Quest Journals
Journal of Research in Agriculture and Animal Science
Volume 9 ~ Issue 5 (2022) pp: 01-12
ISSN(Online) : 2321-9459
www.questjournals.org

Research Paper

A Practical Approach Toward Mechanical Ventilation In Dogs

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ABSTRACT
Mechanical ventilation is currently an uncommon supportive measure in veterinary medicine and its use in largely restricted to academic institutions and speciality practices.(Archambault PM, St-Onge M.2012) As the discipline of veterinary critical care continues to grow application of mechanical ventilation will likely become more widespread.It is inevitable that mechanical ventilation will have a significant role in veterinary medicine.Pilbeam SP(2006). Mechanical ventilation can be a life saving tool for dogs and cats experiencing hypoxaemia respiratory failure and those that develop ventilatory failure. Ventilation of patients with ventilatory failure has been shown to have a better prognosis than ventilation of those ventilated for hypoxaemia respiratory failure.(King LG, Hendricks JC 1994).(Hopper K et al 2007). Mechanical ventilation is similar to providing manual intermittent positive-pressure ventilation (IPPV) except the breathing is controlled by a ventilator. Mechanical ventilation is the use of a machine to perform some or all of the work of breathing.(Clare M 2005) Principle indication for mechanical ventilation include Hypoxaemia , hypercapnia and excessive work of breathing that do not resolves with less invasive therapy. (Drager LF 2003)The prognosis varies with the underlying diseases and the degree of pulmonary pathology.
Successful application of positive pressure ventilation (PPV) the most prevalent form of mechanical ventilation requires appropriate patient selection Manning AM(2002). An understanding of ventilator function and most important intensive nursing care.Matthews NS (2003) Many veterinary patients can benefit from PPV it is hoped that the perception of the ventilator as a precursor of death will change with time so that it can be viewed as a useful and lifesaving adjunct to critical care. Rivers E(2001)

***R**eceived 22 Apr, 2022; **R**evised 02 May, 2022; **A**ccepted 05 May, 2022 © The author(s) 2022.*
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HISTORY
The roman physician galen first time describe mechanical ventilation. if you take a dead animal and blow air through its larynx. then bronchi fills first and lungs attain greatest distension vesalius too describes ventilation by inserting or cane into the trachea of animals.
In 1908 George poe demonstrated his mechanical respirator by asphyxiating dogs and seemingly bringing them back to life MacIntyre NR(2001).

I. INTRODUCTION:-

Mechanical ventilation is typically used after an invasive intubation , a procedure wherein an endotracheal or tracheostomy tube is inserted into the airway.

It is used in acute setting such as in the icu for a short period of time during a serious illness. it may be used at home or in a nursing or rehabilitation institution if patients have chronic illness that require long term ventilation assistance.

PRACTICAL USES OF VENTILATORS
- Normal physiological respiratory rate, inspiratory time, inspiratory:expiratory ratio and tidal volume should be used.
- The ventilator should be set up and checked prior to use to allow detection and correction of any problems before induction of anaesthesia.

- End-tidal carbon dioxide (ETCO2) should be maintained in the normal range of 4.6–6.0 KPa or 35–45 mmHg.
- The thorax should be examined to look at the degree of chest wall movement. There should be noticeable, but not excessive chest wall movement.
- Ventilators should be serviced regularly by the manufacturer and kept clean and in good working order.

CLASSIFICATION OF VENTILATORS

Most veterinary ventilators are volume-controlled ventilators, meaning a constant flow (or volume) is delivered to the patient. Volume-controlled ventilators may be either time cycled, volume cycled or pressure cycled. This refers to the method used to change from the inspiratory to expiratory phase, i.e., the ventilator changes from inspiration to expiration when either a set time, volume or pressure is reached. To use a volume-cycled ventilator, the patient's tidal volume must be set, inspiration will occur and the lungs will inflate until the set tidal volume is delivered. With a pressure-cycled ventilator a peak inspiratory pressure is set. The lungs will inflate until this set pressure is reached. The respiratory rate can also be set and some ventilators also allow the inspiratory to expiratory ratio to be adjusted. Most ventilators require either an electricity supply or an additional gas source to drive the ventilator

SPECIFIC VENTILATORS USED IN SMALL ANIMAL ANAESTHESIA

Minute volume divider ventilators work differently and do not fit into the above classification system. The operator sets the tidal volume; the ventilator uses the fresh gas flow from the anaesthetic machine and divides it into individual breaths of the set tidal volume which are delivered to the patient. Gas from the fresh gas flow is stored within the ventilator until a large enough volume is reached for the ventilator to deliver the next breath. The ventilator stores gas from the fresh gas flow, when enough gas for the next breath is obtained it delivers it to the patient. **The Manley ventilator** is an example of a minute volume divider. The main limitation with the Manley ventilator is that it cannot be used in animals weighing less than approximately 13 kg, as small enough tidal volumes cannot be delivered. Although old, the main advantages of these ventilators are that they do not need a power supply or an additional driving gas and that they are easy to use and very reliable.

The Vetronic 'Merlin' ventilator is a ventilator specifically designed for use in small animals. It is very versatile and can deliver tidal volumes varying between 1 and 800 ml, with the manufacturer stating that it can be used on patients from 50 g to 70 kg. Its working mechanism is volume controlled and can either be volume cycled, pressure cycled or time cycled. It has many controls and variables which provide flexibility and a wealth of information including compliance. This may appear confusing to operators not accustomed to using ventilators; however, it comes with a comprehensive instruction manual. It can deliver PEEP and audible alarms are available to warn of patient disconnection, high airway pressures and a blocked inlet. It can be connected to a rebreathing or a non-rebreathing system. As it is electronically driven, a power supply is required.

The Pneupac Ventilator is a volume-controlled, time-cycled ventilator. Ex-hospital Pneupac ventilators can be sourced secondhand relatively easily and cost effectively. An additional gas source such as medical air or oxygen is required as a driving force. The standard valve can be replaced with a Newton valve for use in small animals below approximately 5–7 kg and it functions as a 'mechanical thumb'.

WEANING OFF THE VENTILATOR

This refers to the process of changing over from the provision of IPPV to spontaneous ventilation. In most healthy patients this occurs easily; however, a pause in ventilation is normally necessary to allow the concentration of carbon dioxide in the blood to increase. Chemoreceptors located in the carotid and aortic bodies detect this increase in blood carbon dioxide levels; information is passed to the respiratory centre in the medulla within the central nervous system, stimulating spontaneous ventilation. Some people suggest providing several manual breaths in quick succession after switching off the ventilator to break the rhythmic ventilation cycle, in order to encourage spontaneous ventilation. The patient's chest excursions and ETCO2 should be monitored to ensure that the patient is ventilating adequately.Mellema MS, Haskins SC(2006)

Potential complications of mechanical ventilation.

Complication	Cause	Ways to minimise risk
Hypotension due to reduced venous return	Increased intrathoracic pressure reduces venous return to the heart by compressing large blood vessels	Use of longer expiratory times and shorter inspiratory times along with avoiding excessive airway pressures
Barotrauma to lungs may lead to lung damage, emphysema, pneumothorax or air embolism	Excessive inflation pressures and volumes	Peak inspiratory pressure should not exceed 20 cmH_2O; often much less is adequate (especially in cats)
Power supply failure leading to ventilator failure	Power cut or fuse blown	Facilities should be available to provide manual IPPV if necessary
Disconnection from ventilator/breathing system	Tubes being pulled or disconnected (by surgeon)	Monitor capnograph and chest excursions

In medicine mechanical ventilation is a method to mechanically assist or replace spontaneous breathing Haskins SC, King LG: (2004)
Types of ventilator are
The Manley ventilator
The Vetronic 'Merlin' ventilator
Pneupac Ventilator

Mechanical ventilation can be
Noninvasive, involving various types of face masks
Invasive, involving endotracheal intubation
Selection and use of appropriate techniques require an understanding of respiratory mechanics.

Indications
There are numerous indications for endotracheal intubation and mechanical ventilation (see table Situations Requiring Airway Control), but, in general, mechanical ventilation should be considered when there are clinical or laboratory signs that the patient cannot maintain an airway or adequate oxygenation or ventilation.
Acute lung injury
apnea with respiratory arrest , including cases from intoxication
Chronic obstructive pulmonary diseases
Severe hypoxemia despite oxygen therapy (PaO_2 <60 mm Hg)
Severe hypoventilation (defined as PCO_2 >60 mm Hg)
Excessive work of breathing
Severe circulatory shock.
Acute respiratory acidosis with partial pressure .
Increased work of breathing as evidenced by significant tachypnoea, retraction and other physical sign of respiratory distress
Concerning findings includeRespiratory rate > 30/minute
Inability to maintain arterial oxygen saturation > 90% with fractional inspired oxygen (FIO_2) > 0.60pH < 7.25$PaCO_2$ > 50 mm Hg (unless chronic and stable)The decision to initiate mechanical ventilation should be based on clinical judgment that considers the entire clinical situation and not simple numeric criteria. However, mechanical ventilation should not be delayed until the patient is in extremis.(Archambault PM, St-Onge M.2012)

Various terminologies used in mechanical ventilation:-
APRV--Airway Pressure release ventilation
ASB-- Assisted spontaneous breathing --also ASV =Assistance spontaneous Ventilation
ASV Adaptive support ventilation --a patented technology --close--loop mechanical respiration, a further development of MMV Can also stand for assisted spontaneous ventilation
ATC - Automatic tube compensation
BIPAP --Biphasic positive airway pressure
CMV--Continuous mandatory Ventilation
CPAP--Continuous positive airway pressure
CPPV--Continuous positive pressure ventilation

EPAP-- Expiratory Positive airway pressure
HFV:- High ferquency Ventilation
-HFFI:-High frequency flow inturruption
-HFJV:-High frequency jet ventilation
-HFOV:- High frequen oscillatory ventilation
-hfppv:- high frequency positive pressure ventilation.
*ILV:- Independant Lung Ventilation--Separate sides positive pressure ventilation
*IPAP:-Inspiratory positive airway pressure
*IPPV:-Intermittent positive pressure ventilation
*IRV:- Inversed ratio ventilation ---Mechanical ventilation with switched respiration phase/time rate.
*LFPPV:- Low frequency positive pressure ventilation
*MMV:- Mandatory minute volume
*NAVA:- Neurally adjusted ventilatory assist

Respiratory Mechanics

Normal inspiration generates negative intrapleural pressure, which creates a pressure gradient between the atmosphere and the alveoli, resulting in air inflow. In mechanical ventilation, the pressure gradient results from increased (positive) pressure of the air source.

Peak airway pressure is measured at the airway opening (Pao) and is routinely displayed by mechanical ventilators. It represents the total pressure needed to push a volume of gas into the lung and is composed of pressures resulting from inspiratory flow resistance (resistive pressure), the elastic recoil of the lung and chest wall (elastic pressure), and the alveolar pressure present at the beginning of the breath (positive end-expiratory pressure [PEEP]—see figure Components of airway pressure). Thus Resistive pressure is the product of circuit resistance and airflow. In the mechanically ventilated patient, resistance to airflow occurs in the ventilator circuit, the endotracheal tube, and, most importantly, the patient's airways. (NOTE: Even when these factors are constant, an increase in airflow increases resistive pressure.)

Components of airway pressure during mechanical ventilation, illustrated by an inspiratory-hold maneuverPEEP = positive end-expiratory pressure.Components of airway pressure during mechanical ventilation, illustrated by an inspiratory-hold maneuver Elastic pressure is the product of the elastic recoil of the lungs and chest wall (elastance) and the volume of gas delivered. For a given volume, elastic pressure is increased by increased lung stiffness (as in pulmonary fibrosis) or restricted excursion of the chest wall or diaphragm (eg, in tense ascites or massive obesity). Because elastance is the inverse of compliance, high elastance is the same as low compliance.(Archambault PM, St-Onge M.2012)

End-expiratory pressure in the alveoli is normally the same as atmospheric pressure. However, when the alveoli fail to empty completely because of airway obstruction, airflow limitation, or shortened expiratory time, end-expiratory pressure may be positive relative to the atmosphere. This pressure is called intrinsic PEEP or autoPEEP to differentiate it from externally applied (therapeutic) PEEP, which is created by adjusting the mechanical ventilator or by placing a tight-fitting mask that applies positive pressure throughout the respiratory cycle.

Any elevation in peak airway pressure (eg, > 25 cm H2O) should prompt measurement of the end-inspiratory pressure (plateau pressure) by an end-inspiratory hold maneuver to determine the relative contributions of resistive and elastic pressures. The maneuver keeps the exhalation valve closed for an additional 0.3 to 0.5 second after inspiration, delaying exhalation. During this time, airway pressure falls from its peak value as airflow ceases. The resulting end-inspiratory pressure represents the elastic pressure once PEEP is subtracted (assuming the patient is not making active inspiratory or expiratory muscle contractions at the time of measurement). The difference between peak and plateau pressure is the resistive pressure.

Elevated resistive pressure (eg, > 10 cm H2O) suggests that the endotracheal tube has been kinked or plugged with secretions or that an intraluminal mass or bronchospasm is present.Increased elastic pressure (eg, > 10 cm H2O) suggests decreased lung compliance due toEdema, fibrosis, or lobar atelectasis

Large pleural effusions , pneumothorax , or fibrothorax Extrapulmonary restriction as may result from circumferential burns or other chest wall deformity, ascites, pregnancy, or massive obesity

A tidal volume too large for the amount of lung being ventilated (eg, a normal tidal volume being delivered to a single lung because the endotracheal tube is malpositioned) Intrinsic PEEP (auto PEEP) can be measured in the passive patient through an end-expiratory hold maneuver. Immediately before a breath, the expiratory port is closed for 2 seconds. Flow ceases, eliminating resistive pressure; the resulting pressure reflects alveolar pressure at the end of expiration (intrinsic PEEP). Although accurate measurement depends on the patient being completely passive on the ventilator, it is unwarranted to use neuromuscular blockade solely for the purpose of measuring intrinsic PEEP. A nonquantitative method of identifying intrinsic PEEP is to inspect the expiratory flow tracing. If expiratory flow continues until the next breath or the patient's chest fails to come

to rest before the next breath, intrinsic PEEP is present. The consequences of elevated intrinsic PEEP include increased inspiratory work of breathing and decreased venous return, which may result in decreased cardiac output and hypotension.

The demonstration of intrinsic PEEP should prompt a search for causes of airflow obstruction (eg, airway secretions, decreased elastic recoil, bronchospasm); however, a high minute ventilation (> 20 L/minute) alone can result in intrinsic PEEP in a patient with no airflow obstruction. If the cause is airflow limitation, intrinsic PEEP can be reduced by shortening inspiratory time (ie, increasing inspiratory flow) or reducing the respiratory rate, thereby allowing a greater fraction of the respiratory cycle to be spent in exhalation.

Means and Modes of Mechanical Ventilation

Mechanical ventilators are

Volume cycled: Delivering a constant volume with each breath (pressures may vary)

Pressure cycled: Delivering constant pressure during each breath (volume delivered may vary) A combination of volume and pressure cycled

Assist-control (A/C) modes of ventilation are modes that maintain a minimum respiratory rate regardless of whether or not the patient initiates a spontaneous breath. Because pressures and volumes are directly linked by the pressure-volume curve, any given volume will correspond to a specific pressure, and vice versa, regardless of whether the ventilator is pressure cycled or volume cycled.

Adjustable ventilator settings differ with mode but include

Respiratory rate
Tidal volume
Trigger sensitivity
Flow rate
Waveform
Inspiratory/expiratory (I/E) ratio
Volume-cycled ventilation
Volume-cycled ventilation delivers a set tidal volume. This mode includes

Volume-control (V/C)
Synchronized intermittent mandatory ventilation (SIMV)
The resultant airway pressure is not fixed but varies with the resistance and elastance of the respiratory system and with the flow rate selected.

V/C ventilation is the simplest and most effective means of providing full mechanical ventilation. In this mode, each inspiratory effort beyond the set sensitivity threshold triggers delivery of the fixed tidal volume. If the patient does not trigger the ventilator frequently enough, the ventilator initiates a breath, ensuring the desired minimum respiratory rate.

SIMV also delivers breaths at a set rate and volume that is synchronized to the patient's efforts. In contrast to V/C, patient efforts above the set respiratory rate are unassisted, although the intake valve opens to allow the breath. This mode remains popular, despite not providing full ventilator support as does V/C, not facilitating liberation of the patient from mechanical ventilation, and not improving patient comfort.

Pressure-cycled ventilation
Pressure-cycled ventilation delivers a set inspiratory pressure. This mode includes
Pressure control ventilation (PCV)
Pressure support ventilation (PSV)
Noninvasive modalities applied via a tight-fitting face mask (several types available)
Hence, tidal volume varies depending on the resistance and elastance of the respiratory system. In this mode, changes in respiratory system mechanics can result in unrecognized changes in minute ventilation. Because it limits the distending pressure of the lungs, this mode can theoretically benefit patients with acute respiratory distress syndrome (ARDS); however, no clear clinical advantage over V/C has been shown, and, if the volume delivered by PCV is the same as that delivered by V/C, the distending pressures will be the same.

Pressure control ventilation is a pressure-cycled form of A/C. Each inspiratory effort beyond the set sensitivity threshold delivers full pressure support maintained for a fixed inspiratory time. A minimum respiratory rate is maintained.

In pressure support ventilation, a minimum rate is not set; all breaths are triggered by the patient. The ventilator assists the patient by delivering a pressure that continues at a constant level until the patient's inspiratory flow falls below a preset level determined by an algorithm. Thus, a longer or deeper inspiratory effort by the patient results in a larger tidal volume. This mode is commonly used to liberate patients from mechanical ventilation by letting them assume more of the work of breathing. However, no studies indicate that this approach is more successful than others in discontinuing mechanical ventilation.

Noninvasive positive pressure ventilation (NIPPV)
NIPPV is the delivery of positive pressure ventilation via a tight-fitting mask that covers the nose or both the nose and mouth. Helmets that deliver NIPPV are being studied as an alternative for patients who cannot tolerate the standard tight-fitting face masks. Because of its use in spontaneously breathing patients, it is primarily applied as a form of PSV or to deliver end-expiratory pressure, although volume control can be used. (See also How To Do Noninvasive Positive Pressure Ventilation .)

Noninvasive Positive Pressure Ventilation (NIPPV) Using Bilevel Positive...
Noninvasive Positive Pressure Ventilation (NIPPV) Using Bilevel Positive Airway Pressure
NIPPV can be given asContinuous positive airway pressure (CPAP)Bilevel positive airway pressure (BiPAP) In CPAP, constant pressure is maintained throughout the respiratory cycle with no additional inspiratory support.With BiPAP, the physician sets both the expiratory positive airway pressure (EPAP) and the inspiratory positive airway pressure (IPAP), with respirations triggered by the patient.

In either mode, because the airway is unprotected, aspiration is possible, so patients must have adequate mentation and airway protective reflexes and no imminent indication for surgery or transport off the floor for prolonged procedures. Obtunded patients and patients with copious secretions are not good candidates. NIPPV also should be avoided in patients who are hemodynamically unstable and in those with evidence of impaired gastric emptying, as occurs with ileus, bowel obstruction, or pregnancy. In such circumstances, swallowing large quantities of air may result in vomiting and life-threatening aspiration. Also, IPAP must be set below esophageal opening pressure (20 cm H2O) to avoid gastric insufflation.

Indications for conversion to endotracheal intubation and conventional mechanical ventilation include the development of shock or frequent arrhythmias , myocardial ischemia, and transport to a cardiac catheterization laboratory or surgical suite where control of the airway and full ventilatory support are desired.

NIPPV can be used in the outpatient setting. For example, CPAP is often used for patients with obstructive sleep apnea , whereas BiPAP can be used for those with concomitant obesity-hypoventilation syndrome or for chronic ventilation in patients with neuromuscular or chest wall diseases.

Ventilator settings

Ventilator settings are tailored to the underlying condition, but the basic principles are as follows.Tidal volume and respiratory rate set the minute ventilation. Too high a volume risks overinflation; too low a volume allows for atelectasis . Too high a rate risks hyperventilation and respiratory alkalosis along with inadequate expiratory time and autoPEEP; too low a rate risks inadequate minute ventilation and respiratory acidosis . A low tidal volume of 6 to 8 mL/kg ideal body weight (IBW) was initially recommended for patients with acute respiratory distress syndrome (ARDS—see sidebar Initial Ventilator Management in ARDS); however, such low tidal volume is usually also appropriate in certain patients who have normal lung mechanics (1 , 2 , 3), such as those who are on mechanical ventilation during surgery (4 , 5). Other patients (eg, those with trauma, obtundation, severe acidosis) may be started at slightly higher tidal volume (eg, 8 to 10 mL/kg). IBW rather than actual body weight is used to determine the appropriate tidal volume for patients who have lung disease and who are receiving mechanical ventilation:

Sensitivity adjusts the level of negative pressure required to trigger the ventilator. A typical setting is –2 cm H2O. Too high a setting (eg, more negative than –2 cm H2O) causes weak patients to be unable to trigger a breath. Too low a setting (eg, less negative than –2 cm H2O) may lead to overventilation by causing the machine to auto-cycle. Patients with high levels of autoPEEP may have difficulty inhaling deeply enough to achieve a sufficiently negative intra-airway pressure.

The I:E ratio (inspiratory:expiratory ratio) is the ratio of time spent in inhalation versus that spent in exhalation. The I:E ratio can be adjusted in some modes of ventilation. A normal setting for patients with normal mechanics

is 1:3. Patients with asthma or exacerbations of COPD (chronic obstructive pulmonary disease) should have ratios of 1:4 or even more to limit the degree of autoPEEP.

The inspiratory flow rate can be adjusted in some modes of ventilation (ie, either the flow rate or the I:E ratio can be adjusted, not both). The inspiratory flow should generally be set at about 60 L/minute but can be increased up to 120 L/minute for patients with airflow limitation to facilitate having more time in exhalation, thereby limiting autoPEEP.

FIO2 (fraction of inspired oxygen) is initially set at 1.0 (100% oxygen) and is subsequently decreased to the lowest level necessary to maintain adequate oxygenation.

PEEP can be applied in any ventilator mode. PEEP increases end-expired lung volume and reduces airspace closure at the end of expiration. Most patients undergoing mechanical ventilation may benefit from the application of PEEP at 5 cm H2O to limit the atelectasis that frequently accompanies endotracheal intubation, sedation, paralysis, and/or supine positioning. Higher levels of PEEP improve oxygenation in disorders such as cardiogenic pulmonary edema and ARDS . PEEP permits use of lower levels of FIO2 while preserving adequate arterial oxygenation. This effect may be important in limiting the lung injury that may result from prolonged exposure to a high FIO2 (≥ 0.6). PEEP increases intrathoracic pressure and thus may impede venous return, provoking hypotension in a hypovolemic patient, and may overdistend portions of the lung, thereby causing ventilator-associated lung injury (VALI). By contrast, if PEEP is too low, it may result in cyclic airspace opening and closing, which in turn may also cause VALI from the resultant repetitive shear forces. It is important to keep in mind that the pressure-volume curve varies for different regions of the lung. This variation means that, for a given PEEP, the increase in volume will be lower for dependent regions compared to nondependent regions of the lung.

Need of mechanical ventilation:-
Treat or prevent hypercapnia (EXCESS RETENSION OF CO2)
Prevent severe hypercapnia in healthy patients
Prevent hypercapnia and increased ICP in neurologic patients
Prevent hypercapnia and increased IOP in ocular (globe) surgery
Prevent hypercapnia and decreased portal flow in hepatic disease
Prevent hypercapnia and cardiovascular effects in cardiac disease patients
Prevent hypercapnia in patients with thoracic insufficiency e.g. chest wall trauma, Guillain-Barré ,acute lung injury
Provide ventilation in patients paralyzed with neuromuscular blocking agents

2. Optimize isoflurane or sevoflurane delivery

Maintain constant delivery of inhalation agent and avoid inadequate agent uptake

3. Improve oxygenation

In patients with major lung collapse, e.g. diaphragmatic rupture, pneumothorax

4. Decrease work of breathing

Lung disease, such as pulmonary edema or bronchospasm, can increase respiratory muscle oxygen requirement from 5% to 25-30% of total oxygen delivery, thus decreasing oxygen available for other organs.

Old or sick patients may benefit from artificial ventilation for this reaso

NON INVASIVE Technique	**INVASIVE Technique**
Oxygen mask	**Laryngeostomy**
Nasal mask	**Endotracheal Intubation**
Oxygen chamber	**Intubation**
Helmet	**Tracheostomy**
Hand AMBU Bags	
e.g COPD Patients	
CPAP	

Negative pressure Ventilation
creats a -ve pressure environment around patients chest.
Thus sucks the air into lungs
Chest expand and Contract
Removes excess CO2 from lungs
Less commonly used
e.g Iron lung

Positive Pressure Ventilation
works by increasing the pressure in the patients airway
Pushes the air into the lungs
Pushes O2 in the lungs
More commonly used
decreased cardiac output by decreasing venous return

Patient positioning

Mechanical ventilation is typically done with the patient in the semiupright position. However, in patients with ARDS, prone positioning may result in better oxygenation, primarily by creating more uniform ventilation. Uniform ventilation reduces the amount of lung that has no ventilation (ie, the amount of shunt), which is generally greatest in the dorsal and caudal lung regions, while having minimal effects on perfusion distribution.Clare M, Hopper K. (2005)

Although many investigators advocate a trial of prone positioning in patients with ARDS who require high levels of PEEP (eg, > 12 cm H2O) and FIO2 (eg, > 0.6), initial trials did not show any improvement in mortality with this strategy (however, these trials were underpowered). A subsequent large, multicenter, prospective trial (1) assessed patients who had moderately severe ARDS (PaO2:FIO2 < 150 mm Hg on an FIO2 ≥ 0.6, PEEP > 5 cm H2O) and who were on a tidal volume of about 6 mL/kg. These patients were randomized to undergo ≥ 16 hours of prone positioning or be left in the supine position during ventilation. The study, which included a total of 466 patients, identified lower 28- and 90-day mortality in the prone-positioning group without a significant incidence of associated complications. Improvements in patient selection and management protocols are thought to account for the better results compared to earlier studies.

Prone positioning is contraindicated in patients with spinal instability or increased intracranial pressure. This position also requires careful attention by the intensive care unit staff to avoid complications, such as dislodgement of the endotracheal tube or intravascular catheters.Clare M, Hopper K. (2005)

Sedation and comfort

Although many patients tolerate mechanical ventilation via endotracheal tube without sedatives, some require IV administration of sedatives (eg, propofol, lorazepam, midazolam) and analgesics (eg, morphine, fentanyl) to minimize stress and anxiety. These drugs can also reduce energy expenditure to some extent, thereby reducing carbon dioxide production and oxygen consumption. Doses should be titrated to the desired effect, guided by standard sedation/analgesia scoring systems (eg, Richmond Agitation Sedation Scale , Riker Sedation-Agitation Scale). Patients undergoing mechanical ventilation for ARDS typically require higher levels of sedation and analgesia. The use of propofol for longer than 24 to 48 hours requires periodic (eg, every 48 hours) monitoring of serum triglyceride levels. There is evidence that continuously administered IV sedation prolongs the duration of mechanical ventilation. Thus, the goal is to achieve adequate but not excessive sedation, which can be accomplished by using continuous sedation with daily interruption or by using intermittent infusions. Pilbeam SP(2006)

Neuromuscular blocking agents are not used routinely in patients undergoing mechanical ventilation because of the risk of prolonged neuromuscular weakness and the need for continuous heavy sedation; however, one study did show reduced mortality at 90 days in patients with early, severe ARDS who received 48 hours of neuromuscular blockade (1). Unfortunately, these findings were not replicated in a larger follow-up study of early neuromuscular blockade in ARDS, comparing it to lighter sedation without neuromuscular blockade (2). Therefore, routine paralysis for severe ARDS is not recommended. Exceptions who may benefit from neuromuscular blockade include patients who fail to tolerate some of the more sophisticated and complicated modes of mechanical ventilation and to prevent shivering when cooling is used after cardiac arrest.

Parameters of Mechanical Ventilation are
Respiratory Rate(f):-Normally
Tidal Volume :(VT)-10 to 15 mL/kg

Oxygen Concentration(FiO2):-
I:E Ratio:- 1:2
Flow Rate :- 6 to 10 litres per minute
Sensitivity /Trigger:-
Pressure Limit:-
PEEP:-

Complications of mechanical ventilation and safeguards

Complications of mechanical ventilation can be divided into those resulting from

Endotracheal intubation

Mechanical ventilation itself Prolonged immobility and inability to eat normallyThe presence of an endotracheal tube causes risk of sinusitis (which is rarely of clinical importance), ventilator-associated pneumonia , tracheal stenosis, vocal cord injury, and, very rarely, tracheal-esophageal or tracheal-vascular fistula. Purulent tracheal aspirate in a febrile patient who has an elevated white blood cell count > 48 hours after ventilation has begun suggests ventilator-associated pneumonia.

Complications of ongoing mechanical ventilation itself include pneumothorax , oxygen toxicity, hypotension, and ventilator-associated lung injury.Oxygen toxicity refers to inflammatory changes, alveolar infiltration, and, eventually, pulmonary fibrosis that can develop following prolonged exposure to high FIO2 (eg, >0.6). Toxicity is both concentration- and time-dependent. An FIO2 > 0.6 should be avoided unless necessary for survival. An FIO2 < 0.6 is well tolerated for long periods.

Ventilator-associated lung injury (VALI), sometimes termed ventilator-induced lung injury, is alveolar and/or small airway injury related to mechanical ventilation. Possible mechanisms include alveolar overdistention (ie, volutrauma) and the shear forces created by repetitive opening and collapse of alveoli (ie, atelectrauma), leading to release of inflammatory mediators resulting in increased alveolar permeability, fluid accumulation, and loss of surfactant.

Procedure for mechanical Ventilation:-

Animal patient on table and properly restraint
anaesthesia and Mouth gag .
Endotracheal Tube through mouth gag.
Connect ventilator to electrical supply
Connect ventilator to oxygen source
Connect ventilator to scavenger system
Insert adapter with expiratory valve and circle hose (if present)
Ventilator control panel: Set respiratory rate (frequency)
Set maximum inspiratory pressure limit (if available)
Insure volume knob is not set too high
Set minute volume 15 ml x kg x 12 (if available)
Adjust inspiratory:expiratory time ratio, usually 1:2 or 1:3 (if available)

6. Remove rebreathing bag from circle and connect tubing from bellows

7. Close circle pop-off valve

8. Turn on ventilator (allow bellows to partially fill with oxygen first)

Adjust tidal volume to achieve target volume or pressure or ETCO2 30-40 mm Hg

9. Observe chest movement

10.There should be no need to alter the oxygen flow into the anesthetic circuit

Modes of Mechanical Ventilation:-

Controlled Mandatory Ventilation(CMV)
Assist-Cont Mandatory Ventilation
Synchronized Intermittent Mandatory Ventilation (SIMV)
Positive Expiratory End Pressure (PEEP)
Continuous positive Airway Pressure (CPAP)

Pressure support Ventilation (PSV)

Complications of Mechanical Ventilation:-
HypotensionPneumothorax
Decreased Cardiac Output Lung Injury Airway Obstruction (LOWER)
CNS Diseases
with healthy lungs
Intracranial or diffuse LMN
Nosocomial Pneumonia
Positive Water Balance
Increased Intracranial Pressure
Alamrs turned off or non functional
nusitis or nasal injury
Mucosal lesions
Aspiration GI Bleeding, Inappropriate Ventilation (Respiratory Acidosis or Alkalosis, Thick secretions patient discomfort due to pulling or jarring ETT or tracheostomy, High PaO2 low PaO2 Anxiety and fear, Dysarrthymias or vegal reactions during or after suctioning , incorrect PEEP setting. Inability to tolearte Ventilator mode
Complication /Cause of death
Pneumothorax
Pulmonary Microboli with necrosis
Tracheal Perforation
Sepsis
Cardiovascular failure

Plan for care of ventilated Patients
Patients Goal:-
effective breathing pattern
adequate gas exchange
nutritional status to meet bodys need
Not develop pulmonary infection
Patient will not develop Problems with related to immobility
indicate understanding for mechanical ventilation.

Nursing Interventions:-
Observe changes in respiratory rate and depth, observe for use for the use of accessory muscles observe the tube misplacement- note and post cm. marking at lip/teeth after x-ray confirmation.
prevents accidental extubation by taping tube securly checking q 2h: resting /sedating as needed
inspect thorax for symmetry of movement . determine adequacy of breathing pattern asymmetry may indicate haemothorax or pneumothorax. measure tidal volume and vital capacity.
Assess to pain
monitor chest Xrays
Maintain ventilation setting as ordered
Elevate patients head to bed 60-90 degree angle this position moves the abdominal contents awat from the diaphragm, whih facilitates its contraction.

Asses for GI problems. preventative measure include reliving anexity, antacids or H2 receptor antagonist therapy
adequate sleep cycles, adequate communication cycles
maintain muscle strength with active /active assistive/passive ROM and preven contractures with the use of span -aids or splints.
Explain purpose/mode and all treatments encourage patients to relax and breath with the ventilator explain alarms
teach importance of deep breathing
provide alternate method of communication
keep call bell within reach
keep informed of results of studies/progress.
demonstrated confidence.

Most hospitals have standardized protocols to reduce complications. Elevating the head of the bed to > 30° decreases risk of ventilator-associated pneumonia, and routine turning of the patient every 2 hours decreases the risk of skin breakdown (see prevention of pressure ulcers).

The most effective way to reduce complications of mechanical ventilation is to limit its duration. Daily "sedation vacations" and spontaneous breathing trials help determine the earliest point at which the patient may be liberated from mechanical support .

II. CONCLUSION

Mechanical ventilation is becoming a critical component of caring for animals with respiratory compromise.One of the most challenging aspects of mechanical ventilation is making the decision to initiate it. All clinicians will be faced with patients that require mechanical ventilation and need to be prepared to intubate and manually ventilate a patient to stabilize it. Mechanical ventilation can have a role in supporting patients with oxygenation and ventilation issues. The goal is to stabilize critically ill patients, maintain them until there is clinical improvement, and ultimately wean them from machine support.

The prognosis for successful weaning depends on the underlying disease and the severity of concurrent issues.

REFERENCES

[1]. Archambault PM, St-Onge M. Invasive and noninvasive ventilation in the emergency department. Emerg Med Clin North Am 2012;30:421–49.

[2]. Aubier M, Trippenbach T, Roussos C, et al. Respiratory muscle fatigue during cardiogenic shock. J Appl Physiol 1981;51:499–508.

[3]. Campbell VL, King LG. Pulmonary function, ventilator management, and outcome of dogs with thoracic trauma and pulmonary contusions: 10 cases (1994–1998). J Am Vet Med Assoc 2000;217:1505–9

[4]. Clare M, Hopper K. Mechanical ventilation: ventilator settings, patient management, and nursing care. Compend Contin Educ Pract Vet 2005.

[5]. Drager LF, Abe JM, Martins MA, et al: Impact of clinical experience on quantification of clinical signs at physical examination. J Intern Med254:257–263, 2003.14.

[6]. Drellich S: Principles of mechanical ventilation. Vet Clin North Am SmallAnim Pract 32(5):1087–1100, 2002.

[7]. Grosenbaugh DA, Muir WW III: Accuracy of noninvasive oxyhemoglobin saturation, end-tidal carbon dioxide concentration, and blood pressure monitoring during experimentally induced hypoxemia, hypotension, or hypertension in anesthetized dogs. Am J Vet Res 59(2):205–212, 1998.

[8]. Hackner SG: Emergency management of traumatic pulmonary contusions.Compend Contin Educ Pract Vet 17(5):677–686, 1995

[9]. Hackett TB: Pulse oximetry and end tidal carbon dioxide monitoring. Vet Clin North Am Small Anim Pract 32(5):1021–1029, 2002.

[10]. Haskins SC, King LG: Positive pressure ventilation, in King LG (ed): Textbook of Respiratory Disease in Dogs and Cats. Philadelphia, WB Saunders,2004, pp 217–229

[11]. Hendricks JC: Pulse oximetry, in King LG (ed): Textbook of Respiratory Disease in Dogs and Cats. Philadelphia, WB Saunders, 2004, pp 193–197..

[12]. Haskins SC: Interpretation of blood gas measurements, in King LG (ed):Textbook of Respiratory Disease in Dogs and Cats. Philadelphia, WB Saunders,2004, pp 181–193.

[13]. Hess DR, Kacmarek RM. Essentials of mechanical ventilation. 2nd edition. New York: McGraw-Hill; 2002

[14]. Hickling KF, Walsh J, Henderson S, et al. Low mortality rate in adult respiratory distress syndrome using low-volume, pressure-limited ventilation with permissive hypercapnia: a prospective study. Crit Care Med 1994;22(10):1568–78.

[15]. Hopper K, Haskins SC, Kass PH, et al. Indications, management and outcome of long-term positive-pressure ventilation in dogs and cats: 148 cases (1990–2001). J Am Vet Med Assoc 2007;230:64–75

[16]. Ilkiw JE, Rose RJ, Martin IC: A comparison of simultaneously collected arterial, mixed venous, jugular venous and cephalic venous blood samples in the assessment of blood-gas and acid-base status in the dog. J Vet Intern Med5(5):294–298, 1991.

[17]. Jubran A, Tobin MJ: Reliability of pulse oximetry in titrating supplemental oxygen therapy in ventilator-dependent patients. Chest 97(6):1420–1425, 1990.9.

[18]. King LG, Anderson JG, Rhodes WH, Hendricks JC: Arterial blood gas tensions in healthy aged dogs. Am J Vet Res 53(10):1744–1748, 1992

[19]. King LG, Hendricks JC: Use of positive-pressure ventilation in dogs and cats: 41 cases (1990–1992). JAVMA 204(7):1045–1052, 1994.

[20]. Laghi F, Tobin MJ. Indications. In: Tobin MJ, editor. Indications for mechanical ventilation. 2nd edition. New York: McGraw-Hill; 2006. p. 129–62.

[21]. Kontoyannis DA, Nanas JN, Kontoyannis SA, *et al*. Mechanical ventilation in conjunction with the intra-aortic balloon pump improves the outcome of patients in profound cardiogenic shock. Intensive Care Med 1999;25:835–8.

[22]. Lee JA, Drobatz KJ: Respiratory distress and cyanosis in dogs, in King LG (ed): Textbook of Respiratory Disease in Dogs and Cats. Philadelphia, WBSaunders, 2004, pp 1–12

[23]. Lumb AB: Diffusion of respiratory gasses, in Lumb AB (ed): Nunn's Applied Respiratory Physiology, ed 5. Woburn, Reed Educational and ProfessionalPublishing Ltd, 2000, pp 200–221.

[24]. Lumb AB: Distribution of pulmonary ventilation and perfusion, in LumbAB (ed): Nunn's Applied Respiratory Physiology, ed 5. Woburn, Reed Educational and Professional Publishing Ltd, 2000, pp 163–189.

[25]. MacIntyre NR: Mechanical Ventilation. Philadelphia, WB Saunders, 2001.

[26]. MacIntyre NR. Mechanical ventilation. In: Vincent JL, Abraham E, Moore FA, et al, editors. Textbook of Critical Care. 6th edition. Philadelphia: Elsevier-Saunders; 2011. p. 328–34.

[27]. Marini JJ, Wheeler AP. Indications and options for mechanical ventilation. In: Marini JJ, Wheeler AP, editors. Critical care medicine. 4th edition. Philadelphia: Lippincott Williams & Wilkins; 2010. p. 129–47.

[28]. Matthews NS, Hartke S, Allen JC Jr: An evaluation of pulse oximeters in dogs, cats and horses. Vet Anaesth Analg 30(1):3–14, 2003.

[29]. Marino PL: The ICU Book, ed 2. Baltimore, Williams and Wilkins, 1998.16. Powell LL: Causes of respiratory failure. Vet Clin North Am Small Anim Pract 32(5):1049–1058, 2002
[30]. Manning AM: Oxygen therapy and toxicity. Vet Clin North Am Small Anim Pract 32(5):1005–1020, v, 2002.
[31]. Mellema MS, Haskins SC: Weaning from mechanical ventilation. Clin TechSmall Anim Pract 15(3):157–164, 2000.
[32]. Pilbeam SP. Establishing the need for mechanical ventilation. In: Pilbeam SP, Cairo JM, editors. Mechanical ventilation physiological and clinical applications. St Louis (MO): Mosby; 2006. p. 63–80.
[33]. Pilbeam SP. Final considerations in ventilator set up. In: Pilbeam SP, Cairo JM, editors. Mechanical ventilation physiological and clinical applications. St Louis (MO): Mosby; 2006. p. 127–49.
[34]. Rivers E, Nguyen B, Havstad S, *et al*. Goal directed therapy in the treatment of severe sepsis and septic shock. N Engl J Me2001;345:1368–77.
[35]. Pilbeam SP. Initial ventilator settings. In: Pilbeam SP, Cairo JM, editors. Mechanical ventilation physiological and clinical applications. St Louis (MO): Mosby; 2006. p. 105–26.
[36]. West JB. Respiratory physiology: the essentials. 8th edition. Baltimore (MD): Lippincott Williams & Wilkins; 2008.