wall excursion with each breath and an adequate tidal volume (VT) is delivered. As a guide a VT of 8-10ml/kg ideal body weight is ideal (see appendix 2).
- Adjust pressure settings to ensure the machine is adequately triggering, there is good chest wall excursion and to reduce dyspnoea
- Within the first hour the patient should feel less breathless and the respiratory rate should begin to decline

Nursing Care

Historically, NIV was an intervention implemented by respiratory physicians (Bolton and Bleetman, 2008) and has been identified as one of the major technological advances in respiratory medicine for the last decade (Credland, 2013).
Its increased use has meant nurses knowledge and responsibility of NIV has been required to increase (Rose & Gerdtz, 2008) to further the understanding of nursing considerations and observations.
Nursing care is determined by the development of an individualised nursing care plan which is underpinned by the nursing process of assessment, diagnosis, planning, implementation and evaluation (Alfaro-LeFevre, 2014). The nursing process permits the use of a critical thinking model to address actual and potential risks of patients (Gulanick & Myers, 2014).
The following information outlines specific nursing considerations required when providing nursing care to the patient requiring NIV. This list is not exhaustive and should enhance the delivery of individualised compassionate care.

Nursing Responsibilities: Assess the physiological response

Ongoing Observations	
Repeat ABGs	• After 1 hour of therapy and 1 hour after subsequent changes in settings • After 4 hours or earlier in patients who are not improving clinically
Frequent clinical monitoring of acutely ill patients	• Every 15 mins in the first hour • Every 30 mins in the 1-4 hourly period • Then hourly
Observations to include	• RR, continuous pulse oximetry, HR, BP, AVPU • Pain Score • Chest wall movement, ventilator synchrony, accessory muscle use. • Patient Comfort, or pain • Daily skin integrity check including skin integrity under the interface.

- Continuous SaO_2 monitoring is to be undertaken.
- ABGs should be repeated within 1-2 hours of commencing and stabilising NIV. An improvement in respiratory acidosis (pH rising, even if slightly).
- If ABGs are improving they should be repeated at a minimum of 4hrly intervals until pH is within the normal range. This may vary according to clinical judgement.

Complications of NIV Nursing Considerations		
Complication	**Reason**	**Intervention**
Facial/nasal pressure injury	Device related pressure injury due to pressure and/or shearing forces of the interface against the skin.	◦ Allow some leak. ◦ Considering alterative mask styles and securing. ◦ Space breaks at regular intervals, to relieve pressure ◦ Check skin integrity when relieving pressure and daily. ◦ Apply pressure redistributing foam to skin to reduce shearing.
Corneal irritation and conjunctivitis to eyes	Due to leakage of high flow medical gas	◦ Readjustment of mask to decrease leakage to eyes ◦ Regular eye care by nursing staff
Dry nasal/ mucous membranes, congestion	Increased flow of gas through mouth and nose	◦ Humidify circuits in prolonged NIV (>24 hours) ◦ Regular oral and nasal care ◦ Consider intra-nasal medication to manage symptoms.
Dry mouth/irritation of the buccal mucosa	Related to ill-fitting masks and/or high flow medical gas	◦ Regular oral hygiene ◦ Promote adequate fluid intake to stimulate flow of saliva ◦ Consider intravenous fluids ◦ Promote a nutritious diet to enable tissue growth and repair ◦ Consider humidification
Gastric Distention/air insufflation into the stomach	Can occur when IPAP >22cmH_2O	◦ Monitor abdominal distention during NIV ◦ Assess patients pain and record on SAGO Chart ◦ Analyse ABGs and consider decreasing inspiratory pressure, where applicable in consultation with physician ◦ Consider a wide bore nasogastric tube
Aspirate pneumonia		◦ Do not use NIV during episodes of emesis ◦ Provision of anti-emetic therapy
Claustrophobia	NIV interface potentially causing agitation	◦ Nurse with 1:1 ratio whilst patient agitation is present, to promote efficacy and coordination of respiratory cycling. ◦ Provide emotional support with mask placement and tolerance
Poor inflation of the chest	Due to poor posture whilst receiving NIV	◦ Reposition patient, sit upright and promote lung expansion. ◦ Consider a soft collar or towel to support the head and prevent the chin to fall to the chest. ◦ Investigate presence of bronchospasm, mucus plugging, pneumothorax, atelectasis/collapse, consolidation, pulmonary oedema or kinking/obstruction of NIV circuit tubing. ◦ Diagnose via x-ray in consult with physician ◦ Refer to physiotherapist for chest percussion
Inability to call for help	Due to mask	◦ Nurse with 1:1 ratio during acute implementation. ◦ Provide call bell to patient with instructions. ◦ Provide education to the patient on release of mask in the event of vomiting or expectoration of secretions

Predicting Success or failure of NIV (within 1-2hrs of commencement)	
Success	**Failure**
• ↓ $PaCO_2$ • ↑ pH (gradually increasing to normal values)	• pH<7.25 • PaCO2 > 80mmHg • GCS < 8 or Marked Confusion • Multi-organ Failure

SECTION 5:

Learning outcomes from this section:
- Interpreting ABGs in determining if the criteria for NIV is met or in measuring effectiveness of NIV

Arterial Blood Gases

Arterial blood gas analysis is a commonly performed procedure within the hospital setting 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.

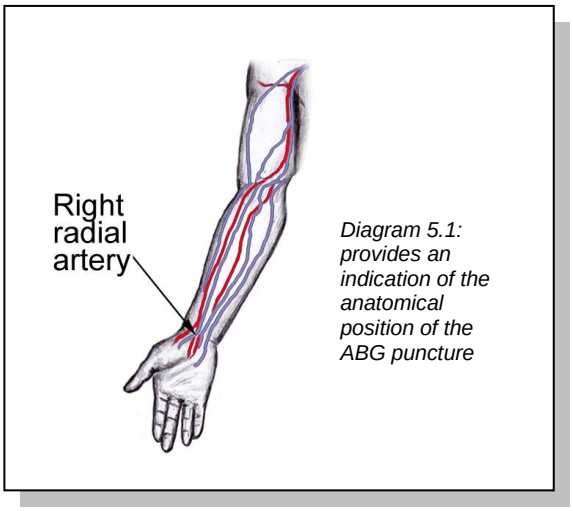


Diagram 5.1: provides an indication of the anatomical position of the ABG puncture

Obtaining an ABG sample:

When performed by an appropriately trained clinician, arterial blood sampling is a relatively safe procedure.
Complications include: Pain, syncope, bleeding, haematoma, altered circulation to distal limb, and infection.

Once obtained, air bubbles must be expelled within the syringe to minimise gas exchange with atmospheric air prior to analysis.

Measured Values:

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

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

As discussed previously, type 2 respiratory failure (T2RF) is defined as a $PaCO_2$ above normal (hypercapnia). Acute, chronic, or acute on chronic type 2 respiratory failure all have different pH and bicarbonate levels.

Increased carbon dioxide within the blood is dissolved as carbonic acid in the plasma, which pushes down the pH.

Acute hypercapnia has a low pH and normal bicarbonate levels. This is because the renal system has not yet compensated the blood pH.

Chronic hypercapnia is displayed with a normal pH, but high bicarbonate levels. This is known as compensated respiratory acidosis, where the kidneys have increased the serum bicarbonate in order to correct a low pH (renal compensation).

In acute on chronic type 2 respiratory failure, a low pH and high bicarbonate level is seen. The high bicarbonate indicates a raised CO_2 for greater than 48 hours, and the acidosis indicates an acutely raised CO_2 in addition to chronically raised CO2.

Below are some typical ABG results for comparison			
Value	**ACUTE T2RF**	**Chronic T2RF**	**ACUTE ON CHRONIC T2RF**
pH	7.31 Low	7.38 Normal	7.26 Low
PaCO2	55 mmHg High	63 mmHg High	72 mmHg High
HCO3	26.8 mmol/L Normal	36 mmol/L High	31.2 mmol/L High
Base excess	+0.9 Normal	+10.5 High	+4.4 High

Venous versus Arterial gases:

Regular arterial puncture can be a painful and distressing experience for the patient. Venepuncture is less invasive, less painful and performed by many health professionals. Venous pH is an accurate indicator of arterial pH, as venous blood pH is consistently 0.03 below arterial pH.

Carbon dioxide levels are less accurate, as venous results can be 0 to +20 mmHg above arterial. Venous PCO_2 can be used to track trends by comparing with previous CO_2 levels. Venous oxygen is not an accurate indicator of arterial oxygenation as it is affected by numerous factors. This should not be used to assess lung function (Byrne et al, 2014). Bicarbonate levels are also inaccurate, as this is a calculated value derived from pH and PCO_2 when using ABG analysis.

Below is an example of a blood gas analysis where both arterial and venous samples where obtained from a single person at the same time.

Value	ABG	VBG
pH	7.40	7.37
PaO_2	82 mmHg	N/A
$PaCO_2$	40 mmHg	40 – 60 mmHg
HCO3	23.9 mmol/L	N/A

Assessment

To be deemed competent to manage a patient receiving NIV requires successful completion of theoretical and practical assessments.

The theoretical assessment consists of the following 30 questions. The pass mark of 80% is a requirement to progress to the practical assessment.

Completing participant details allows the package to be sent to a relevant assessor for marking, enables completed packages to be included in your learning record on HETI online and allows for the provision of feedback.

Participant Name: _____ Payroll number: _____

Role/Designation: _____ Unit/Facility: _____

Contact details (phone and/or email): _____

Theoretical Assessment

Section 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 SaO_2% do symptoms of hypoxia normally begin?

_____

8. Name 3 factors affecting 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. What will happen to the pH when CO2 increases in the blood stream? (circle one)

Reduce Increase Nothing

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:

_____

_____

_____

16. List 5 indications for NIV:

_____

_____

_____

_____

_____

17. List 5 contraindications to NIV:

_____

_____

_____

_____

_____

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 SpO2 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

Section 3:

1. List 5 benefits of NIV:

_____

_____

_____

_____

_____

Section 4:

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

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

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

_____

_____

_____

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

IPAP 20 cmH_2O, EPAP 5 cmH_2O, Rate 14 bpm, FiO_2 .28

What Pressure Support will the patient receive?

_____

4. What type of NIV mask would you select in a patient with claustrophobia?

_____

_____

Section 5:

1. From which artery are Arterial Blood Gases (ABG) typically sampled?

_____

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

_____

3. In compensated respiratory failure, would you expect to see a high or low bicarbonate level?

_____

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

_____

Total Correct answers:

Assessment of Practice

All of the following elements must be achieved in order to complete the program

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

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

Learning Package: Reflection on Learning

This document guides your reflection on the extent to which the package meets your professional development needs, and how you plan to apply your learnings into practice. This tool is not part of the assessment process, and has been included as a document you may wish to include in your professional portfolio.

*Time taken completing the learning package:*_____

What was your purpose in completing this learning package?

_____

_____

_____

_____

Did you achieve this by completing the learning package?

_____

_____

_____

Reflecting on the content, what key learnings have you obtained?

_____

_____

_____

_____

Which learning ('s) will you apply to practice immediately? How will you do this?

_____

_____

_____

_____

What learning needs have you identified as a result of completing this learning package?

_____

_____

_____

How do you plan to address these needs?

_____

_____

_____

_____

Signature:_____ Date:_____

Learning Package Evaluation Form

Please respond to the following questions:

1. The learning outcomes of the package were clearly identified and appropriate?	Yes	No
2. Was the content sufficient to meet to enable you to meet the learning outcomes?	Yes	No
3. The activities were interesting and supported the learning	Yes	No
4. The package was presented logically and the content was easy to follow and made sense.	Yes	No
5.The assessment process was clearly described	Yes	No

6. My most relevant learning(s) from this package were:

_____

_____

7. The learning('s) that I can immediately apply to practice are:

_____

_____

8. The least relevant component(s) of this package were:

_____

_____

9. Some suggestions I would make to improve the package would be:

_____

_____

10. Further comments:

_____

_____

Return to:
Nick Yates, CNC Respiratory Failure & Non-Invasive Ventilation Dept of Respiratory and Sleep Medicine, JHH
nick.yates@hnehealth.nsw.gov.au

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Appendix 1: Respiratory Physiology

INTRODUCTION

The following pages provide an overview of the function of the respiratory system to support the application of this knowledge in the determination for and management of NIV. This knowledge is foundational, and provides an opportunity to review current knowledge and serve as a reference point.

Review 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

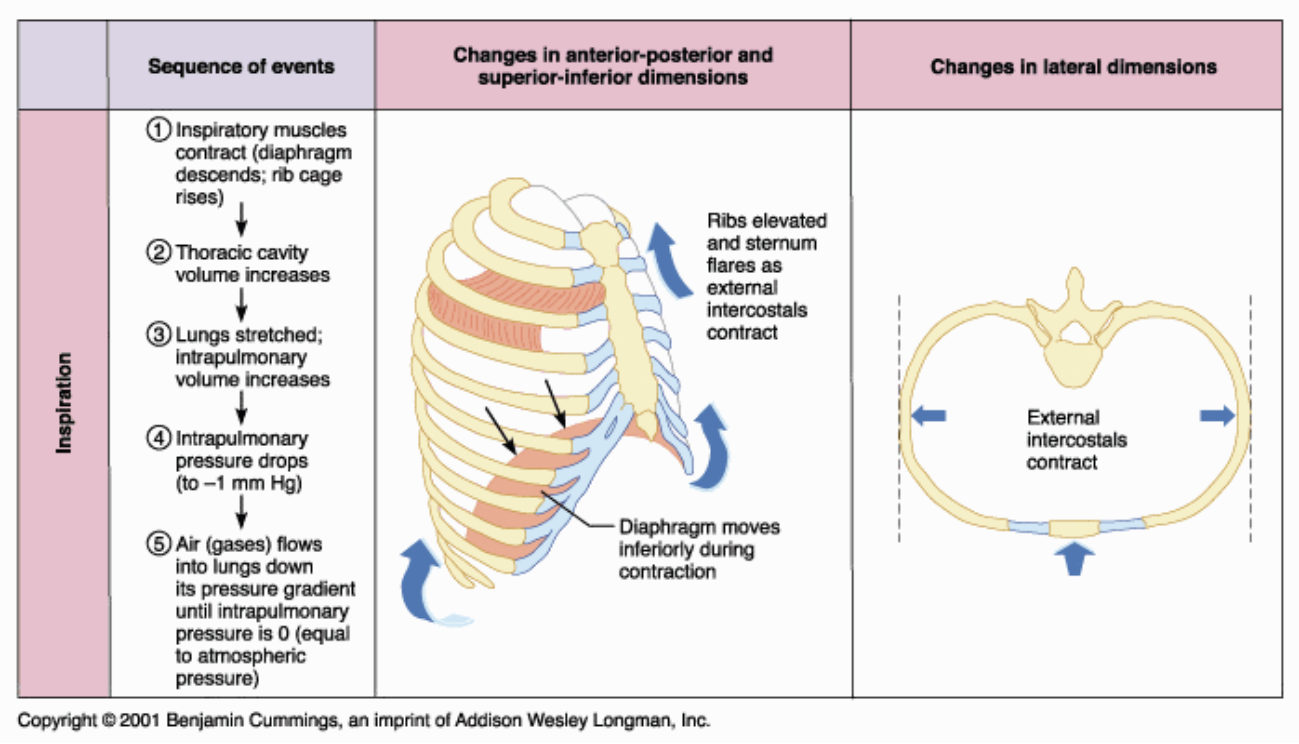
Breathing

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

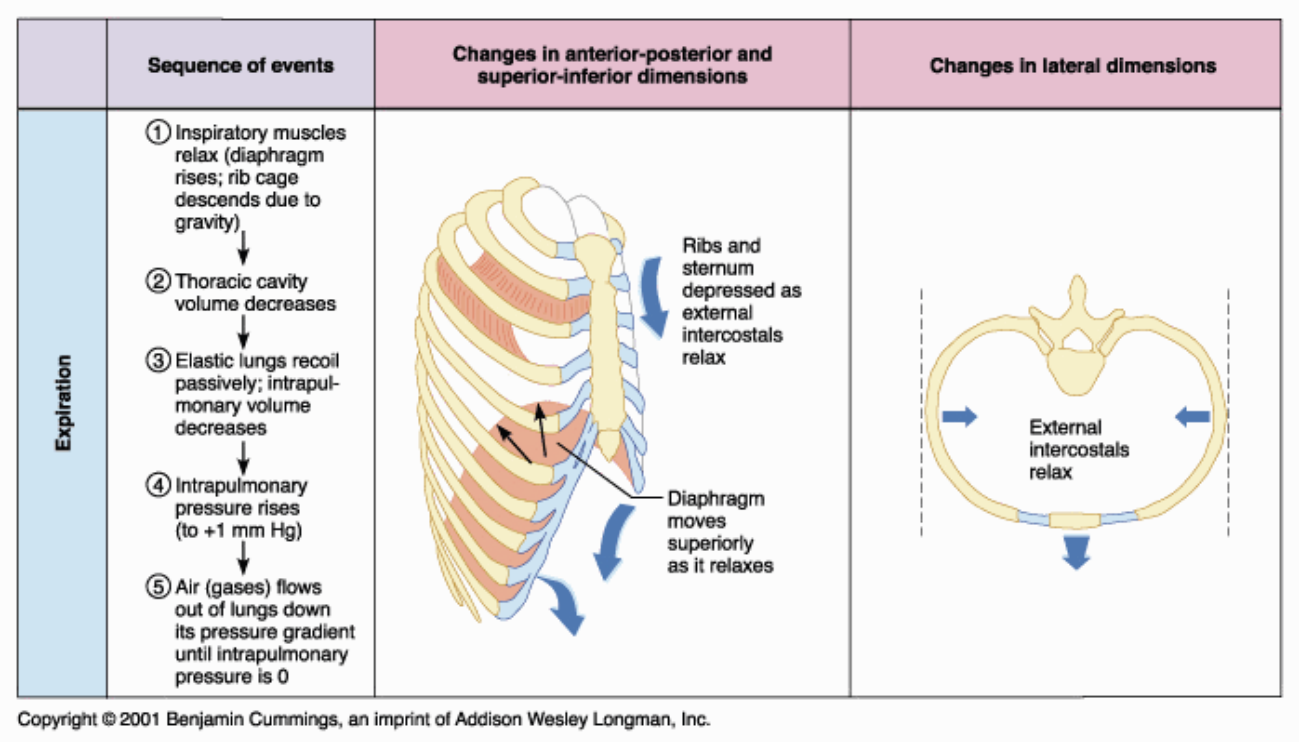
Inspiration is the process of taking air into the lungs. It is the active phase of ventilation because it is the result of muscle contraction. During inspiration, the diaphragm and intercostal muscles contract, enlarging the thoracic cavity. The diaphragm, doing most of the respiratory work during quiet 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.



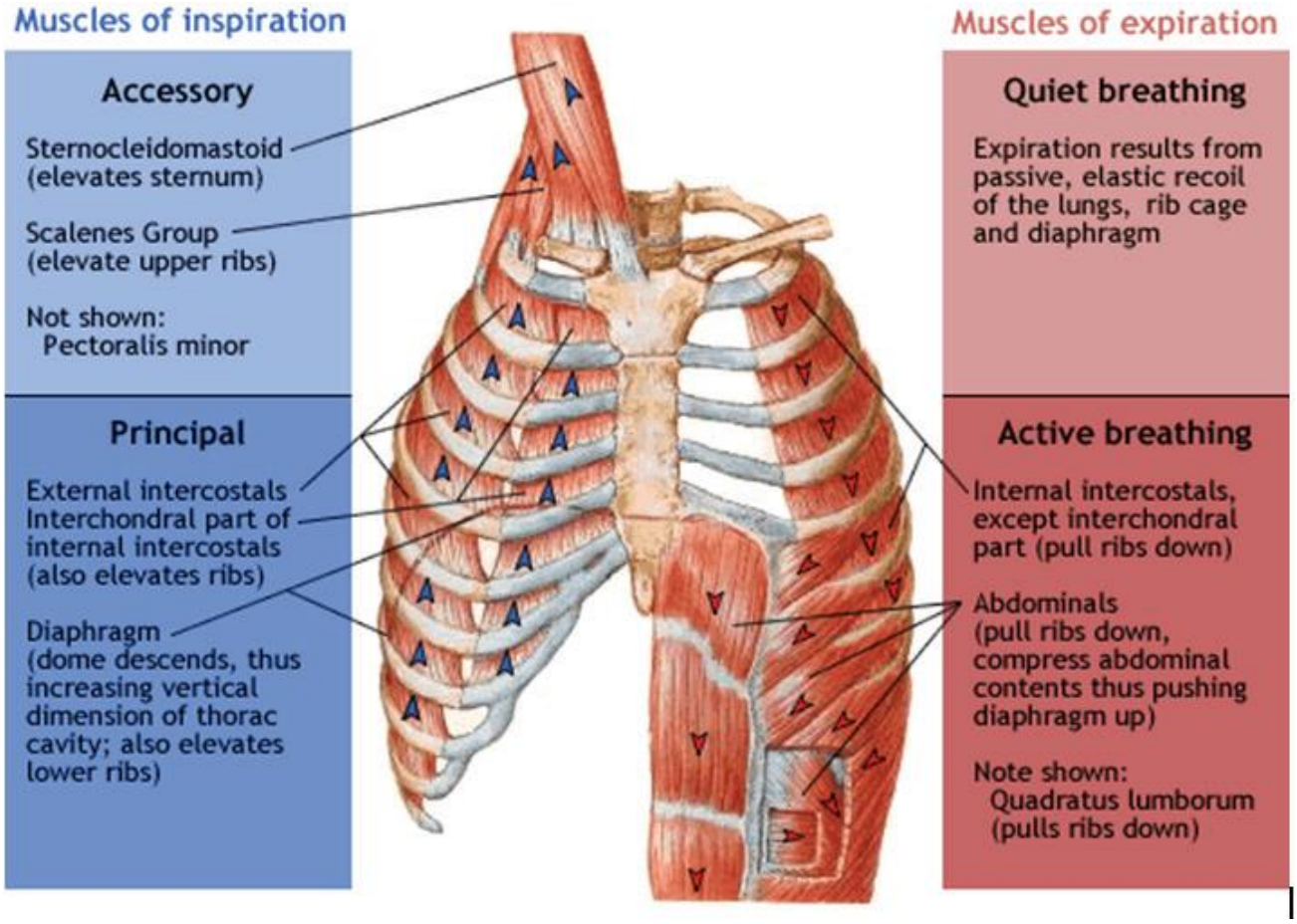
Expiration

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.

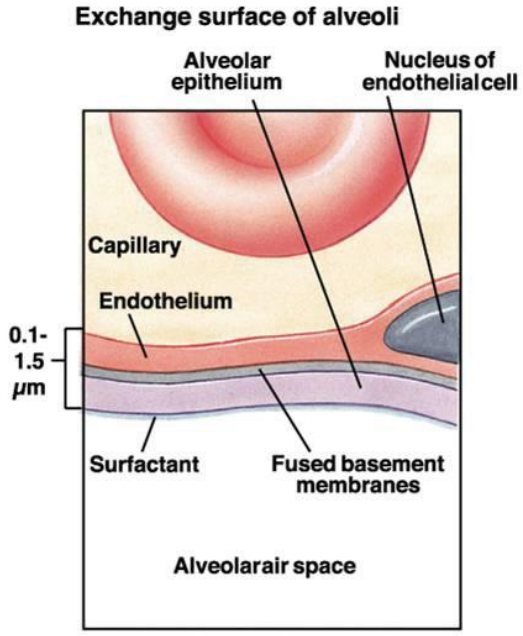
Gas diffusion is affected by:

- 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.



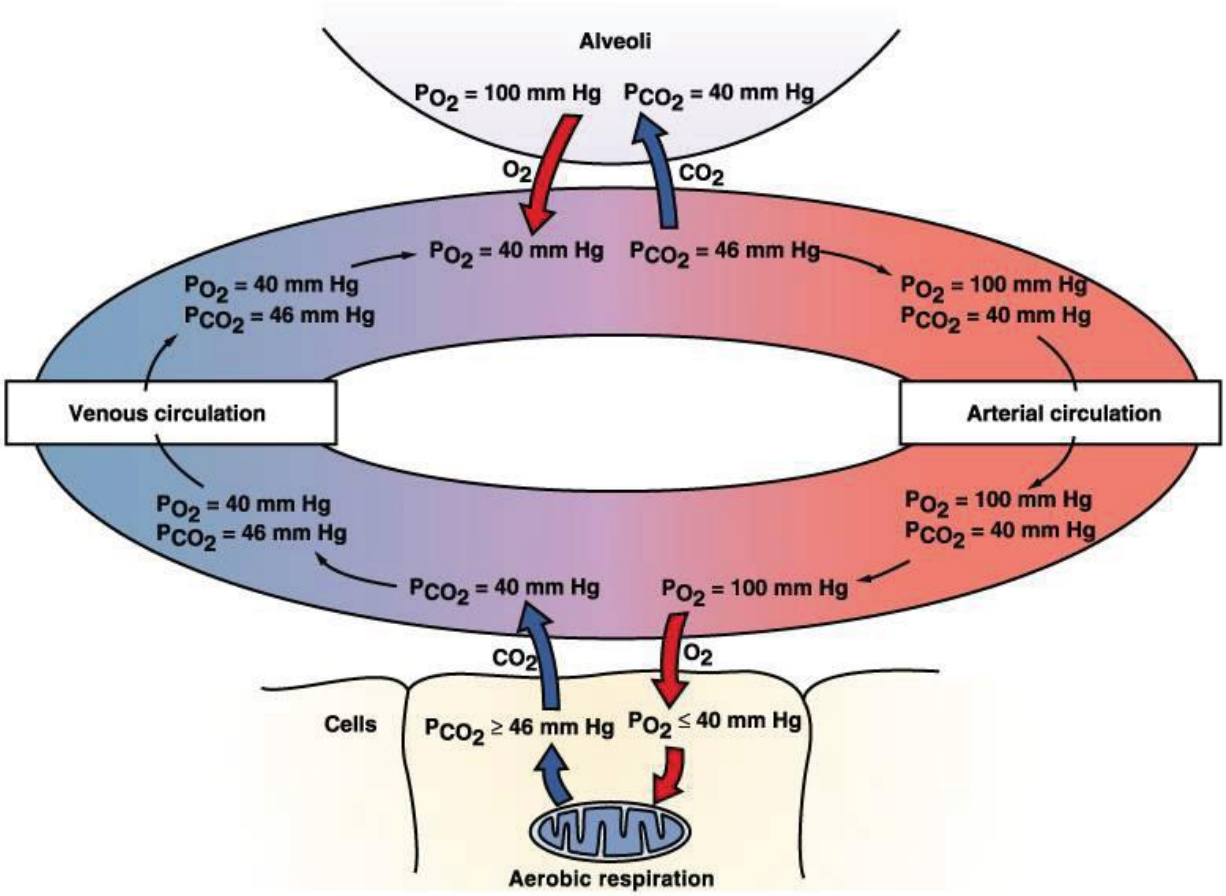
Gas concentrations are expressed as partial pressures. In a mixture of gases, each gas contributes to the total pressure according to its concentration. For example if a gas is 50% of total, it produces 50% of the pressure. The pressure of room air, or atmospheric pressure, at sea level is 760 mmHg. 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%.

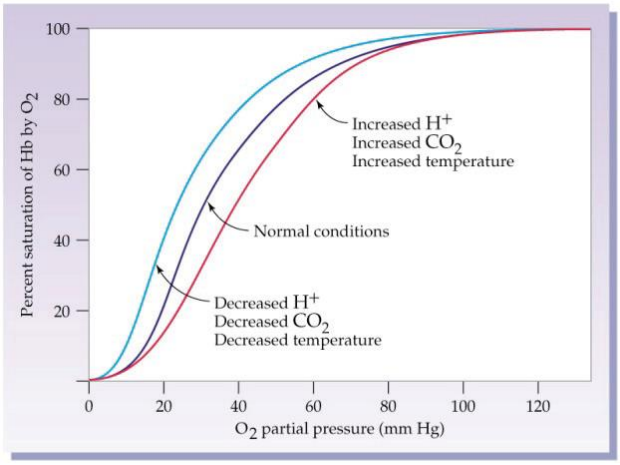
PaO2		SaO2
150	**mmHg**	100 %
100	**mmHg**	97 %
60	**mmHg**	90 %
40	**mmHg**	70 %

The table illustrates the relationship between PaO2 and SaO2:

Where PaO2 is 150mmHg despite increases in PaO2 there is minimal change in the SaO2 as haemoglobin cannot combine with more oxygen.

PaO2 60mmHg is when symptoms of hypoxia begin, despite a drop of half the dissolved oxygen concentration, the haemoglobin is still 90% saturated. Small changes in SaO2 correspond with large changes in PaO2.

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.

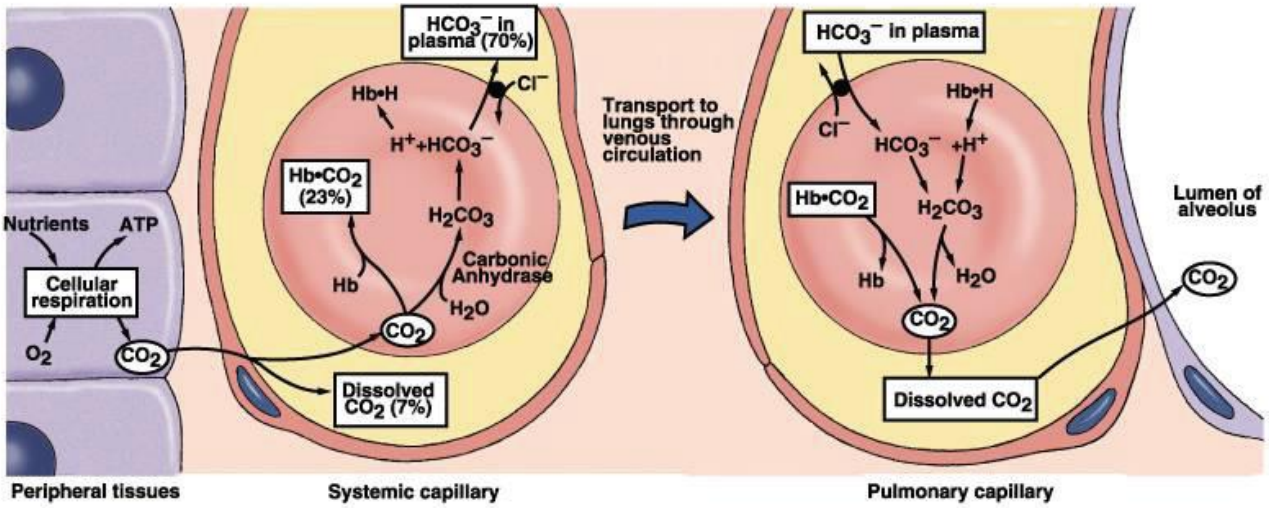


The diagram illustrates:
The Oxyhaemoglobin Dissociation Curve

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.

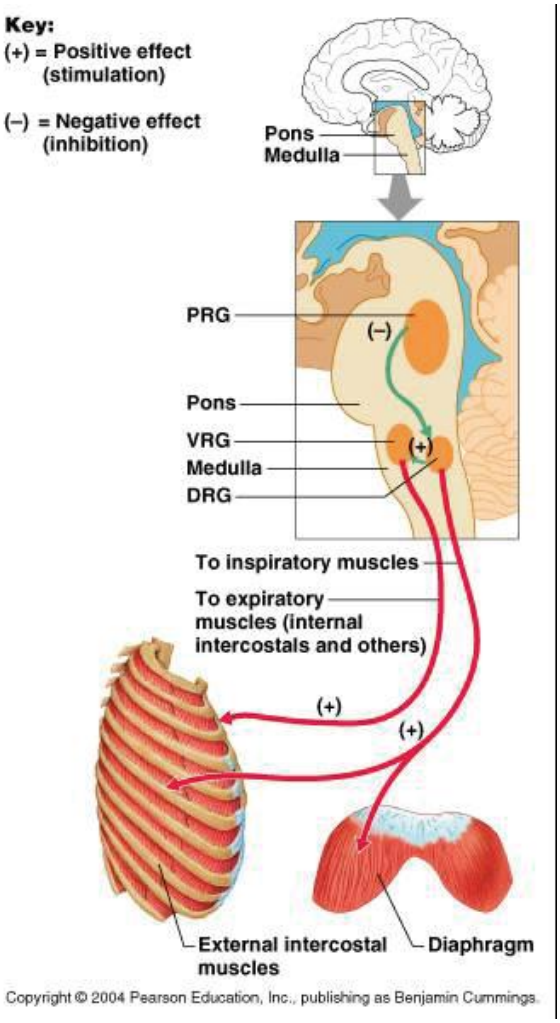


Control of Breathing

Central Control of Breathing

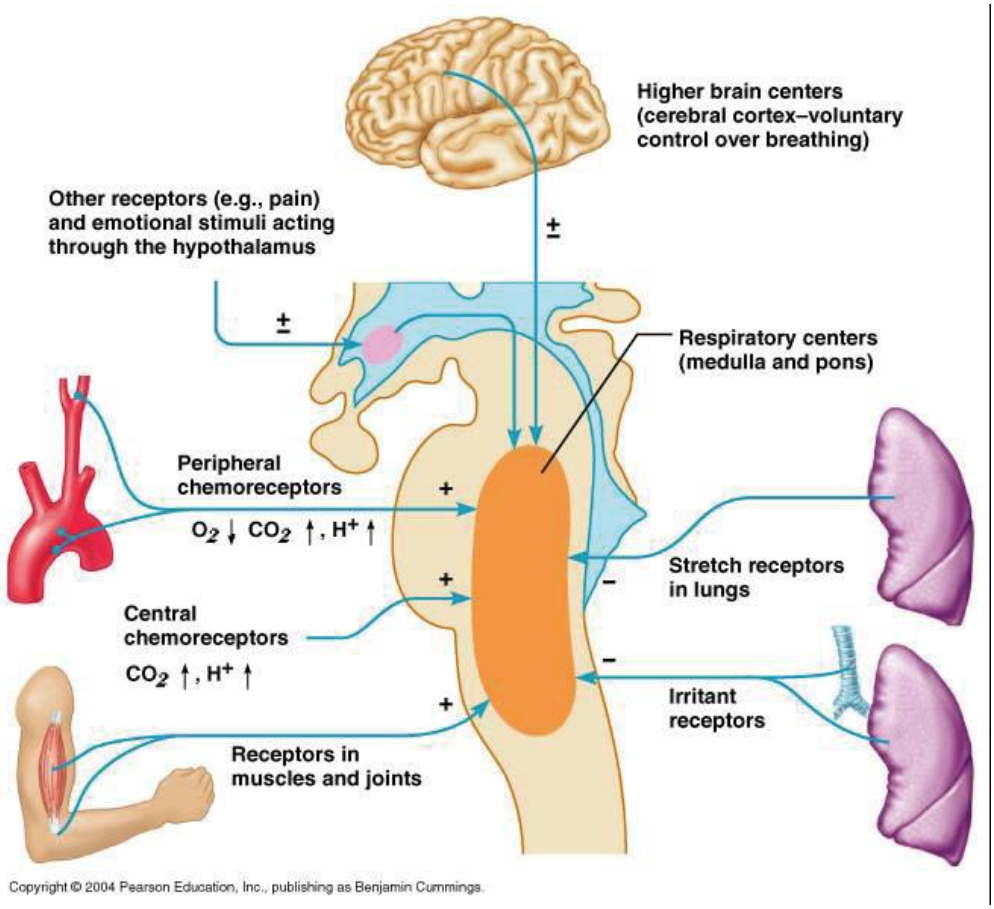
The respiratory centre is found in the brain stem. The pons and medulla oblongata are both integral to the control of breathing. The medulla oblongata rhythmically stimulates the intercostal muscles and diaphragm — making breathing possible. The pons also participates in the reflexes that regulate breathing. 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.

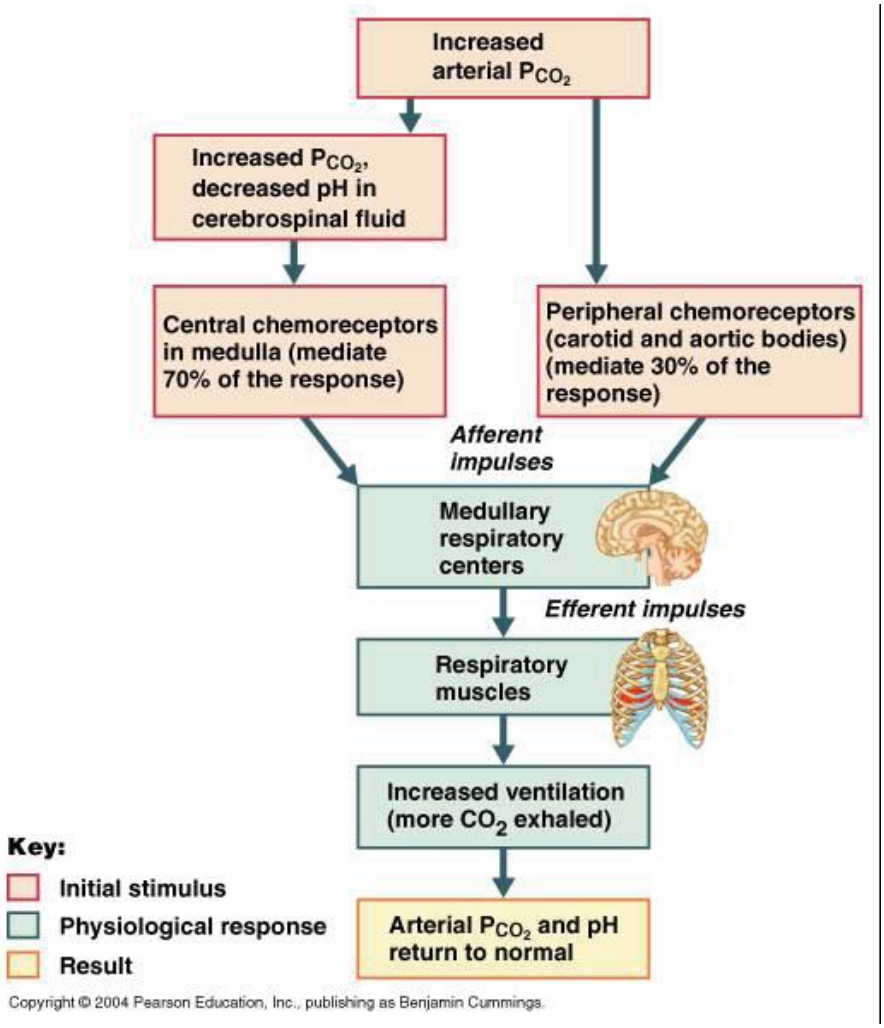
Stretch receptors in the lungs detect over distension, resulting in a negative stimulus to the brain stem, reducing the rate and depth of breathing, protecting against trauma to the airways. Irritant receptors in the bronchi and lungs will also cause a reduction in ventilation, to help prevent deep inhalation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

Appendix 2: Tidal Volume Chart

Height	IBW (*BMI = 23*)	Target VT 6 mL/kg	Target VT 7 mL/kg	Target VT 8 mL/kg
1.50 m / 59 in	52.0 kg	310 mL	360 mL	420 mL
1.55 m / 61 in	55.0 kg	330 mL	390 mL	440 mL
1.60 m / 63 in	59.0 kg	350 mL	410 mL	470 mL
1.65 m / 65 in	62.5 kg	380 mL	440 mL	500 mL
1.70 m / 67 in	66.5 kg	400 mL	470 mL	530 mL
1.75 m / 69 in	70.5 kg	420 mL	490 mL	560 mL
1.80 m / 71 in	74.5 kg	450 mL	520 mL	600 mL
1.85 m / 73 in	78.5 kg	470 mL	550 mL	630 mL
1.90 m / 75 in	83.0 kg	500 mL	580 mL	660 mL

COPD / OHS:

Height	IBW (*BMI = 23*)	Target VT 8 mL/kg	Target VT 9 mL/kg	Target VT 10 mL/kg
1.50 m / 59 in	52.0 kg	420 mL	470 mL	520 mL
1.55 m / 61 in	55.0 kg	440 mL	500 mL	550 mL
1.60 m / 63 in	59.0 kg	470 mL	530 mL	590 mL
1.65 m / 65 in	62.5 kg	500 mL	560 mL	630 mL
1.70 m / 67 in	66.5 kg	530 mL	600 mL	670 mL
1.75 m / 69 in	70.5 kg	560 mL	640 mL	710 mL
1.80 m / 71 in	74.5 kg	600 mL	670 mL	750 mL
1.85 m / 73 in	78.5 kg	630 mL	710 mL	790 mL
1.90 m / 75 in	83.0 kg	660 mL	750 mL	830 mL