MECHANICAL VENTILATION

LEARNING PACKAGE AND COMPETENCY

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&

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Introduction

Mechanical ventilation is a supportive therapy used to assist patients who are unable to maintain adequate oxygenation or carbon dioxide elimination. There are invasive and non-invasive techniques of mechanical ventilation, both of which require a working knowledge of underlying anatomy and pathophysiology of ventilation and of the disease processes which affect ventilation.

Mechanical ventilation is frequently utilised for patients in the Emergency Department (ED) requiring resuscitation and ongoing respiratory support for variety of illness and injury. Therefore nurses working in the ED need to develop knowledge in the physiological effects of mechanical ventilation, the ventilation techniques that can be used and skills in assessment and interpretation of ventilation observations.

In the past few years there has been an increase in the number of methods by which positive pressure ventilation can be delivered. The increasing number of methods available to deliver mechanical ventilation has made it difficult for clinicians to learn all that is necessary in order to provide a safe and effective level of care for patients receiving mechanical ventilation.

Despite the method by which mechanical ventilation is applied the primary factors to consider when applying mechanical ventilation are:

- the components of each individual breath, specifically whether pressure, flow, volume and time are set by the operator, variable or dependent on other parameters
- the method of triggering the mechanical ventilator breath/gas flow
- potential complications of mechanical ventilation and methods to reduce ventilator induced lung injury
- methods to improve patient ventilator synchrony; and
- the nursing observations required to provide a safe and effective level of care for the patient receiving mechanical ventilation
This mechanical ventilation self learning package and competency assessment are designed to educate emergency nurses on anatomy, physiology and the principles and practice of invasive mechanical ventilation for adult patients and for the purpose of maintaining a high standard of clinical competency for all staff involved in the care of the mechanically ventilated patient.

This is an annual competency and a 100% pass is mandatory.
The respiratory system is comprised of numerous organs and structures and is dependent on the proper functioning of most body systems. The anatomical structures that aid in lung function during inspiration and expiration are the lungs, the diaphragm, the intercostal muscles and the abdominal and accessory muscles. The mouth and throat are also vital in ventilation and maintaining a healthy, normal functioning respiratory system. The respiratory system can be divided into the upper respiratory system and the lower respiratory system.

The upper respiratory system consists of the nostrils (external nares), nasal cavity, nasal vestibule, nasal septum, both hard and soft palate, nasopharynx, pharynx, larynx and trachea. The hard palate serves to separate the oral and nasal cavities. The nose is the primary entrance for air into the respiratory system. The nasal cavity is lined with mucous membranes which work to filter, warm and humidify the incoming air. Dust, bacteria and other chemicals present in the air get caught in the mucous and are denatured in the stomach once swallowed with the mucous, preventing them from entering the lungs (Martini & Bartholomew, 2007). The pharynx provides a passageway for the respiratory and
digestive tracts. The larynx is primarily responsible for vocalization but is important in protecting the lower airways from foreign substances/objects. The larynx facilitates coughing. The upper airway is also important because it must always stay open for breathing to occur.

The lower respiratory tract consists of the trachea, bronchi, bronchioles, lungs, diaphragm and rib cage. The trachea, a fairly rigid pipe, dividing at the carina branches off into what is known as the bronchi (more commonly called bronchial tubes). These two main bronchi have branches forming the bronchial tree. Where it enters the lung, there are then secondary bronchi. In each lung, the secondary bronchi divide into tertiary bronchi and in turn these divide repeatedly into smaller bronchioles. The bronchioles control the ratio of resistance to airflow and distribution of air in our lungs. The bronchioles open into the alveolar ducts. Alveolar sacs are at the end of the ducts. These sacs are chambers that are connected to several individual alveoli, which make up the exchange surface of the lungs (Martini & Bartholomew, 2007).

The lungs are enclosed in the thoracic cage, consisting of the sternum, vertebral column, ribs – with intercostals muscles – and the diaphragm. The lungs are separated into right and left by the mediastinum (containing the heart, large vessels, trachea and oesophagus). Each lung is divided into lobes – upper and lower – on each side and a third (middle) lobe on the right. The lobes are functionally independent of each other. Two layers of pleura – visceral and parietal – surround each lung. The lungs lie adjacent to the chest wall and the gap between is the pleural space, containing pleural fluid. The lungs are made up of elastic fibres that give it the ability to handle large changes in air volume.

There are no attachments between the lung and the walls of the chest cage except where it is suspended at its hilum from the mediastinum, therefore, the lung ‘floats’ in the thoracic cavity, surrounded by the very thin layer of pleural fluid that lubricates the movements of the lung within the cavity. The lungs are ‘linked’ to the chest wall by a sub-atmospheric or negative pressure within the pleural space. The lungs are elastic structures that will collapse like a balloon and expel most of the contained air through the trachea whenever there is no force to keep them inflated. The structures that enlarge the thoracic cavity are the muscles of respiration: the diaphragm, the intercostal muscles and the abdominal muscles.

The diaphragm is a thin dome shaped sheet of muscle and ligament inserted into the lower ribs and innervated by the phrenic nerve. During inspiration it contracts and moves downwards, increasing the vertical dimension of the thoracic volume and displacing the abdominal contents downwards. During expiration it relaxes, moves upwards and decreases the vertical dimension of the thoracic volume.
Innervation of the intercostal muscles is supplied by the intercostal nerves. During inspiration, they contract and increase the anterior – posterior and transverse diameters of the thoracic cage by pulling up and out. During expiration, they relax and decrease the diameter of the thoracic cage and force the air out.

The accessory muscles include the sternocleidomastoids and scalene muscles. During inspiration, they contract to increase the dimensions of the thoracic cage, but are only used to overcome large resistances (asthma, exercise etc).

The function of the upper airway must be taken into consideration when mechanically ventilating a patient, as the upper airway is bypassed either via an endotracheal tube or tracheostomy. Therefore, filtering, warming and humidifying the mechanical ventilation circuit is imperative in maintaining a healthy respiratory system.

![Muscles of inspiration and expiration](image-url)

**FIGURE 2.3** Muscles used during quiet and active breathing. (Image from Ingraham, 2004)
THE NORMAL PHYSIOLOGICAL PROCESS OF INSPIRATION/EXPIRATION

The goal of inspiration and expiration is to exchange oxygen (O2) and carbon dioxide (CO2). Oxygen is required for many cellular metabolic functions. CO2 is produced during these processes and requires excretion from the body. The exchange of these gases between the internal environment and external environment occurs primarily in the lungs.

There are three phases of respiration:

1) Pulmonary ventilation (inspiration/ exhalation)
2) Pulmonary respiration (gas exchange at the lung)
3) Tissue respiration (gas exchange at the tissues)

For this process to be efficient there must be an intact and functioning respiratory system for adequate gas exchange and for the cardiovascular system to deliver and remove respiratory gases. The respiratory system can be divided into the upper and lower respiratory tract. Together, they function to provide the body with oxygen and remove carbon dioxide (i.e. gas exchange).

Pulmonary ventilation is the movement of air in and out of the lungs and pulmonary respiration is the process of gas exchange, that is, the movement of oxygen from the atmosphere into the bloodstream and the movement of carbon dioxide from the bloodstream into the atmosphere.

The alveolar-capillary membrane is where the exchange of oxygen and carbon dioxide occurs. O2 is circulated throughout the body, via the blood and then CO2 is excreted from the body via the lungs (expiration). This process happens via diffusion (moving molecules from an area of high concentration to an area of low concentration).
BREATHING REGULATION

Regulation of breathing is determined by the respiratory centre in the medulla oblongata and the pneumotaxic centre in the pons which receives and sends impulses to the central receptors in the brain and the peripheral receptors in the carotid arteries. The medulla oblongata rhythmically stimulates the intercostals 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. Breathing can also be affected by emotional factors (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 central receptors respond to carbon dioxide levels in the blood and the peripheral receptors respond to blood pH, so a high CO2 with a low pH stimulates breathing, whereas a low CO2/high pH impedes breathing (Martini & Bartholomew, 2007).
MECHANICS OF VENTILATION

The mechanics of ventilation refer to the physical factors that govern airflow into and out of the lungs. Air/gas move from an area of higher pressure to an area of low pressure, the aim being to equalize pressures.

Inspiration is an active process, during which, air flows from the atmosphere to the lungs in response to pressure gradients. As air is breathed in, the diaphragm flattens and pushes downward and the external intercostal muscles contract and lengthen, increasing the size of the thoracic cavity, causing the intrapleural pressure to become more negative. This negative pressure causes air to rush in through the upper airways and down into the lower airways (Pierce, 2007). This expands the alveoli, which in turn causes atmospheric air to enter the lungs. When equilibrium between the airway pressure and intrapleural pressure is achieved, the airflow stops.

Expiration is a passive event, requiring little to no effort from the muscles of respiration. During expiration the inspiratory muscles relax, the relaxation reduces the size of thoracic cavity and squeezes air out of the lungs. When the pressure gradient between the alveolar and intrapleural pressure is gone, the airflow stops.
AIRWAY PRESSURES

Pressures in the respiratory system are always described relative to the atmosphere (i.e. 760 mmHg), therefore; a negative pressure (e.g.: -4mmHg) is lower than atmospheric pressure (760 - 4 = 756) and a positive pressure (e.g.: +4mmHg) is higher (760 + 4 = 764). There are two primary pressures referred to when discussing airway pressures - intrapulmonary and intrapleural pressure.

Intrapulmonary pressure (alveolar pressure) is the pressure in the alveoli which rises and falls with respiration. Intrapulmonary pressure changes as the intrapleural pressure changes. During Inspiration intrapulmonary pressure is about -1 cm H$_2$O, and during exhalation it is about +1 cm H$_2$O. (Pilbeam & Cairo, 2006).

Intrapleural pressure is the pressure in the pleural cavity which also fluctuates with breathing but remains approximately 4 mmHg less than intrapulmonary and atmospheric pressures (see diagram below). This negative pressure occurs due to a balance between the elasticity of the lungs, the surface tension of the alveoli and the elasticity of the chest wall (Marieb, 2007).
GAS EXCHANGE

The movement of gases occurs via passive diffusion across the alveolar membrane. The capillary and alveolar walls are very thin, allowing rapid exchange of gases by passive diffusion along concentration gradients.

CO₂ moves into the alveolus as the concentration is much lower in the alveolus than in the blood, and O₂ moves out of the alveolus as the continuous flow of blood through the capillaries prevents saturation of the blood with O₂ and allows maximal transfer across the membrane. There are approximately 300 million alveoli in each lung which provide roughly 200ml/minute of oxygen at rest (Pierce, 2007).

VENTILATION/PERFUSION RELATIONSHIPS

When the number of alveoli that are ventilated equals the number of alveoli that are perfused, ventilation and perfusion are equally matched. This is normal. If more alveoli are perfused than are ventilated, a ventilation-perfusion (V/Q) mismatch called shunting results. Pulmonary shunting results from problems that prevent air exchange in the alveoli (e.g. atelectasis). If more alveoli are ventilated that are perfused, a V/Q mismatch called dead space results. Pathologic pulmonary dead space results from problems that interfere with blood flow to the alveolar capillaries, e.g. pulmonary embolism (Pierce, 2007).

OXYHAEMAGLOBIN DISSOCIATION CURVE (Table 1)

The oxyhaemoglobin dissociation curve depicts the relationship between the partial pressure of oxygen (O₂ dissolved in blood, the- PaO₂) and oxygen saturation (SaO₂). It shows the degree to which O₂ remains associated with haemoglobin (Hb) under different PaO₂ levels (i.e. how readily Hb acquires and releases O₂ into the surrounding fluid).

What does it tell us?

- The ‘plateau’ portion of the curve reflects the PaO₂ and SaO₂ in the lungs (pulmonary capillaries)
  - Partial pressure is high, O₂ binds readily to Hb

- The ‘steep’ portion reflects the PaO₂ and SaO₂ in the tissue (systemic capillaries)
  - Partial pressure is less, O₂ released readily from Hb

- At a PaO₂ of approx 26mmHg, Hb is 50% saturated
How is this significant?

- Large changes in PaO\(_2\) can be tolerated without significantly altering saturation (in the absence of disease or other conditions)
- An SaO\(_2\) (or SpO\(_2\)) of 95-100% on room air suggests that arterial blood is close to fully saturated, which is adequate for normal cell function

Once PaO\(_2\) levels fall below 60mmHg, O\(_2\) saturation decreases rapidly (Hb affinity decreases, O\(_2\) is released). Left shift of the curve is a sign of haemoglobin’s increased affinity for oxygen (e.g. at the lungs). Similarly, right shift shows decreased affinity, as would appear with an increase in body temperature, hydrogen ion concentration, 2,3-diphosphoglycerate (also known as bisphosphoglycerate) or carbon dioxide concentration, known as the Bohr effect (Pierce, 2007).

Oxyhaemoglobin dissociation curve (Table 1)
ALTERATIONS TO THE NORMAL PHYSIOLOGICAL PROCESS OF INSPIRATION/EXPIRATION

The lungs are the most important organ to consider when mechanically ventilating a patient. Non-functioning and ill-functioning lungs are a common reason for mechanical ventilation. Respiratory failure, pneumonia, asthma, Acute Pulmonary Oedema (APO), Pulmonary Embolus (PE) and Chronic Obstructive Pulmonary Disease (COPD), Acute Respiratory Distress Syndrome (ARDS), among others, are examples of conditions that may require a patient to receive mechanical ventilation.

The lungs are the largest organ in the body. The total surface area is more than 100 square yards—the size of a tennis court. The delicate gas exchange membrane is 1/50th the thickness of tissue paper. Millions of tiny capillaries carry red blood cells throughout the body, transporting oxygen in and carbon dioxide out. These red cells also contain mechanisms that defend against cigarette smoke, air pollution, and other noxious materials. Thus, the red cells are not only servants to every organ and tissue, but they are also the defenders against cancer, emphysema, and other lung diseases.

White cells, which defend us against infection, also traverse the lungs. In fact, at any one minute at least 17 billion white blood cells are travelling through our lungs. Billions are stored there, poised and ready to attack bacteria, viruses, and other infectious invaders that we inhale every day.

The total surface of the alveolar membrane varies from 100 to 200 square meters, depending on the size of the person. The overall surface area of the alveolar membrane has frequently been equated to that of a tennis court.

The central premise of positive pressure mechanical ventilation is that gas flows along a pressure gradient between the upper airway and the alveoli. The magnitude, rate and duration of flow are determined by the doctor or Critical Care trained Registered Nurse (CCRN). When initiating mechanical ventilation, we change the mechanics of normal ventilation from one which operates under negative pressure to positive pressure. Positive pressure ventilation (PPV) can have serious short and long term complications (Pierce, 2007). Before discussing the ‘how to’ of mechanical ventilation, it is necessary to review respiratory anatomy and physiology and define some key concepts to assist you with understanding the principles of mechanical ventilation.

When a patient is connected to positive pressure mechanical ventilation, the normal physiological process of inspiration and expiration alters. Airway pressures (intrapulmonary and intrapleural) become positive, forced pressures (a reversal of normal chest pressures) occur.
Atmospheric air and O2 is now being ‘forced’ into the lungs. The patient is probably sedated and perhaps paralysed by use of medication.

Control of ventilation has been transferred from the respiratory centre in the brain, chemoreceptors and accessory muscles of respiration, to a ventilator machine and its operators, via an endotracheal tube (ETT).

The positive pressures introduced result in alterations in gaseous exchange, circulatory flow, hormone secretion and metabolic stability.

Consider possible complications or difficulties in mechanically ventilating a patient such as cardiovascular changes (reduced venous return and cardiac output due to the loss of negative intrathoracic pressure, constriction of pulmonary vasculature, reduced venous return, reduced preload and cardiac output), pulmonary changes (asynchronous breathing, atelectasis, barotrauma, pneumothorax, tension pneumothorax, pneumomediastinum, gastrointestinal alterations, acid-base balance disturbances and water balance disturbances).

Compliance and resistance, as well as elasticity, dead space and surface tension are factors that affect how easily a patient can be ventilated.

**Compliance** denotes distensibility (stretchiness) of the lung and chest wall combined or the ease with which the lungs and thoracic wall can be expanded. When compliance is low, the lungs are stiffer and more effort is required to inflate the alveoli. Conditions that worsen compliance, such as pulmonary fibrosis, produce restrictive lung disease. Compliance also varies within the lung according to the degree of inflation. Poor compliance is seen at low volumes (because of difficulty with initial lung inflation) and at high volumes (because of the limit of chest wall expansion), with best compliance in the mid-expansion range. Factors effecting chest wall compliance include pneumothorax, haemothorax, abdominal distension, obesity and penetrating or blunt chest wall trauma. Compliance is affected by elasticity and surface tension. Less elasticity and increased surface tension lowers compliance. Compliance decreases in conditions that scar the lung tissue (Tuberculosis), cause it to be filled with fluid (APO, pulmonary fibrosis) or impede lung expansion (ARDS, pneumonia).

**Resistance** describes the opposition to airflow caused by friction. Generally, the smaller the diameter, the greater the friction, and thus the greater the amount of resistance. Therefore, one small airway provides greater resistance than one large airway. However, resistance to airflow also depends on the number of pathways present not just the size. The medium sized airways therefore provide the greatest amount. An increase in resistance resulting from airway narrowing such as bronchospasm leads to obstructive airways disease. Physiological factors effecting lung resistance include bronchospasm, emphysema, foreign body or obstruction, excessive secretions and Tracheobronchial Malacia. Mechanical factors
Effecting lung resistance include filters or heat moisture exchangers (HME’s), endotracheal tube size, water in tubing, gas flow rate and the expiratory valve of the ventilator.

- Notice the diameter of the airway decreases when the patient has been intubated.
Surface tension- is the force exerted at all air-water junctions. In the lungs it is caused by a thin layer of alveolar fluid. This force must be overcome to expand the lungs during inspiration. The presence of surfactant in the alveoli fluid reduces its surface tension, preventing the alveoli from completely collapsing during expiration.

Elasticity- is the ability of the lung to recoil to its resting volume and normal shape after the stretching force is released.

Dead space- refers to inhaled air that does not take part in gas exchange. Dead space can be further categorised into anatomical and alveoli or physiological dead space. Anatomical dead space describes the air in the conducting airways which does not come into contact with the alveoli. Approximately one third or 150mls of each normal breath is considered anatomical dead space. Ventilator tubing, inline suction units and filters all increase dead space. Alveolar dead space occurs with the ventilation of relatively underperfused or nonperfused alveoli, resulting in limited gas exchange taking place. It is negligible in normal lungs, however, is increased with underlying lung conditions such as PE, APO or pneumonia. An increase in the amount of dead space results in an increase in minute volume and therefore increased work of breathing.
INDICATIONS OF USE FOR MECHANICAL VENTILATION

Mechanical Ventilation is required for a number of clinical conditions. The principle aim of mechanical ventilation is to support gas exchange by increasing alveolar ventilation and subsequent arterial oxygenation (Curtis et al).

Mechanical Ventilation is primarily used for the management of respiratory failure and respiratory conditions including supporting cardiopulmonary gas exchange (pulmonary ventilation, pulmonary respiration and arterial oxygenation), increasing lung volume (end-expiratory lung inflation and functional capacity), reducing the work of breathing, reversing hypoxemia and acute respiratory acidosis, relieving respiratory distress, preventing or reversing atelectasis and respiratory muscle fatigue. Other clinical conditions indicating the use of mechanical ventilation include permitting sedation and and/or neuromuscular blockade, decreasing oxygen consumption, reducing intracranial pressure, stabilizing the chest wall and for cardiopulmonary support. Mechanical ventilation is also used in conjunction with airway interventions for the patient with decreased GCS or the patient unable to maintain own airway due to trauma, burns or foreign body.

So, in short, mechanical ventilation is indicated if:

- The partial pressure of oxygen in arterial blood (Pa O$_2$) cannot be maintained above 50mm Hg despite high levels of delivered oxygen.

  **Clinical example: Acute Respiratory Distress Syndrome (ARDS).**

- The partial pressure of carbon dioxide in arterial blood rises above 50mmHg

  **Clinical example: Acute Respiratory Failure (ARF) related to opioid overdose.**

- Ventilation becomes inefficient and/or the patient is exhausted.

  **Clinical example: bronchospasm, flail chest and impending respiratory failure.**

- Airway protection

  **Clinical example: tracheal injury, oedema, severe head injury, reduced GCS and facial fractures.**
Oxygen uptake via the lungs is dependent on a number of factors. Some can be manipulated to a large extent by mechanical ventilation:

- PaO2 can be manipulated by altering FiO2, alveolar pressure and ventilation.
- Ventilation-perfusion matching (re-opening collapsed alveoli, thereby reducing intrapulmonary shunting) can be manipulated by using and increasing PEEP to splint open alveoli.

Carbon Dioxide elimination via the lungs is largely dependent on alveolar ventilation. Alveolar ventilation = RR x (Vt – dead space), where Vt=tidal volume.

So, to improve oxygenation:

- Increase FiO2
- Increase mean alveolar pressure and mean airway pressure (ie. Increase PEEP and/or I:E ratio
- Re-open alveoli with PEEP

To reduce carbon dioxide elimination:

- Increase respiratory rate
- Increase tidal volume
PHASES, TYPES AND MODES OF VENTILATION

PHASES Mechanical Ventilation has four phases that imitate spontaneous breathing.

TRIGGER- Inspiration commences: The trigger variable opens the inspiratory valve and initiates gas flow. This trigger can be a decrease in gas pressure or gas flow, due to the inspiratory effort of the patient. The amount of effort the patient has to produce is referred to as the sensitivity.

BREATH CONTROL & LIMIT- Inspiration is performed: The breath delivered by the mechanical ventilator can be either set to reach a target inspiratory pressure or a target inspiratory volume. These are known as pressure-controlled or volume-controlled breaths. As a safety measure the breaths can be pressure (most commonly) flow, or volume limited.

CYCLE- End of inspiration and switch to exhalation- Once the preset pressure or volume is reached, the ventilatory cycle is completed, triggering the end of inspiration and the start of exhalation. This commencement of exhalation is known as the ‘cycle’. Pressure cycled ventilation delivers a breath until a preset pressure is reached within the patient’s airway. A timed breath, on the other hand, is delivered over a preset time interval and breaths are ‘time cycled’ to exhalation. During spontaneous breaths, most ventilators are flow cycled, ie- a preset deceleration in the inspiratory flow rate causes the machine to cycle from inspiration to exhalation. At the end of the cycle, the gas flow stops and the expiratory valve opens.

BASELINE- Allows exhalation- The patient exhales to a baseline pressure set on the ventilator which is at or above atmospheric pressure. When this baseline is set above atmospheric pressure it is known as positive end-expiratory pressure (PEEP). Used at the end of exhalation, PEEP prevents total collapse of the alveoli and therefore promotes adequate gas exchange.

TRIGGER- Recognises the end of exhalation and switches to inspiration – The 4 phases of a breath commence again.
CONTROL

Bendigo Health ED utilises the Oxylog 3000 which can be set as either a volume-controlled or pressure-controlled ventilator for temporary use on patients with a tidal volume of 50mls upwards. Volume-controlled ventilation is the most common form of mandatory ventilation currently used in adult medicine because it provides a consistent breath-to-breath tidal volume. Termination of the delivered breath is signalled when a set volume leaves the ventilator.

By choosing either volume or pressure controlled ventilation, the clinician determines the control variable that will be used to establish gas flow to the patient. Control variables are independent variables.

Most ventilators of recent times have the ability to ventilate in a number of ways:

**Volume Controlled**

This type of ventilation terminates inspiration after delivering a pre-set volume of gas regardless of the pressure required to deliver it, and then allows for passive exhalation. It delivers a constant, adequate tidal volume even when airway resistance increases. Pressure is variable, dependent upon resistance, compliance and inspiratory effort. It is commonly used in those with weak respiratory muscles, hypoxia or hypercapnia. Volume controlled ventilation guarantees a specific tidal volume delivery regardless of changes in lung compliance and resistance or patient effort. Volume controlled ventilation is used when the goal is to maintain a certain PaCO2. It is also used in those with weak respiratory muscles, hypoxia or hypercapnia. Try to avoid volume control in those with bronchospasm or ARDS (if severe). The main disadvantage of volume controlled ventilation is barotrauma/volutrauma due to high alveolar and ventilation pressures. In volume targeted ventilation, the volume provided is constant and independent of what happens to pressure when the patient’s lung characteristics change or when the patient’s effort changes. The selection of using volume or pressure ventilation is based on whether consistency of tidal volume delivery is important or the limiting of pressure delivery is important.

**Pressure Controlled**

Allows the clinician to set pressure as the independent variable; the pressure remains constant and volume delivery changes as lung characteristics change. The target inspiratory pressure is set and tidal volume must be monitored. Pressure controlled ventilation (PCV) terminates inspiration when a pre-set target pressure is reached, then allows for passive exhalation. Pressure controlled ventilation is commonly used for those at high risk of barotrauma such as ARDS, bronchospasm and paediatric patients, as having a preset pressure reduces the risk of over distension of the lungs. This method may also be more comfortable for the spontaneously breathing patient. Disadvantages include variation of tidal volume...
delivery and decrease in tidal volume delivery, particularly when lung condition deteriorates.

BREATH TYPE

Depending on the distribution of the ventilatory workload between the patient and the ventilator, 3 clinically different breath types can be provided during mechanical ventilation:

Mandatory

A machine-cycled breath that is triggered, limited and cycled by the ventilator. The machine performs the work of breathing with nil assistance from the patient. Mandatory breaths are also known as ‘controlled’ or ‘ventilator’ breaths.

Assisted

A machine-cycled breath that is triggered by the patient, limited by the ventilator and cycled by the ventilator. The patient performs only the work required to trigger the ventilator and the machine performs the rest of the work of breathing.

Spontaneous

This is a patient cycled breath that is triggered by the patient and usually limited and cycled by the patient. The patient performs all of the work of breathing with minimal support from the machine.

Modern Ventilator modes are a combination of all 3 breath types.

MODES

The term ‘mode’ describes a breath type and pattern of breath delivery during mechanical ventilation. The mode is determined by the following factors:

- Targeted control variable (volume or pressure)
- Type of breath (mandatory/controlled, assisted, spontaneous)
- Timing of breath delivery (mandatory ventilation, intermittent mandatory or spontaneous ventilation)

Many modes of ventilation have shorthand terms or acronyms used to describe the way a ventilator performs in a particular situation. These terms are often invented by the physicians, therapists or manufacturers who develop that particular type of ventilation. Unfortunately, little consistency exists in the way manufacturers name modes, therefore the terminology can be confusing and difficult to grasp. Basically there are 4 breath delivery techniques:
Controlled Mandatory Ventilation (CMV) also known as Intermittent Positive Pressure Ventilation (IPPV).

Synchronous Intermittent Positive Pressure Ventilation (SIPPV) - also known as IPPV assisted or as Assist Control.

Synchronous Intermittent Mandatory Ventilation (SIMV).

Spontaneous mode (also referred to as ‘CPAP’, continuous positive airway pressure, mode)

As previously mentioned, the names of modes vary according to the manufacturer of the ventilator. Each brand of ventilator may have different names for modes as well as using various combinations of modes. Some modes found on the Oxylog 2000, Oxylog 3000 and Drager Savina mechanical ventilators will be described further on in this learning package.

The Oxylog 2000 & 3000 are usually used as “time-cycled, volume-constant, pressure-limiting” ventilators. (Oxylog 2000 in IPPV mode only and Oxylog 3000 unless in PCV mode).
VENTILATORY PRESSURES

Airway Pressure (Paw)
For gas flow to occur there must be a positive pressure gradient. In spontaneous respiration, gas flow occurs due to the generation of a negative pressure in the alveoli relative to atmospheric or circuit pressure, as is the case during in CPAP mode. Inspiration is occurring below the horizontal line (Figure a).
Positive pressure mechanical ventilator breaths deliver flow and volume to the patient as a result of the development of a positive pressure gradient between the ventilator circuit and the patient’s alveoli (Figure b). In positive pressure breaths, inspiration is occurring above the horizontal line.

a.  

b.  

There are four pressures to be aware of in regards to mechanical ventilation. These are the:
- **Peak Inspiratory Pressure (PIP).** The peak pressure is the maximum pressure obtainable during active gas delivery.
- **Plateau Pressure.** The plateau pressure is defined as the end inspiratory pressure during a period of no gas flow.
- **Mean Airway Pressure.** The mean airway pressure is an average of the system pressure over the entire ventilatory period.
- **End Expiratory Pressure.** End expiratory pressure is the airway pressure at the termination of the expiratory phase and is normally equal to atmospheric pressure or the applied PEEP level.

During the delivery of a positive pressure breath, system pressure can be measured in a variety of locations, these include:
- internal to the ventilator - inspiratory / expiratory;
- at the Y piece of the ventilator circuit;
- at the airway opening; and
- at the carina - by applying the pressure monitoring line to a tracheal tube with an extra lumen.
The farther away the site of measurement is from the alveoli the greater the potential for difference between the pressure reading on the ventilator and the pressure in the alveoli. Increased resistance to airflow in the ventilator circuit, the endotracheal tube, or the patient’s conducting airway will be reflected in an increased difference between peak inspiratory and alveolar pressure.

Some mechanical ventilators use mbar as the unit of pressure measurement, however, by convention, cmH2O is the unit of measure usually referred to in Australia. At the pressure levels encountered during mechanical ventilation it is acceptable to equate mbar with cmH2O.
VENTILATOR SETTINGS

Despite age, gender or race, the patient’s set tidal volume (VT) is equivalent to 6-8mls/kg (IBW – ideal body weight).

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<td>215</td>
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<td>220</td>
<td>111.5</td>
</tr>
</tbody>
</table>
MONITORING & NURSING CONSIDERATIONS

Specific and careful monitoring of the mechanically ventilated patient is vital in maintaining patient comfort, safety and appropriate and effective care. (See policy ‘Care of the Invasively Ventilated Patient – Adult and Paediatric Patient’, found on Prompt).

All patients receiving Intermittent Positive Pressure Ventilation (IPPV) require:

- Constant 1:1 care by a CCRN
- Management by CCRN’s in close collaboration with an M.O.
- Naso-gastric or oro-gastric tube insertion.
- All artificial airways must be secured and checked regularly.
- Venous thromboembolism (VTE) prophylaxis.
- Stress ulcer prophylaxis.
- Sedation and analgesia as per “Sedation For Intubated Patients – Intensive Care Unit”.
- One hourly vital sign assessment and PRN.
- One hourly respiratory observations and PRN.
- One to two hourly assessment of sedation score.
- Assessment for sedation breaks.
- Head of bed elevation to 45° unless contraindicated.
- Aspiration of gastric tube as per “Nutritional Assessment and Management - Intensive Care Unit”.
- Assessment and documentation of tracheal tube cuff pressure at least eight hourly.
- Performance and documentation of mouth care at least 4 hourly.
- Daily assessment for extubation readiness.
- Use of closed-circuit suction devices (“in-line” suction), changed at least daily.
- Use of humidified inspired gas (via either HME or Fisher-Paykel® warm humidifier)
- Replacement of ventilator circuits if soiled.
- Daily assessment of patient bowel function.
- Monitoring of bloods, ABG’s, X-rays and scans.
- IDC and hourly fluid balance chart.

The nursing management of the ventilated patient in the emergency department has changed over the last decade due to increased length of stays in the ED. As a result the CCRN has become increasingly aware of the complex care needs of the ventilated patient. The nursing management of these patients involves monitoring, troubleshooting problems as they arise, evaluating response to ventilation and identifying any potential complications. The following section outlines the general management principles.
Monitoring
The primary goals of monitoring are to prevent complications, detect problems and monitor response to treatment. Monitoring essentials include assessment of the patient themselves, the ventilator and its settings and other equipment utilised. Regular physical assessment should be performed including lung auscultation, palpation and inspection. Assessing response to treatment includes evaluation of ventilation (e.g. WOB, pt-ventilator synchrony, acid-base status). Continual haemodynamic monitoring, including BP, HR, RR, SpO₂ and etCO₂ is performed, in addition to monitoring of fluid balance and sedation requirements. The ability to analyse ventilator waveforms is a further important skill when monitoring the patient, providing useful information on effectiveness of ventilation and aiding the CCRN in diagnosing problems. See section below for further information.

Sedation
The ventilated patient generally requires ongoing sedation and analgesia for anxiety, pain and relaxation. Inadequate sedation is counterproductive in the initiation of ventilation as it can lead to patient-ventilator asynchrony and ‘fighting’ the machine. In the ED, deeper sedation and paralysis is utilised in the initial period when diagnosis and assessment is taking place. In the long term, high doses of sedation for prolonged periods of time are associated with delayed weaning from ventilation. Paralysing agents are utilised together with sedation for induction, however, may not be required in an ongoing manner. A number of different agents are utilised depending on the indication for ventilation and on the patient’s haemodynamics and underlying medical condition. The primary agents used are either a morphine and midazolam infusion, which has the benefit of analgesic properties as well as sedation; or propofol, which is quicker acting, has a shorter half-life but has no analgesic properties. All sedatives and analgesics have adverse effects including producing hypotension.

Suctioning
Suctioning is performed according to the needs of each individual patient. Regular assessment of the patient will assist in determining their suctioning needs. Pre oxygenation with 100% O2 is required prior to suctioning the patient. There are various ways in which the patient may be suctioned, including the use of a y-suction catheter or an in-line suction unit which means the circuit is less interrupted. Although an important aspect of caring for the ventilated patient, suctioning can have complications, including exacerbating raised ICP and acting as a secretion stimulant.
**Fluid balance**
An IDC should be inserted in all ventilated patient unless contraindicated (e.g. urethral trauma, unstable pelvic fracture) and hourly urine measures recorded. A fluid balance chart should be commenced.

**Thermoregulation**
The ability to thermoregulate is lost in the paralysed and heavily sedated patient (loss of shivering reflex), therefore, it is important to ensure temperature is monitored and the patient is kept warm. This can be difficult in the ED particularly in the initial period when assessment is taking place. Warm blankets and fluids or the Bair Hugger can be used to keep the patient warm. An IDC that measures core temperature should be inserted unless MRI scan has been requested. Report urine output <0.5ml/kg (this often equates to <30ml/hr) or a trend of decreasing output.

**Patient positioning**
Positioning the ventilated patient in the ED is difficult due to constraints of equipment and undiagnosed injuries. As a general rule, the ventilated patient should be nursed head up at 30-45° degrees. This assists lung expansion, improves gas exchange, reduces the risk of aspiration and ventilator associated pneumonia and reduces ICP.

**Pressure area care**
It is important to consider position and equipment that may lead to pressure areas and prevent them if possible. For example, ETT position, invasive lines, hard collars and lying in the supine position for prolonged periods can all cause pressure areas. All clothing must be removed and sheets clean, smooth and dry underneath patients. Patient position should be altered each 2-4 hours if possible.

**Oral Care**
Patients with an ETT can either experience hypersalivation or dry mouth. Regular mouthcare reduces the incidence of ventilator acquired pneumonia. In the case of excessive saliva, regular suctioning is required to prevent pooling of secretions. Routine mouth care each 2-4 hours should be maintained, with either a toothbrush and toothpaste or with ‘Toothette®’ swabs that contain peroxide & sodium bicarbonate. Oral care must always be concluded with subglottal suctioning, above the ETT cuff, using a Y-suction catheter to remove pooled secretions.
**Eye Care**
Paralysis and sedation results in the loss of the blink reflex which can lead to corneal drying, abrasions and infection. Eye care (a normal saline eye toilet) should be performed every 2-4 hours including the administration of ‘artificial tears’, either eye drops or ointment and the use of a mild adhesive tape such as Micropore® to keep eyes closed if the patient is unconscious or sedated.

**Documentation**
Documentation of the care of the ventilated patient should follow a structured approach. When documenting ventilator settings, it is important to remember to document the set parameters (eg. ventilator tidal volume, ventilator mandatory breath rate, pressure support, PEEP) and from the *values screen* record the derived values (eg. tidal volume of spontaneous breaths, minute volume, peak inspiratory pressure). Other documentation includes changes and response to treatment, fluid balance and vital signs.

Other important aspects in the care of the ventilated patient include maintaining the dignity of the patient and including the family, which primarily involves good communication.

*Observe your patient constantly and track changes for early intervention as needed.*
COMPLICATIONS OF VENTILATION

There are a number of problems that arise both in the short and long term as a result of mechanical ventilation. It is important that the CCRN caring for the ventilated patient be aware of these and knows how to either prevent or identify them if they arise. The following outlines the primary complications of mechanical ventilation (Pierce, 2007).

- Barotrauma - excessive pressure in the alveoli causing injury and rupture. Air from the alveoli escapes into the:
  - pleura (pneumothorax or tension pneumothorax)
  - mediastinum (pneumomediastinum)
  - pericardium (pneumopericardium)
  - under the skin (subcutaneous emphysema)

An immediately life-threatening complication - tension pneumothorax - may arise. All clinicians caring for mechanically ventilated patients must be able to recognise this emergency.

Manifestations of tension pneumothorax:
- Rapid deterioration
- \( HR, BP, VT, PIP, hypoxia \)
- Extreme agitation if conscious
- Decreased breath sounds
- Hyper resonance to percussion
- (tracheal deviation)
- (distended neck veins)
- Cardiac arrest

Interventions DRABCD
- Disconnect from ventilator
- Laerdal bag, \( FiO2=1.0 \)
- Call code blue
- M.O.- Immediate needle thoracostomy (wide bore jelco, 2nd intercostal space midclavicular line or between mid/ant axilla line 5th intercostal space) then insert chest tube

- Volutrauma – lung injury caused by end-inspiratory over distension. It results in an increase in alveolar capillary membrane permeability, pulmonary oedema, and the accumulation of neutrophils and proteins in the interstitial and alveolar spaces.

- Gas trapping (auto PEEP) - the spontaneous development of PEEP as a result of insufficient expiratory time; caused by rapid respiratory rate, airflow obstruction, for example.
• Oxygen toxicity - prolonged exposure to high oxygen levels can lead to pulmonary parenchymal changes. Symptoms of oxygen toxicity often mimic Adult Respiratory Distress Syndrome (ARDS).

• Cardiovascular - raised intra-thoracic pressure leads to reduced venous return & cardiac output, resulting in hypotension.

• Gastrointestinal
  o Gastritis/ ulcers
  o Decreased gastric blood flow → ischaemia, GI bleeds

• Neurological
  o Raised Intracranial Pressure (ICP) - increases in superior vena cava and jugular vein pressure as a result of positive pressure ventilation and PEEP can reduce cerebral venous outflow, leading to raised ICP

• Other (immediate intervention required)
  o Intubation of right main bronchus (detected clinically or on x-ray)
  o ETT out of position/unplanned extubation
  o Incorrect settings
  o Mis-assembly of circuits or parts
  o Inadequate Ventilation

• Other (long term)
  o Tracheal damage/ necrosis
  o Acid-base disturbances
  o Infection
  o Ventilator dependency
  o Pressure areas
  o Alterations in fluid balance (fluid retention)

The immediate complication that can arise is the drop in cardiac output from the vasodilatory affects of intubation drugs and from the effects of positive pressure on venous return, resulting in hypotension!!
TROUBLESHOOTING MECHANICAL VENTILATION

When caring for a ventilated patient, it is important to have an understanding of the potential problems that may arise and how to troubleshoot them. The majority of problems that require troubleshooting arise from issues concerning the patient (e.g. increased airway pressures) or problems with equipment (e.g. disconnection, leaks).

Ventilator Alarms

Appropriate use of ventilator alarms is essential for patient safety. It is important that when you set the ventilator up, you also ensure the appropriate alarm settings are set. If the ventilator alarms, always start with an assessment of the patient and work your way back to the machine. There are a number of ventilator alarms which you must be aware of and know how to troubleshoot if they arise.

Low Pressure Alarm (PAW LOW)

A low pressure alarm indicates loss of circuit pressure and is activated when the machine no longer detects a pressure difference of more than 5–mbar between inspiratory pressure (Pinsp) and expiratory pressure.

• Causes:
  ▪ Patient: expiratory leak (chest tube, inadequately inflated ETT cuff, NGT in trachea)
  ▪ Ventilator: incorrect ventilator settings (Pinsp, RR), inspiratory leak - circuit disconnected, loose connections, faulty flow sensor

• How to identify it:
  ▪ ↓ Ppeak/ Pinsp/ VT

• Management:
  ▪ Check pt, circuit, ventilator
  ▪ Correct cause of leak
  ▪ Adjust ventilator settings (↑Pinsp, VT)

High Pressure alarm (PAW HIGH)
A high pressure alarm indicates that inspiratory pressures have exceeded the set pressure limit (Pinsp/ P\text{MAX}). This is also referred to as high PIP’s (peak inspiratory pressures) or Ppeak.

- **Causes:**
  - Patient: reduced compliance - resp. disease, consolidation, oedema, tension pneumothorax, lung collapse, abdominal distension; patient-ventilator asynchrony (‘fighting’ the ventilator), pt. coughing, airway obstruction
  - Ventilator: incorrect settings (↑VT, high flow - I:E ratio), kinked circuit tubing, build up of fluid in circuit, unsuitably low high pressure alarm limit.

- **How to identify it:**
  - ↑ Ppeak/ Pplat (look at waveforms and figures)

- **Management:**
  - Check patient for air entry, cough, ETT blockage, agitation/sedation levels, consider suctioning
  - Check ventilator for appropriate settings & alarm limit, circuit for patency & condensation
  - Consider further sedation
  - Adjust ventilator- ↓ VT, adjust flow adjust alarm limit if unsuitable
  - Consider pressure controlled mode, spontaneous (CPAP) mode

A PAW HIGH alarm will be activated when Pmax has been breached; once activated the ventilator terminates the breath. The pressure max alarm limit should be set a 10cm H2O above the patient’s average peak pressure (Ppeak). **Remember that patients with airway pressures > 40cm H2O are at risk of barotrauma.**
**High minute volume (HIGH MV)**

A high MV alarm indicates that the MV has exceeded that of the upper MV alarm limit. Set the high MV alarm limit 1-2L/min above the acceptable MV. This is often approximately 10-15% above the expected MV.

**Causes:**
- Patient: Hyperventilation due to pain or anxiety, acid-base disturbance; pt-ventilator asynchrony, gas trapping, neurological pathology
- Ventilator: Inappropriate settings- high RR/ VT

**How to identify it:**
- High VT/VTe, (VTe refers to tidal volume exhaled) increase in spontaneous breath rate, check flow waveform for auto-PEEP

**Management:**
- Check pt (consider analgesia, sedation), assess ABG
- Adjust settings (↓ VT/ RR, if auto-PEEP- ↓ PEEP, ↑ exp time)
- Change mode (consider PCV, CPAP)
- Assess suitability of MV alarm limit

**Low minute volume (LOW MV)**

A low MV alarm indicates the MV has failed to reach the set limit of the lower MV alarm parameter. The low MV alarm limit is usually set approx 500ml-1L below the acceptable MV or 10% below the expected MV.

**Causes:**
- Patient: PCV mode- inadequate Pinsp/RR, ↓ compliance (↑ PIP), fatigue
- Patient : CPAP/spont mode- inadequate pressure support (PS), fatigue
- Patient : sedation, neurological pathology, increased intrathoracic pressures limiting MV eg- pneumothorax
- Ventilator: disconnection, air leak, high pressure alarm reached causing ‘dumping’ or termination of VT, insufficient flow

**How to identify it:**
- PAW HIGH alarm (if in PCV and Pinsp reached)
- Decreased RR and or VT
- Increased RR/ use of accessory muscles
- Audible leak
- Check flow waveforms

- Management:
  - Check pt, assess for muscle fatigue (↑ RR, irregular breathing pattern, accessory muscle use), assess for over sedation
  - Fix leak/ reconnect
  - Increase flow (I:E ratio)
  - Adjust ventilator settings (↑ RR VT/Pinsp/PS), change mode

A LOW MV alarm is activated when the minute volume drops below the set minimum value (approx. 10% below the expected minute volume). If the patient is being ventilated at 12 bpm with a tidal volume of 500mls the expected MV is 6.0L (MV = TV x RR). Therefore, the low minute ventilation alarm should be set at approximately 5.0L.

If you are ever in doubt or are unable to troubleshoot a problem, disconnect the patient, hand ventilate and get help!!

High frequency

When setting up the ventilator for use, Frequency is a value that needs to be set. The frequency should represent a normal respiratory rate for a patient’s age and medical condition (this can be altered to allow for CO₂ retention and expulsion and to alter tidal volumes). The high frequency alarm should be set when each new patient uses the ventilator. If the patient’s frequency exceeds that set in the high frequency alarm, an alarm will sound. Check the patient and ventilator to confirm this, and troubleshoot the patient and ventilator to establish why this alarm was triggered in order to correct it.

Apnoea

ED’s ventilators have apnoea alarms. When an apnoea occurs, the device outputs an alarm signal and starts volume-controlled mandatory ventilation with the parameters frequency
Apnoea ventilation can be overridden by pressing the “alarm reset” button, this will revert ventilation to the previously selected mode and selected values.

**EMERGENCY VENTILATORS**

In the Emergency Department at Bendigo Health, we have 3 ventilators: Oxylog 2000, Oxylog 3000 and Drager Savina.

The Oxylog 2000 has the following modes:

- Intermittent Positive Pressure Ventilation (IPPV)
- Synchronised Intermittent Positive Pressure Ventilation (SIPPV)
- Synchronised Intermittent Mandatory Ventilation (SIMV)
- Continuous Positive Airway Pressure (CPAP)
- Adjunct:
  - Positive End Expiratory Pressure (PEEP)

The Oxylog 3000, as with the 2000, it has IPPV and SIMV. The Oxylog 3000 has a mode called IPPVassist that is the equivalent of the SIPPV mode on the Oxylog 2000. In addition the Oxylog 3000 has PCV mode. The Oxylog 3000 has the same adjuncts as the 2000. In addition, it has Pressure Support (PS), as an adjunct. Pressure Support is sometimes termed Assisted Spontaneous Breaths (ASB) by the manufacturer.

<table>
<thead>
<tr>
<th>FiO2</th>
<th>The fraction of inspired oxygen</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flow</td>
<td>This is the peak inspiratory flow rate of gas during delivery of the breath. Usually set around 40 – 60 L/min but can be adjusted according to the patient breath pattern. If a patient has air hunger or airways disease this flow may need to be increased in ‘volume control’</td>
</tr>
<tr>
<td>Term</td>
<td>Description</td>
</tr>
<tr>
<td>------</td>
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</tr>
<tr>
<td><strong>Minute Volume (MV)</strong></td>
<td>The volume of gas expired over one minute. Calculated as RR x VT = MV. Also known as minute ventilation.</td>
</tr>
<tr>
<td><strong>I:E Ratio</strong></td>
<td>The ratio of inspiratory time to expiratory time. Usually on a fully ventilated patient this is approximately 1:2. On the Oxylog 3000 this is changed by manipulating the inspiratory time setting in PCV.</td>
</tr>
<tr>
<td><strong>Inspiratory pressure (Pinsp)</strong></td>
<td>The set value of the target pressure level in PCV breaths.</td>
</tr>
<tr>
<td><strong>Inspiratory time (Tinsp)</strong></td>
<td>The set value of the target pressure level in PCV breaths. Increasing the inspiratory time may assist in recruiting alveoli and improving oxygenation. In pressure controlled ventilation the inspiratory time is sometimes extended to increase the tidal volume. Extending the inspiratory time can cause haemodynamic compromise and be a source of patient discomfort (patient ventilator synchrony). The normal inspiratory time is 0.8 – 1.2 seconds.</td>
</tr>
<tr>
<td><strong>Peak Inspiratory Pressure (PIP)</strong></td>
<td>The peak or highest pressure achieved during inspiration (also called peak airway pressure - Ppeak).</td>
</tr>
<tr>
<td><strong>Plateau Pressure</strong></td>
<td>The end inspiratory pressure during the period of no gas flow. It reflects lung and chest wall compliance. It should not exceed 35 cmH₂O as higher levels can induce lung injury.</td>
</tr>
<tr>
<td><strong>Plateau Time (Tplat)</strong></td>
<td>Refers to a potential inspiratory pause at the end of inspiration.</td>
</tr>
<tr>
<td><strong>Positive End Expiratory Pressure (PEEP)</strong></td>
<td>This is the pressure remaining in the alveoli to keep them open at the end of expiration.</td>
</tr>
<tr>
<td><strong>Pressure Max (Pmax)</strong></td>
<td>The maximum pressure the ventilator can generate to deliver a tidal volume. When the pressure limit is reached the ventilatory breath is terminated.</td>
</tr>
<tr>
<td><strong>Pressure Support (PS)</strong></td>
<td>The level of assistance the patient receives during a spontaneous breath to overcome dead space between the patient and ventilator. At the onset of inspiration, the pre-selected amount of PS is delivered then held constant throughout inspiration, which promotes the flow of gas into the lungs. PS is also a mode of ventilation that is used for spontaneously breathing patients.</td>
</tr>
<tr>
<td><strong>Ramp</strong></td>
<td>A comfort feature in NIPPV (Non-invasive positive pressure ventilation) that allows an incremental rise in pressures over a set period of time.</td>
</tr>
<tr>
<td><strong>Respiratory Rate</strong></td>
<td>The frequency of breaths set per minute that will be delivered by the modes.</td>
</tr>
<tr>
<td><strong>(RR)</strong></td>
<td>ventilator</td>
</tr>
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</tr>
<tr>
<td><strong>Rise Time</strong></td>
<td>Sets how quickly the inspiratory pressure rises to achieve the target inspiratory pressure in pressure control and pressure support ventilation. The higher the value the quicker the target pressure is achieved. 50% is the default value.</td>
</tr>
<tr>
<td><strong>Tidal volume (VT)</strong></td>
<td>The volume of breath delivered to the patient. It is normally set at 5-7mls/kg IBW.</td>
</tr>
</tbody>
</table>
OXYLOG 2000

- Ventilation modes IPPV, SIPPV, SIMV, CPAP
- Ventilation frequency 5 to 40 breaths/min
- Tidal volume VT 0.1 to 1.5 L
- Breath-time ratio 1:3 to 2:1
- Inspiratory pressure limitation 20 to 60 mbar
- PEEP 1 to 15 mbar
- Minute volume (for TI:TE = 1:1.5) 1 to 25 L/min
- O2 concentration 100% or 60% by vol. O2

IPPV (Intermittent Positive Pressure Ventilation)

- Volume-Cycled/Mandatory Ventilation

This is a method of ventilation in which the ventilator delivers a preset number of breaths (rate) of a preset tidal volume, regardless of the patient’s attempts to breathe on their own. Because this is a mandatory/control mode, it demands the use of muscle relaxants and sedatives to maintain unconsciousness, as it prevents the patient breathing spontaneously.

IPPV is indicated for use in patients with apnoea caused by a central nervous system, or neuromuscular dysfunction (such as paralysis or severe brain trauma), drug overdose and status asthmaticus or medically induced unconsciousness for control of ventilation.

Advantages of IPPV include decreasing the work of breathing, decreasing oxygen consumption and carbon dioxide production because it completely controls the patient’s breathing.

Decreased venous return and cardiac output (due to constant increased intrathoracic pressure), respiratory muscle atrophy (causing difficulty in weaning patient), the danger of
barotrauma due to increased intrathoracic pressure and stacked breaths (a ventilator breath on top of a spontaneous breath) are all disadvantages of IPPV. IPPV also doesn’t allow the patient to compensate when CO2 levels increase.

**SIPPV (Synchronous Intermittent Positive Pressure Ventilation)**

- Volume-cycled/Assisted Ventilation

SIPPV is a method of ventilation in which the ventilator delivers a preset number of breaths (rate) of a preset tidal volume. Between the machine initiated breaths, the patient may trigger spontaneous breaths. When the patient begins to take a breath, the negative inspiratory effort triggers ventilation and the machine delivers a breath of the preset tidal volume. The breaths are over and above the set rate. This mode of ventilation augments breathing without completely controlling it.

Advantages of SIPPV include that it makes weaning easier by preventing muscle atrophy and encourages the patient’s response to CO2 levels.

Disadvantages include decreased venous return and cardiac output due to constant increased intrathoracic pressure and possible barotrauma due to increased intrathoracic pressures. There is a risk of hyperventilation as all breaths will be of the set volume.

**SIMV (Synchronous Intermittent Mandatory Ventilation)**

- Volume-Cycled or Pressure-Cycled/Assisted Ventilation

In this mode, a respiratory rate and tidal volume (or inspiratory pressure) is dialled up. The patient’s own breaths are monitored by the ventilator. If the patient breathes spontaneously during the ventilatory cycle, then the ventilator delivers the breath synchronously with the patient’s effort (and the patient may even obtain a tidal volume larger than the set volume). If the patient does not breathe, then a mandatory breath is delivered. If the patient’s respiratory rate exceeds that dialled up on the ventilator, then the excess breaths are at the patient’s chosen volume. These excess spontaneous breaths should be assisted by the use of the Pressure Support feature, to prevent high respiratory workload.

The advantage of SIMV is that it prevents muscle atrophy, facilitating weaning by the graduated exercising of respiratory muscles. It allows the patient’s response to changes in CO2 levels by changing their respiratory rate. SIMV reduces the need for large amounts of sedation and muscle relaxants allowing the patient some control and decreasing anxiety. A slower ventilator rate is possible, which decreases the risk of barotrauma. SIMV provides lower intrathoracic pressures, augments the thoracic pump mechanism aiding in venous return, ensuring a less depressed cardiac output and allows for higher levels of PEEP.
Gas consumption
Control approx 1.0 L/min
MV (Air Mix) approx. 50% of the set MV
MV (No Air Mix) approx. 100% of the set MV

Display
The following parameters can be invoked in the display depending on the ventilation mode

- In IPPV/SIPPV - MV, FLOW, PEAK, MEAN, PEEP, Tinsp., frequency
- In SIMV - MV, CPAP, FLOW, PEAK, MEAN, PEEP, frequency, Vt exp.
- In CPAP - frequency, VT exp.

Alarms
- Supply pressure low Warning when supply pressure drops below approx. 2.0 bar in IPPV, SIPPV or SIMV mode
- Paw high Set via Pmax knob. Warning when set value for Pmax is reached
- Paw low Warning when a pressure difference > 10 mbar is not built up over a time of > 20 s in IPPV, SIPPV or SIMV mode
- Leakage Warning when the expiratory tidal volume drops below 60% of the inspiratory tidal volume. (The leakage alarm is not active in CPAP mode).
- Apnoea Alarm is activated if no change in breathing phases is detected within a 25s time span.
- Frequency high Warning when the measured frequency exceeds 50% of the set frequency in SIPPV mode

Power supply
230 VAC from mains power pack. Internal supply voltage NiCd rechargeable battery with 7.2 V/1000 mAh. Operation time Max. 6 hours (with rechargeable NiCD battery). The duration of operation on battery will reduce when high respiratory rates, tidal volumes and PEEP is used. Charge time with quick charge 8 hours, then switch to trickle charge.

Preparation & Assembly of the Oxylog 2000:
It is essential that the circuit is correctly put together. Even if circuit is assembled incorrectly, the self test may still say “OK”.

1. The rubber disc in the housing must not be damaged or bent, otherwise the valve will not work properly.
2. Place diaphragm in breathing valve – ensure this is inserted correctly.  
3. Fit cover and turn approx. 90 degrees clockwise, this will lock the cover into place.  
4. Plug flow sensor into breathing valve; note preferred position (groove).  
5. Push angled connector onto flow valve.  

NB: when using a bacterial filter always connect the filter to the angled connector (at end of picture 5).  

6. Connect ventilation hose to breathing valve. BEWARE: do not use electrically conductive hoses, as these can endanger both ventilator and the operator during defibrillation.  

7. Connect flow measuring hoses to sockets on flow sensor (hoses are different dimensions and will only fit on their correct sockets.)
Checking the Oxylog 2000:

The Oxylog requires checking once every shift, each time the ventilation valve is changed and each time care/assembly is carried out.

- Visually check the circuit to ensure correct assembly and that the circuit is not damaged and is clean.
- Connect the test lung. The test lung is to be connected to the Y-piece; a size 7 catheter (the metal piece we fit into the test lung to simulate the resistance of the airways) and a 2L breathing bag/test lung to simulate compliance.
- Set Vt knob to 0.5L
- Set Freq knob to 12/min
- Set Ti:Te knob to 1:2
- Set Pmax knob to 60mbar
- Set switch for ventilation modes to IPPV
- Set on/off switch to on (I)
- The ventilator carries out an electrical and pneumatic self test.
- NB: “TEST OK” may appear even when the circuit is incorrectly assembled.
- The red alarm indicator and the green LED “DC power available” will light up during test phase, the alarm tone will also beep twice
- The self test takes approximately 6 seconds to complete.

Once check is complete, leave Oxylog 2000 on and select the settings you wish to use. Connect patient. Once patient connected to the Oxylog, observe the chest wall movements for synchrony with the ventilator, listen for equal air entry in right and left lungs, monitor pulse oximetry and ETCO2 for adequate oxygenation. Comprehensive respiratory assessment and documentation is then required.

Oxylog 2000- Important Points:

Apnoea in CPAP mode- After 25 sec of patient apnoea, an alarm will sound. The ventilator will NOT provide apnoea backup breaths. The operator MUST alter the dialled up frequency (eg. to RR=15) to ensure the apnoeic patient is delivered breaths.

FiO2 in CPAP mode- Regardless of the selected FiO2, the patient will receive 100% oxygen from the ventilator in CPAP mode.
Areas of use
Mobile use for emergency medical care or primary care of emergency patients:
- During transport in emergency rescue vehicles or by helicopter,
- In accident and emergency departments, in the recovery room.
Mobile use for secondary transfers:
- During transfer by road or air
- When moving ventilated patients around the hospital.

Intended Medical Use
Oxylog® 3000 is a time-cycled, volume-constant and pressure controlled emergency and transport ventilator for patients with a tidal volume from 50mL upwards.

Ventilation modes
The Oxylog 3000 has four main modes for breath delivery:

- IPPV/IPPVAssist (CMV/CMVAssist*)
  Intermittent Positive Pressure Ventilation
  Controlled and assisted volume-constant ventilation with PEEP for CPPV.
- SIMV/ASB (SIMV/PS)
  Synchronized Intermittent Mandatory Ventilation, with Pressure Support
  Procedure for weaning patients off the ventilator after they have started spontaneous breathing, with adjustable pressure support during spontaneous breathing.
- CPAP/ASB (CPAP/PS)
  Continuous Positive Airway Pressure
  Spontaneous breathing with positive airway pressure and adjustable pressure support.
- PCV+/PS* (BIPAP/ASB**)
  Pressure-controlled ventilation combined with spontaneous breathing during the complete breathing cycle, and adjustable pressure support above the CPAP level.
  Biphasic Positive Airway Pressure- the Oxylog also uses the term ‘BIPAP’ to describe PCV.
Special modes

- Apnoea Ventilation
  In CPAP mode, the Oxylog 3000 will switch over automatically to volume-controlled mandatory ventilation, if breathing stops for the apnoea period set by the operator. The operator must also set the apnoea VT and RR when commencing ventilation.
- Non-invasive ventilation (NIV)
  The Oxylog 3000 can be used for mask ventilation with leakage compensation. However, specific circuits and masks approved for use with the Oxylog 3000 must be available.

Monitoring

- Airway pressure Paw
- Expiratory minute volume MV
- Apnoea
- Rapid shallow breathing: High frequency alarm

The Oxylog 3000 is able to deliver either volume controlled or pressure controlled breaths. In volume controlled ventilation (SIMV/CMV/IPPV/SIPPV), a desired tidal volume is delivered at a specific flow; however the airway pressure generated may be higher than is desirable. In pressure controlled modes (PCV/CPAP), flow occurs until a preset peak pressure is met over a specified inspiratory period, but the tidal volume may be lower than that desired.

Synchronised Intermittent Mandatory Ventilation (SIMV)

SIMV is a mode of ventilation that is the most commonly used mode in the ED; it is also the default setting on the Oxylog 3000. The Oxylog 3000 only delivers volume controlled breaths in SIMV mode. It delivers a preset number of breaths (frequency) at a set tidal volume. If the patient generates a spontaneous breath, the ventilator detects this and synchronises the delivery of mandatory breaths with the patient’s spontaneous effort. Spontaneous breaths are ‘supported’ to a preset pressure level (pressure support): the patient determines the rate, the duration of inspiration and the tidal volume. When SIMV is used, the patient can receive three different types of breath:

- Controlled (Mandatory) breaths.
- Assisted (synchronized) breaths.
- Spontaneous breaths, which can be pressure supported.

When do we use SIMV?

- When the patient cannot sustain spontaneous ventilation for long periods (e.g. paralysis, head injury)
- When we want patients to rest their own respiratory muscles and assist maintaining $\text{PaCO}_2$ (chronic lung diseases)
- Weaning from mechanical ventilation
Advantage of this mode:

- There is a guaranteed minimum volume with each mandatory breath
- The patient can generate spontaneous breaths without a preset tidal volume
- There is less chance of atrophy of respiratory muscles as the patient participates in ventilation
- There is less haemodynamic compromise as ventilation occurs at a lower mean airway pressure compared with IPPV.

SIMV settings

- Rate (can influence the I:E ratio/ Flow rate)
- Tidal Volume (can influence the I:E ratio/ Flow rate)
- Time (influences the I:E ratio/ Flow rate)
- Pressure support
- PEEP
- Pmax
- Trigger
- FiO₂

**Controlled Mandatory Ventilation (CMV)**

CMV is a form of volume controlled ventilation (not often used), whereby a set amount of breaths are delivered at a set volume. The ventilator initiates inspiration, controls tidal
volume and rate. If the patient initiates a breath, the ventilator does not recognise it and continues to deliver its mandatory breaths. The main advantage of CMV is that the patient receives guaranteed minute ventilation. The disadvantage of this mode is that the patient cannot control their breathing pattern as it does not recognise or allow for, or support spontaneous effort/breaths.

When do we use CMV?

- In those patients where paralysis and deep sedation are indicated i.e. head injury
- For those patients with no respiratory effort (high-level spinal cord lesion, head injury, Guillain-Barre syndrome)

CMV settings:

- Rate
- Tidal Volume
- TinSp (adjusts the I:E ratio/Flow rate)
- PEEP
- Pmax
- FiO₂
**Continuous positive airway pressure (CPAP)**

This is a purely spontaneous mode, i.e. the patient has to be able to generate their own breaths and sufficient volume. Both inspiration and expiration are supported to a preset pressure (PS and PEEP). If the patient becomes apnoeic and apnoea ventilation is activated, the ventilator automatically switches into SIMV mode initiating volume-controlled ventilation. CPAP provides positive pressure at the end of exhalation which assists in preventing alveolar collapse, improves the functional residual capacity, enhances oxygenation and assists in splinting airways open which reduces airway resistance. CPAP and PEEP are identical in their physiological effects - CPAP is the term used in a spontaneously breathing patient to describe this mode and PEEP is the term used to describe the pressure at end of expiration. ‘CPAP’ is also a term used to describe a form of non-invasive positive pressure ventilation, where a mask is used.

When is it used?

- On the spontaneously breathing patient who requires support (e.g. COPD, atelectasis)
- During spontaneous breathing trials
- As a weaning mode prior to extubation

**CPAP settings:**

- PEEP
- Pressure support
- Apnoea alarm
- Pmax
- FiO₂

Apnoea ventilation can only be activated when ‘invasive ventilation’ INV is selected; it will not work in NIV mode!!

**Pressure control ventilation (PCV)**

This form of ventilation has a set target inspiratory pressure (Pinsp), a variable volume and flow rate, a set inspiratory time and rate. The tidal volume varies according to the set inspiratory pressure (Pinsp), inspiratory time (Tinsp) and patient conditions which effect factors such as compliance, resistance, and inspiratory effort. The pressure is maintained for the duration of inspiration. Therefore, longer inspiratory times lead to higher mean airway pressures and greater tidal volumes. In this mode, the patient is able to cycle
between controlled and spontaneous breaths, with synchronisation of pressure levels. Spontaneous breaths can also be supported with pressure support. The main advantage of pressure controlled ventilation is that it reduces pulmonary barotrauma. Reduced airway pressures are often achieved through limiting the application of inspiratory pressure.

When is it used?

- Paediatrics
- Patients with non-compliant lungs and high airway pressures and poor oxygenation, e.g. chronic lung disease

PCV settings:

- Rate
- Tinsp
- Pinsp
- PEEP
- Pmax
- PS
- Trigger
- FiO₂

It is important that all settings be decided and confirmed prior to connecting the ventilator to the patient!!
**Instructions for use**

**NB:** A device check should be performed daily and after a new circuit is connected.

1. To switch on, briefly press the »O« key.
2. To switch off, hold down the »O« key for about 3 seconds, to confirm the switch-off prompt, press rotary knob.
3. Keys for selecting the ventilation modes:
   - IPPV (CMV), SIMV, CPAP
   - BIPAP (PCV+)*
   - SIMV/ASB (SIMV/PS)*
   - CPAP/ASB (CPAP/PS)*
   - BIPAP/ASB (PCV+/PS)*

The operating concept takes into account the various purposes for which the ventilator is used. When configured accordingly, Oxylog 3000 starts in SIMV with user-configured starting values for I:E (1:1.5 as default setting) and PEEP (5 mbar as default setting).

4. The most important ventilation parameters are set with the aid of the controls below the screen:
   - Tidal volume **VT** [mL],
   - Ventilation frequency **Freq.** [1/min],
   - Max. inspiratory pressure **Pmax** [mbar],
   - **O2** concentration **O2** [%]

5. Other parameters can be set in the screen window via the central rotary knob when selected accordingly (e.g. TinSp, PEEP, Δ ASB (Pressure Support), Pinsp).

To select parameter = turn rotary knob
To activate parameter = press rotary knob
To set value = turn rotary knob
To confirm value = press rotary knob

**Connecting a patient to the Oxylog 3000**

- Ensure ventilator attached to oxygen.
- Check the assembly of the circuit as described for the Oxylog 2000.
- Connect a blue test lung containing a narrow insert, to the elbow of the circuit.
- Turn the ventilator ‘on’.
- A self test will commence.
- During the self test, quickly press the rotary knob to enter a ‘device check and configuration’.
- Select ‘device check’ and follow the instructions. Set the tidal volume, RR, Pmax and FiO2.
- The ventilator should inflate the test lung at regular intervals and a pressure waveform should appear on the screen.
- Once satisfied with the ventilator function, Set the mode and parameters suitable for the patient
• Remove the test lung, insert a HME and connect elbow to the ETT.
• Use the values key to scroll through the measured parameters.
• If you need to alter any values, use the settings key. Select your choice by turning the rotary knob and press the rotary knob to confirm your change.
• To set and alter alarms, use the alarm key. Select your choice by turning the rotary knob and then pressing the rotary knob to confirm the change.
• Once patient connected to Oxylog and desired settings have set, observe the chest wall movements for synchrony with ventilator, listen for equal air entry in right and left lungs, monitor pulse oximetry and ETCO2 for adequate oxygenation.
• Perform a comprehensive patient assessment and document care.

NB: For full product information and instructions for use, please refer to Oxylog 3000 instructions for use manual.
Areas of Use

The Savina ventilator is most commonly used in an ICU setting, but is compact and mobile enough to suit an Emergency Department. This ventilator is on wheels and has a large O2 cylinder attached to allow for transport around the hospital.

Ventilation Modes

To activate a ventilation mode, hold down the appropriate mode key for about 3 seconds or alternatively briefly press the appropriate ventilation mode key and then press the rotary knob to confirm the change.

CMV AC – referred to in the manual as “IPPV Assist”

SIMV – Synchronous Intermittent Mandatory Ventilation

CPAP PS – Continuous Positive Airway Pressure with Pressure Support

PCV+ - Pressure Controlled Ventilation plus (as referred to in the manual) also known as Bi-Phasic Positive Airway Pressure (BIPAP)
CMV (Controlled Mandatory Ventilation)

- Volume-Cycled or Pressure Cycled/Mandatory Ventilation

In this mode, the ventilator delivers a preset number of breaths (RR) at a preset tidal volume or pressure. CMV is not sensitive to the patient’s spontaneous breathing effort and will work independently to those efforts regardless of the underlying breathing pattern. It is difficult for a patient to take their own breath. CMV does not allow for patient spontaneous breathing and can contribute to barotrauma. CMV is not commonly used due to its asynchronous nature. The user manual refers to CMV as IPPV assist.

SIMV (Synchronous Intermittent Mechanical Ventilation)

- Volume-Cycled or Pressure-Cycled/Assisted Ventilation

In this mode, a respiratory rate and tidal volume or inspiratory pressure is dialled up. The patient’s own breaths are monitored by the ventilator. If the patient breathes spontaneously during the ventilatory cycle, then the ventilator delivers the breath synchronously with the patient’s effort (and the patient may even obtain a tidal volume larger than the set volume). If the patient does not breathe, then a mandatory breath is delivered. If the patient’s respiratory rate exceeds that dialled up on the ventilator, then the excess breaths are at the patient’s chosen volume. These excess spontaneous breaths should be assisted by the use of the Pressure Support feature, to prevent high respiratory workload.

The tidal volume of the spontaneous breaths is dependent upon the muscular respiratory effort that the patient is able to generate. The advantage of SIMV is that it prevents muscle atrophy, facilitating weaning by the graduated exercising of respiratory muscles. It allows the patient’s response to changes in CO2 levels by changing their respiratory rate. SIMV reduces the need for large amounts of sedation and muscle relaxants allowing the patient some control and decreasing anxiety. A slower ventilator rate is possible, which decreases the risk of barotrauma. SIMV provides lower intrathoracic pressures, augments the thoracic pump mechanism aiding in venous return, ensuring a less depressed cardiac output and allows for higher levels of PEEP.

Pressure Support Ventilation (PSV/ASB)

- Pressure-Cycled/Spontaneous Ventilation
Although PEEP assists in ventilation in the expiratory phase by effectively splinting the alveoli open and reducing the workload required to inflate them, it has little effect on the inspiratory phase.

Pressure Support Ventilation (PSV) was developed to assist in the inspired phase of spontaneous ventilation. Pressure Support is also known as Assisted Spontaneous Breaths (ASB). The patient initiates the breath, triggering ventilatory assistance. The airway is subsequently pressurized to a preset level (e.g., between 5 and 30 cmH2O). Providing additional pressure during the triggered breath allows for adequate inspiratory volumes.

As a standalone mode, PSV augments the patient’s spontaneous respiratory activity by delivering a preset amount of inspiratory pressure. Delivery of this preset amount of pressure is triggered by the patients’ inspiratory effort and is held constant throughout inspiration. PSV assists spontaneous ventilation by promoting gas flow into the lungs, reducing WOB, reducing respiratory muscle wastage and augmenting inadequate spontaneous tidal volume to the desired degree (to the preset maximum airway pressure selected). Tidal volumes, however, are variable; therefore alveolar ventilation is not guaranteed. This mode is useful when weaning a patient from mechanical ventilation and when the patient is on long term mechanical ventilation.

Pressure support ventilation aims to overcome reduced pulmonary compliance and increased airways resistance (bronchospasm). Most patients can be ventilated in this way as long as they maintain the ability to initiate their own breaths.

PSV is usually used in addition to mandatory breathing, such that any spontaneous breaths are pressure supported.

**CPAP** (Continuous Positive Airway Pressure)

- Pressure-cycled/Spontaneous Ventilation

This mode allows a positive airway pressure to be set to prevent alveolar collapse, so improving oxygenation in settings such as acute pulmonary oedema. Patients must be spontaneously breathing to use this mode. A constant pressure is maintained in the ventilator circuit and therefore, in the patients’ lungs. The aim is to increase the baseline pressure in the lungs throughout ventilation. This holds the many alveoli open that would normally collapse and increases the volume of lung used in gas exchange. This decreases the normal physiological shunt (that is the amount of blood that passes passively through the lungs without being involved in gas exchange). CPAP is similar to PEEP in effect, but PEEP is only one of two pressures that occur in ventilation (mechanical or spontaneous), being relevant only at end expiration. Whereas CPAP aims to maintain a single constant pressure on inspiration and expiration.
**Preparation: Assembling**

(See Savina – Instructions for use Manual for further information and illustrations)

**Mounting the expiration valve:**

1. Attach diaphragm to expiration valve.
2. Attach rubber bushing to port.

**Inserting the expiration valve:**

3. Turn knurled sleeve to left as far as it will go.
4. Insert expiration valve.
5. Fit container to water trap.

**Fitting the flow sensor:**

1. Push the socket to the left as far as it will go.
2. Insert the flow sensor, with the probe facing towards the ventilator, into the mounting and push it into the socket as far as it will go.
3. Push the flow sensor to the right into the rubber lip of the expiration valve as far as it will go.
4. Push down the flow sensor cover.

**Fitting the bacterial filter:**
An inspiratory bacterial filter should always be used as it protects the patient from contamination from the ambient air drawn in.

Push the bacterial filter onto the inspiration port.

Note: The use of expiratory bacterial filters on this ventilator is not recommended as it increases resistance to exhalation above normal requirements. Breathing resistances in the patient connection cannot be directly monitored by the ventilator.

Connect O2 supply:

The O2 hose should be screwed into the side of the Savina, either from the medical O2 pipeline or an O2 cylinder.

Note: An O2 cylinder will provide only a short period of ventilation and should only be used in the transport of a patient or when the medical gas supply is not working.

Once preparation is complete connect a test lung to the y-piece.

Savina Checking Procedures

This check must be performed with every new circuit set up and prior to patient use. This check tests the functioning of lamps/LED’s, displays and audible alarms, ventilation function, PEEP function, expiratory volume measurement, measurement of inspiratory O2 concentration (FiO2), lower alarm limit MV, upper alarm limit Paw, mains power failure alarm and O2 alarm.

1. Ensure entire machine has been cleaned with alcohol prowipes after previous use.
3. Turn on the power switch at the back of the ventilator and allow the machine to run a 20 second self test (user manual p.36).
4. Ensure interior of circuit remains clean by attaching a sterile grey anaesthetic bag (alternatively, insert the plunger of a sterile 10ml syringe into the end of the circuit.
5. IMPORTANT: Ensure that the high pressure alarm limit is active.
   - Ensure Pmax is “ON”
6. Check Pmax alarm has been set at an appropriate level eg. 30cm H2O by:
   - Press “Alarms”
   - Select Paw and set safe limit.
7. Ensure large O2 cylinder is at least half full.

8. Attach label with date and time of new circuit, sign name and cover with a clean sheet if not using immediately.

9. Store in the appropriate area.

**Operation**

**Starting up:**

1. Turn the power switch on the back of the Savina to ‘on’. The ventilator will automatically run through a 20 second self-test shown on the start-up page.
2. At the end of the test phase, ventilation will automatically start with the last ventilation mode and ventilation parameters set.
3. Check the settings in the display fields and adjust as necessary.

**Setting ventilation modes and parameters**

- Press the appropriate ventilation mode key. The yellow LED light will light up.
- To set value=turn rotary knob, to confirm= press the rotary knob. The yellow LED in the key will go out.

Parameter settings for the active ventilation mode do not become operative until confirmed.

If you fail to confirm the new settings within 15 seconds, the previous setting will remain operative.

**Extreme settings:**
Certain ventilation parameters are limited by Savina to a threshold value. Extreme values can only be set after confirmation by pressing the rotary knob. (See table on p.43 of the instruction manual for a list of parameters and thresholds).

Settings:

To change or check settings, press the settings keys. Scroll through the settings with the rotary knob. Remember to confirm all changes by pressing the rotary knob.

Lock:
To protect the settings against modification press the Lock key.
When locked, the yellow LED light will light up; to unlock simply push the lock key again and the light will go off.

Flow Acceleration
- With the parameter ‘FlowAcc’ the pressure and flow increase can be influenced. A greater FlowAcc produces a steeper pressure and flow increase.
- A FlowAcc setting of 100 cmH2O/sec should suit most patients.
- FlowAcc (and Tinsp) can be used to adapt the pressure and flow curve to suit the patient’s needs.

AutoFlow
- The Autoflow function can be activated in the Settings menu.
- With Autoflow, the inspiration flow rate is automatically adjusted to changes in lung condition and to the spontaneous breathing and demand of the patient.
- The inspiratory flow is decelerated and regulated by Autoflow, so that the selected tidal volume can be reached with minimum airway pressure.
- As a general rule, Autoflow should always be activated.

Non-invasive Ventilation (NIV)
- Non-invasive positive pressure ventilation is available on the Savina ventilator.
- Can be used with either a face or nose mask.
- **NEVER** ventilate an intubated patient in the Mask/NIV mode.
- **Circuits and masks approved for use with the Savina ventilator must be used.**
- NIV ventilation is covered in depth in the NIV Learning Package.

How to select mask/NIV:
- Switch ventilator to standby. Hold standby key down for 3 seconds.
- Press alarm reset to switch off the audible alarm
- Select patent connection line=press rotary knob.
- Select mask/NIV line=press rotary knob.
- Confirm=press rotary knob.
A cautionary message will appear briefly.
Continue to set ventilation parameters as normally. Recheck all alarms.
When switched off, the Savina will automatically resume in invasive ventilation.

Apnoea Ventilation

For automatic switchover to volume controlled ventilation if the patient stops breathing. Apnoea ventilation can be activated in the following modes:

- SIMV
- SIMV/ABS
- SIMV/ABS/AUTOFLOW
- CPAP
- CPAP/ASB
- BIPAP
- BIPAP/ASB

- Apnoea ventilation can be set by going into settings.
- A Vt and frequency must be set.
- When apnoea ventilation is activated, press the alarm reset button to deactivate.

Setting Alarm Limits

1. Press the Alarms key.
2. Alarms 1/1 will appear on the display screen. (This page displays all the alarm limits that can be set. See p.55 of manual).

In the event of an alarm:

1. The red or yellow LED flashes.
2. The alarm message is displayed in the right hand corner of the top line if the screen.

There are three priority classes of alarms:
!!! = Warning (red LED flashes)
!! = Caution (yellow LED flashes)
!= Advisory (yellow LED is continuously lit).

Every time an alarm sounds the CCRN must immediately assess the patient and the ventilator to find the cause of the alarm breach and to rectify the problem. The alarm reset key can then be hit to erase the message from the screen. The silence button will silence the alarm for 2 minutes or until the alarm is remedied.

Displaying measured values
- Press values key. The screen page Values 1/2 appears.
- The airway pressure is indicated by a bar display. Other measured values are displayed numerically.

Caring for the Savina

Flow sensor:
1. Remove the flow sensor cover from the flow sensor
2. Push to flow sensor to the left as far as it will go and pull out.
   Note: the flow sensor must not be sent to CSSD to be cleaned as they are not temperature-stable and would be destroyed.
3. Disinfect flow sensor after every use in chlorhexidine solution for 1 hour.
4. Leave sensor to air dry for at least 30 minutes.

Expiration valve:

Turn sleeve to left and remove. The expiration valve and rubber bushing get sent to CSSD for sterilization.

Clean and wipe the machine with alcohol prowipes after every use!

Circuit

The Savina circuit tubing is completely disposable along with the bacterial/HME filters. All of this equipment is to be single patient use only and discarded when the patient no longer requires ventilation.

Note: All information regarding the Savina is in the Instructions For Use Manual located on top of the Savina.
COMPETENCY ASSESSMENT

1. Why is the upper respiratory tract important?

2. What is the major function of the lower respiratory tract?

3. Explain the difference between pulmonary ventilation and pulmonary respiration.

4. In a non-ventilated patient, is the process of inhalation positive or negative?

5. Where does the exchange of carbon dioxide and oxygen occur?

6. How is breathing regulated? What factors can affect breathing?

7. What is significance of the oxyhaemoglobin dissociation curve?

8. List 4 common reasons for mechanically ventilating a patient.

9. What is compliance?

10. What is resistance?

11. What factors affect compliance and resistance?
12. What is the principal aim of mechanical ventilation?

13. When is mechanical ventilation indicated? Give 3 examples.

14. Briefly describe each ventilator mode and state which ventilators in the ED use which mode.

- IPPV
- SIPPV
- SIMV
- CPAP
- PCV
- CMV
- PSV/ASB

15. What type of ventilator is the Oxylog 2000?
16. What type of ventilator is the Oxylog 3000?

17. What type of ventilator is the Savina?

18. What settings can you change on the ventilator to increase oxygenation?

19. What settings can you change on the ventilator to decrease carbon dioxide?

20. Briefly describe PIP, Plateau Pressure, Mean Airway Pressure and End Expiratory Pressure?
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