Artificial Ventilation of the critically ill patient

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This presentation is a review with some observations from experience gleaned from the provision of artificial ventilation for our small animal patients, particularly for thoracic surgery, intracranial trauma and in the treatment of snake envenomation.

As such from an Evidenced Based Medicine point of view, the information varies in its quality from basic physiological research to anecdotal practice experience. References are provided where applicable.

However, some really interesting recent developments in medical practice and in the animal research literature have occurred that will influence and change our clinical practice.
Artificial Ventilation of the critically ill patient

- Respiratory physiology texts referred to include:
  - Nunn’s Applied Respiratory Physiology 5th E, Lumb AB, 2000
  - Pathophysiology of Respiratory Disease – the essentials 3rd Ed. JD West 1987
  - Textbook of Medical Physiology 11th Ed, Guyton AC & Hall JE 2006
  - Lumb and Jones Veterinary Anaesthesia and Analgesia 4th Ed, Tranquilli WJ et al 2007
  - Veterinary Anaesthesia 10th Ed Hall LW and Clarke KW 2001
  - Textbook of Respiratory Disease in Dogs and Cats, LG King 2004
HISTORY OF ARTIFICIAL VENTILATION

Versalius in 1555 first showed that a pig with an open chest could be kept alive by intubating the trachea and intermittent inflation of the lungs. Toward the end of the 18th century there developed considerable interest in resuscitating people rescued from drowning. The Humane Society offered prizes for new ideas and apparatus for resuscitation.
HISTORY OF ARTIFICIAL VENTILATION

The rectal insufflation of tobacco smoke proved to be of no value.
Many ideas for equipment based on pumps and bellows were put forward. Unfortunately, failure to appreciate the dangers of high airway pressure led to deaths from tension pneumothorax. As a result, bellows resuscitation fell into disrepute.
Sub-atmospheric techniques were investigated
Interest developed in iron lung techniques, developing an intermittent external sub-atmospheric pressure to expand the thorax.
The polio epidemics of 1920s, 40s and 50s were a major stimulus to development of techniques for mechanical ventilation. Intermittent positive pressure ventilation (IPPV) was first described by Grundel and Waters in 1928. In the 1940’s Gordon Knight of the Royal Veterinary College introduced IPPV to clinical veterinary practice.
Terms used

- artificial ventilation = controlled ventilation
  - intermittent positive pressure ventilation (IPPV)
  - Iron lung
- positive end-expiratory pressure (PEEP)
- continuous positive airway pressure (CPAP)
definitions

compliance:

- the increase in lung volume for each unit of pressure applied
  - The slope of the pressure-volume curve
  - “stiff lungs” have low compliance
  - highly compliant lungs inflate easily
Compliance

- Much like the spaces between the coils of a slinky toy, alveoli are more expanded in the top most lung and collapsed in the dependant lung, irrespective of body position because of gravitational force.
- At high pressures and volumes the lung is stiffer, harder to inflate and the compliance is smaller as shown by the flatter slope of the curve.
Compliance

Pressure-volume curves of air filled lung vs saline filled lung (which abolishes surface tension)

Note
- open circle = inflation
- closed circles = deflation
- high compliance in saline filled lung
- lower hysteresis in saline filled lungs
Compliance

- At high pressures and volumes the lung is stiffer, harder to inflate and the compliance is smaller as shown by the flatter slope of the curve.

- The tendency of the lung to return to its resting volume i.e. elastic recoil, is due to 1) Geometric arrangement of elastic fibres 2) Surface tension of the liquid film lining the alveoli 3) Alveolar interdependence. The expanded thorax also has elastic recoil.

- The alveolar surface tension is greatly modified by the presence of surfactant. Saline ventilated lungs are much more compliant than air ventilated lungs.

- As saline abolishes surface tension forces, this graph comparing saline filled lungs to air filled lungs, shows that surface tension contributes a large part to the static recoil force of the lung.

![Graph comparing saline and air inflation]
Compliance

- Surfactant is largely responsible for hysteresis – the difference between the inspiratory and expiratory F-V curve.
- Surfactant decreases the surface tension in air filled alveoli thus increases compliance and reduces the work required for expansion.
- Surfactant, by maintaining a low surface tension in small surface areas, counters the tendency for small alveoli to empty into large alveoli.
- Finally surfactant by reducing surface tendency reduces the tendency to suck fluid into the alveolar spaces, thus protects against the transudation of fluid.

Consequences of the lack of surfactant are the pathophysiological features of Infant Respiratory Distress Syndrome (Hyaline Membrane Disease):
- stiff, low compliant lungs,
- areas of atelectasis and
- alveoli filled with transudate.
Compliance

- When providing artificial ventilation we aim to ventilate the patient in the middle of the Pressure-Volume curve where the lungs are inflated above FRC and a small amount of pressure produces a substantial change in volume.
minute volume:

- the volume of air inspired each minute equals tidal volume x respiratory rate

- Note that tidal volume = alveolar ventilation plus physiological dead space ventilation

- $\dot{V}_T = (V_A + V_D) \times \text{rate}$
minute volume:

- Importantly, minute alveolar ventilation is inversely proportional to the arterial partial pressure of carbon dioxide:

\[ P_{a\,CO_2} = K \times \frac{\dot{V}_{CO_2}}{\dot{V}_A} \]
This is one of the reasons capnography is so useful, as the top of the plateau is the concentration of carbon dioxide in mixed alveolar gas (End tidal CO$_2$ = ETCO$_2$).

Generally alveolar carbon dioxide partial pressure is almost equivalent to pulmonary arterial carbon dioxide partial pressure except in cases of severe V/Q abnormalities.

Normal P$_A$CO$_2$ in dogs is around 30-40mmHg and cats 20-30mm Hg.
Hence, hyperventilation results in low ETCO$_2$ and undersventilation results in high ETCO$_2$.
Indications for IPPV

- primary ventilatory failure
  - anaesthetic overdose
  - tick or snake envenomation
- inadequate ventilation secondary to disease
  - obesity
  - sepsis, SIRS, MOPS
- neuromuscular blockade
- open thoracic cavity
- thoracoscopy
- control of intracranial pressure
- acute lung pathology
  - pulmonary oedema
  - ARDS & IRDS
  - infection
  - severe aspiration/chemical pneumonitis
  - trauma: pulmonary contusion
- hypotensive anaesthesia
Types of Respiratory Failure

1) Hypoxaemic respiratory failure
2) Hypercapnic respiratory failure
3) Mixed
Hypoxaemic Respiratory Failure (Type 1)

- PaO$_2$ < 60mmHg with normal or low PaCO$_2$ → normal or high pH
- Most common form of respiratory failure
- Lung disease is severe to interfere with pulmonary O$_2$ exchange, but overall ventilation is maintained
- Physiologic causes: V/Q mismatch and shunt
- Caused by a disorder of heart, lungs or blood
Hypercapnic Respiratory Failure (Type II)

- PaCO$_2$ >50 mmHg
- Hypoxemia is always present
- pH depends on level of HCO$_3^-$
- HCO$_3^-$ depends on duration of hypercapnia
- Renal response occurs over days to weeks
- Causes: drugs, neuromuscular disease (MG, Polyradiculomyelopathy), toxins, venoms, severe lung disease, respiratory muscle fatigue (acute on chronic), high spinal injury, pleural space disease, partial URT obstruction, respiratory centre dysfunction, Chronic obstructive airways disease
Hypoxia must be treated as the first priority by the administration of oxygen.

However this will do nothing to improve the $P_{CO_2}$ and may make it worse (in patients who have lost their ventilatory sensitivity to carbon dioxide and are reliant upon hypoxic drive to maintain ventilation).

The only way to reduce the arterial $P_{CO_2}$ is to improve alveolar ventilation.
Treatment of Ventilatory Failure

- For relief of hypoxia due to hypoventilation only small increases in inspired oxygen concentration ($\text{Fi}_{\text{O}_2}$) are required.

- In hypoventilation up to an alveolar carbon dioxide ($P_{\text{CO}_2}$) of almost 100 mm Hg (13kPa), supplying an $\text{Fi}_{\text{O}_2}$ of 30% is sufficient to maintain a normal alveolar $P_{\text{O}_2}$.

- Clearly however, this is an unacceptable $P_{\text{CO}_2}$ and there is a limit when the provision of enriched inspired oxygen is not sufficient without improving the alveolar ventilation.
Figure 26.5 Alveolar $P_{CO_2}$ as a function of alveolar ventilation at rest. The percentages indicate the inspired oxygen concentration that is then required to restore normal alveolar $P_{O_2}$.
Essentially almost all anaesthetised spontaneously breathing patients hypoventilate.

It is not uncommon for spontaneously breathing anaesthetised horses to have a $P_{CO_2}$ of 60-80mm Hg but this evokes sympatho-adrenal responses including elevation of circulating catecholamines, BP, Cardiac output and contractility.

Patients (not anaesthetised) with chronic respiratory disease may have an arterial $P_{CO_2}$ as high as 60mm Hg

Dogs with $P_{CO_2}$ over 95mm Hg undergo narcosis

However, it is desirable to maintain $P_{CO_2}$ values within reasonable limits in all anaesthetised animals (ETCO$_2$ cats 20-30mm Hg, dogs 30-45 mm Hg, horses < 60mm Hg)
Indications for Ventilation

Abby weighs 76lbs
lives in Atlanta, Georgia, USA
record holder (if still alive):
the fattest cat in the world
Mechanical aspects of IPPV

diaphragmatic excursion:

- abdominal contents exert maximum pressure over the dependent part of the diaphragm
- with spontaneous ventilation, stretched diaphragm has a mechanical advantage
Mechanical aspects of IPPV

diaphragmatic excursion:

- with IPPV, dependent part of the diaphragm presents the greatest resistance to inflation
- preferential expansion of upper lung lobes early in inspiration.
- large tidal volumes favour recruitment of dependent alveoli & over ventilation of uppermost alveoli
Intrapleural pressure

a. spontaneous ventilation

a. controlled ventilation
adverse effects of IPPV

circulation

- reduction of blood flow to right atrium
- increase in pulmonary input impedance
- reduced capacitance of pulmonary bed
adverse effects of IPPV

circulatory effects minimised by:

- increase in tone of capacitance vessels
  - minimise depression of the sympathetic nervous system
  - adequate circulating volume
- minimise mean airway pressure
  - Inspiratory to expiratory time ratio I:E ratio < 1:2
  - abolition of thoracic & abdominal m. tone
- maximise inspiratory flow rate
  - “fast” & “slow” alveoli
adverse effects of IPPV

barotrauma

- High inspiratory pressures can lead to pneumothorax or pneumomediastinum

- **closed chest**: damage with inflation pressures > 70 cm H₂O

- **open chest**: damage with inflation pressures > 40 cm H₂O
adverse effects of IPPV

Question:
What inspiratory pressure are you generating when you provide IPPV to a patient?
adverse effects of IPPV

Answer:

Unless the airway pressure is measured with a manometer, you don’t know!
adverse effects of IPPV

Barotrauma or volutrauma?

- Hyperinflation (volutrauma) injures the diseased lung
- Overdistension is more insidious and ultimately more injurious than barotrauma
- Hyperinflation leads to gross oedema, increased lung weight and cellular damage
adverse effects of IPPV

Barotrauma or volutrauma?

- Total lung volume is reduced in pulmonary disease associated with trauma or sepsis
- Ventilation with a normal tidal volume may approach total lung capacity
- Damage is diffuse & heterogenous in the diseased lung. (Slow and fast alveoli)
adverse effects of IPPV

Volutrauma and the diseased lung

- Extensive research on ARDS and animal models of acute lung injury
- Lung injured animals subject to
  - high airway pressures & normal tidal volumes
    gross pathology changes similar to paired control
  - high tidal volumes led to gross oedema, increased lung weight & cellular damage
- Damage due to overdistension is synergistic with toxin-induced pulmonary endothelial injury
adverse effects of IPPV

Volutrauma and the diseased lung

- Strategies to limit tidal volume and airway pressure are advantageous:
  - limit tidal volume (25% fewer deaths in ARDS)
  - avoid high airway pressures,
  - permissive hypercapnia (not head trauma patients)
  - pressure limit ventilation
  - PEEP (provided normovolaemia)
  - Posture (prone responsive)
Volutrauma and the diseased lung

- **PEEP** (provided normovolaemia)
  - improves oxygenation by decreasing pulmonary shunt and dead space ventilation, often permitting a lowering of the inspired oxygen concentration
  - at low lung volumes cyclical airway opening & closure occurs and shearing injury results which is prevented by PEEP
  - alveolar recruitment
adverse effects of IPPV

In Summary:

There is a considerable differences in the ventilatory requirements of a patient with healthy lungs compared to that with lung damage or disease.

Provision of artificial ventilatory must match individual patient needs in order to optimise outcomes.
adverse effects of IPPV

Volutrauma and the diseased lung

Reviews:
adverse effects of IPPV

acid-base disturbance

- **over ventilation** → **respiratory alkalosis**
  - shift Hb-O\textsubscript{2} dissociation curve to the left
  - cerebral vasoconstriction

- **under ventilation** → **respiratory acidosis**
  - hypercapnia
  - risk of hypoxaemia (if air)
adverse effects of IPPV

hazards of breathing 100% oxygen

- absorption atelectasis
adverse effects of IPPV
hazards of breathing 100% oxygen

Figure 9.4. Reasons for atelectasis of alveoli beyond blocked airways (A) when O$_2$ and (B) when air is breathed. Note that in both cases, the sum of the gas partial pressures in the mixed venous blood is less than in the alveoli. In B, the P$_{O_2}$ and P$_{CO_2}$ are shown in parentheses because these values change with time. However, the total alveolar pressure remains within a few mm Hg of 760.
With the provision of 100% oxygen the partial pressure gradient across the alveolus is greatly increased and if the alveolus is partly obstructed, the absorption of oxygen literally collapses the alveolus as the gas diffusing into the pulmonary capillary blood does so at a rate faster than it is replaced.
100% oxygen
100% oxygen
50% oxygen 50% nitrogen
50% oxygen 50% nitrogen
(a) Blood flow

Breathing air

Breathing 100% O₂

1% shunt breathing O₂

PaO₂ 80 kPa (600 mmHg)

(b) Age 22

Breathing air

Breathing 100% O₂

10.7% shunt breathing O₂

PaO₂ 67 kPa (506 mmHg)

(c)

Unstable
Liable to collapse

Stable

Inspired oxygen concentration (%)

Inspired ventilation/perfusion ratio
The first picture presents a graph of blood flow to alveolar units of all possible V/Q ratios in a healthy young adult breathing room air compared to the blood flow vs V/Q ratio of the same subject breathing 100% oxygen. There is little difference. Note that there is little shunting of blood when breathing 100% O2 and thus the PaO2 is very high – 600 mm Hg.

The second graph depicts the same curves in a subject with minor lung disease, in this case age - the subject is 44 yrs. Note that the older subject breathing room air has a large tail to the curve on the left - many alveolar units have very low V/Q ratios compared to the young adult and these low V/Q units are receiving some blood flow. Provision of 100% O2 results in absorption atelectasis and collapse of these alveoli, thus the left side tail disappears however the shunt fraction increases dramatically to 10% and as a result the PaO2 falls to 500mm Hg.

The third graph rearranges this data to show the divide between stable alveoli and unstable alveoli (liable to collapse) is dependant on their V/Q ratio and the inspired oxygen tension. The lower the V/Q ratio the more liable an alveolus is to collapse even with an only slightly enriched inspired oxygen tension.
adverse effects of IPPV

hazards of breathing 100% oxygen

- absorption atelectasis

- pulmonary oxygen toxicity
  - onset after 12 hours of 100% O₂
  - 40% O₂ may be given indefinitely
  - death in 2-3 days
Avoid oxygen toxicity by using an oxygen and air blended gas mixture so that the inspired oxygen tension is no more than 50-60%.
adverse effects of IPPV

- drying of airway
  - worse with dry gases
  - paralyse tracheal cilia
  - inspissation of mucus

- ventilation associated pneumonia
  - Mortality in man approaches 25%
Management of the ventilated patient
Gaining control of respiration

1. hyperventilation
2. sedation
   - may be unnecessary
   - very light isoflurane anaesthesia
   - Injectable sedatives/anaesthetics
3. neuromuscular blockade with sedation/anaesthesia
4. tracheostomy
Hyperventilation

- The apneoeic threshold is the PaCO2 level at which spontaneous ventilation ceases.
- A PaCO2 reduction of 5 to 9 mm Hg from normal values through conscious hyperventilation (humans) or by artificial ventilation of sedated or anaesthetised animals produces apnoea.
- The distance between the resting PaCO2 and the apneoeic threshold is (5-9 mm Hg) is relatively constant irrespective of anaesthetic depth.
Sedation: very light isoflurane anaesthesia

Advantages:

- Bronchodilator
- Fast recovery (can easily test wean)
- Equipment readily available
- If healthy lungs, excellent method
- Other inhalational agents: Desflurane
Sedation: very light isoflurane anaesthesia

Disadvantages:

- Depresses Pulmonary Hypoxic Vasoconstriction, (the major reflex limiting the impact of shunt)
- In ventilated dogs with acute lung injury (oleic acid) introducing 0.25 to 0.5% isoflurane worsened the severity of
  - V/Q mismatch
  - shunt (increases from 38% to 42% and 48%)
  - the fall in $\downarrow P_{AO2}$ (from 72 mm Hg to 62 and 56 mm Hg)
  - $\downarrow CO$
  - $\downarrow O_2$ delivery (from 573 ml/kg/min to 529 and 505) (Putensen et al 2002).
- Dose dependant decrease cardiac output (little at 0.25%-0.5%)
Sedation: injectable drugs

• Opioid + benzodiazepine
  • fentanyl 0.2-1.0 ug/kg/min + midazolam 0.2-0.4 mg/kg/hour
  • Problems: Tolerance, tachypnoea, hyperthermia, active metabolites of morphine

• Propofol
  • Infusion
  • Problems: Tolerance, lipidaemia, catastrophic propofol infusion syndrome in man

• Pentobarbitone
  • 1-2 mg/kg/hour
  • May be used with diazepam CRI especially during weaning
  • Problems: Hypotension, prolonged recovery, hyperreflexia with use < 2 days, withdrawal dysphoria 3-7 days
management of the ventilated healthy patient

- **ventilation parameters:**
  - peak inspiratory pressure 10-20 cm H$_2$O
  - respiratory rate 10 - 20 breaths/min
  - tidal volume 8-12 ml/kg
  - Minute volume 150-250ml/kg/min
  - I:E ratio 1:2 or 1:3
  - ETCO$_2$ (mmHg) 30-40 dog, 20-30 for cats

- **artificial sigh**
  - reverse alveolar collapse
  - 30-35 cm H$_2$O & hold for 3 seconds
  - Repeat 30-60 minutes
management of the ventilated patient with lung disease

- ventilation parameters:
  - peak inspiratory pressure slowly increased to 35 cm H$_2$O
  - respiratory rate up to 60 breaths/min
  - tidal volume 4-8 ml/kg
  - ETCO$_2$ up to 50 mmHg (permissive hypercapnia)
  - I:E ratio 1:2
  - PEEP up 20 cm H$_2$O
  - Artificial sigh 40 cm H$_2$O for 3 sec q30mins
management of the ventilated patient

- airway maintenance
  - humidified gas/nebulization
  - regular suction, postural drainage & chest percussion every 4 hrs.
    - give 100% O2 for 3 mins prior to suctioning
    - perform chest coupage,
    - inject saline 0.2ml/kg into ET tube,
    - hyperinflate a few times,
    - introduce suction catheter (dia < 50% ET tube) as far as it will go,
    - Tx for 5 secs, repeat if productive
  - change Ventilator tubing and Endotracheal tubes (low pressure cuffs) every 24 hrs
  - Inflate cuff only enough to stop leak, change the position of the cuff every 4 hours (flush then suction out mouth and pharynx first)
management of the ventilated patient

- maintain circulating volume
  - minimise CVS effects of IPPV
  - minimise viscosity of secretions
- Position
  - Reposition every 4 hrs
  - Passive ROM exercise all joints
  - Sternal or 45o roll to either side is best for lung function
- Mouth Care
  - Wash and suction every 4hrs with sterile saline
- Eye care
  - Corneal drying Tx: artificial tears & bland ointment q2hrly
management of the ventilated patient

positioning the patient:

either

- sternal recumbency

or

- 45° to either side of sternal

will minimise ventilation/perfusion mismatch
management of the ventilated patient

● Bladder care
  ■ Human infant absorbent nappies
  ■ If not urinating
    – Express bladder 3x daily
    – In dwelling urinary catheter (flush prepuce or vestibule every 8 hrs with dilute betadine or chlorhexidine)

● Colon
  ■ Clip hair around anus
  ■ Enemas daily if required

● Nosocomial Infection
  ■ Wash your hands!
  ■ Sterile tube, breathing circuit and nebuliser/humidifier

● Nutrition
  ■ Early nutrition
  ■ Tube feeding (nasal, oesophageal, gastric)
  ■ Gastric stasis is common, check for gastric residue before feeding (<10ml/kg approx), Use prokinetics if needed
monitoring the ventilated patient

- observation of patient & ventilator
monitoring the ventilated patient

- observation of patient & ventilator
- blood gas, blood urea, PCV
monitoring the ventilated patient

- observation of patient & ventilator
- blood gas, blood urea, PCV
- capnography
monitoring the ventilated patient

- observation of patient & ventilator
- blood gas, blood urea, PCV
- capnography
- pulse oximetry
monitoring the ventilated patient

- observation of patient & ventilator
- blood gas, blood urea, blood gas
- capnography
- pulse oximetry
- temperature
monitoring the ventilated patient

- observation of patient & ventilator
- blood gas, blood urea, blood gas
- capnography
- pulse oximetry
- Temperature
- Hydration
weaning from the ventilator

- As soon as an animal is stable on a ventilator, frequently test if less aggressive ventilator settings can be used, try
  - reducing the PEEP,
  - increasing the I:E ratio,
  - decreasing the peak inspiratory pressure,
  - if the ventilator has patient triggered functions, try introducing one of these.

- Ability to wean depends on the problem the patient was ventilated for and the functions available in the ventilator being used.
weaning from the ventilator

- **Patients with intracranial disease**
  - No sedation for intubation and ventilation
  - As they regain sufficient consciousness to object to intubation they are probably able to adequately ventilate

- **Patients with neuromuscular disease**
  - Sedation often contraindicated and tracheotomy is required so return of muscular strength can be assessed. Return of strength parallels ability to ventilate

- **Patients with pulmonary paranchymal disease**
  - Wean when only minimal ventilator settings required to maintain oxygenation
  - If PaO2 > 80mmHg, PEEP < 4cmH2O and FiO2 < 40%
  - Or if PaCO2 < 45mm Hg with normal tidal volume
weaning from the ventilator

- **Ventilators with pressure support weaning modes**
  - Intermittent mandatory ventilation (IMV)
  - Spontaneous breathing with Continuous positive airway pressure (CPAP)
  - Progressively ↓ ventilator rate so patient has to trigger more breaths (assisted breathing with gradual increase in the trigger pressure)
  - Try spontaneous breathing trials of increasing duration

- **Ventilators without weaning modes**
  - Take patient off the ventilator for short periods of spontaneous breathing trials.
  - Progressively increase the weaning time as the patient’s condition and strength permits
weaning from the ventilator

- reduce sedation
- connect to anaesthetic machine
- give 100% O2 for 5 minutes before weaning
- reduce ventilatory rate to 2 bpm
- assess adequacy of ventilation
- if OK give oxygen supplementation - via nasal catheter (100-150 ml/kg/min)
weaning from the ventilator

evidence of unsuccessful weaning:

- elevated respiratory rate
- anxiety
- minute ventilation < 150 m/kg
- $\text{SpO}_2 < 90$
- $\text{PaCO}_2 > 50-60 \text{ mmHg}$
- Struggling movements but loss of rhythmic ventilation pattern
Methods for Ventilation

- Manual
- Mechanical
- Mechanical/electrical
- Microprocessor
manual ventilation

circle absorber

Bain
mechanical ventilators

respiratory cycle is composed of:

- inspiratory phase
- cycling from inspiration to expiration
- the expiratory phase
- cycling from expiration to inspiration
mechanical ventilators
cycling from inspiration to expiration

- **time cycling**: (eg. Campbell)
  - inspiration terminated when a preset time has elapsed
  - may be volume or pressure limited

- **pressure cycling**: (eg. Bird Mk 7)
  - inspiration terminated when a preset pressure is reached at the mouth
  - increase in airway resistance or reduction in compliance result in decreased tidal volume
mechanical ventilators
cycling from inspiration to expiration

- Time cycled volume limited
  eg Ohmeda 7000 anaesthetic ventilator
  - Electronic controls
  - volume assured ventilation
    (within pressure limits)
  - Can vary minute volume, rate, I:E ratio
  - Minute volume, tidal volume & I:E ratio
    vary depending on anaesthetic machine fresh gas flow rate
mechanical ventilators
cycling from inspiration to expiration

- **Volume/time/pressure/flow cycling**
  (eg. Evita ventilator)
  - advanced microprocessor machine
  - volume assured pressure limited ventilation
  - can be patient triggered by flow or pressure as little as 0.5cm water
  - Set volume, then machine analyses the patient’s lung compliance curve, generating a pressure-flow time curve that delivers the set volume but at the lowest possible inspiratory pressure. Ideal for stiff lungs (ARDS).
  - Known as Pressure controlled volume guaranteed ventilation
nearly all ventilators act as pressure generators during the expiratory phase

passive flow along pressure gradient

(expiratory pressure generated by ventilator)

-atmospheric

-positive (PEEP)

-negative *(NEVER indicated)*
mechanical ventilators
cycling from expiration to inspiration

- Time cycling
- Patient trigger
plumbing

connection to the patient:

- **direct**
  - patient breathes ventilator driving gas
  - not suitable for all types of ventilators
Patient connected directly to ventilator
connection to the patient:

- **direct**
  - patient breathes ventilator driving gas
  - not suitable for all types of ventilators

- **via anaesthetic breathing system**
  - replace bag with ventilator connection
  - close breathing system spill valve
  - deliver oxygen mixture to the breathing system
connection to the patient:

via anaesthetic breathing system

Method 1) The bag in the bottle:

There are two separate gas sources, one driving the ventilator connected to a bottle, the other the patient breathing system connected to a bellows (within the bottle).
Close the spill valve!!
Fresh gas

ventilator controlled spill valve

ventilator

Driving gas for ventilator
Method 1) The bag in the bottle:
plumbing

connection to the patient:

- via anaesthetic breathing system

Method 2) Direct connection

- Using a connection pipe that has a volume at least 2x the tidal volume used.

- There is still 2 gas sources, one driving the ventilator, the other the patient breathing system
My ventilator doesn’t have a bellows!!
There is an easy solution:

Simply connect the ventilator to the attachment for the rebreathing bag using 22mm corrugated tubing using 10cm of tubing per kilogram weight.
Spill valve

fresh gas

inspiration
Spill valve

fresh gas

inspiration
Spill valve

fresh gas
Spill valve

expiration

fresh gas
Spill valve

fresh gas

expiration

ventilator
Spill valve

expiration
Spill valve

expiration

fresh gas
P

Spill valve

fresh gas

expiration
management of the ventilated patient

CAUTION: compliant tubing

- compliant tubing used in the patient breathing system will “absorb” a proportion of the delivered tidal volume

- can be very significant with long lengths of tubing or small patients
Positive End-expiratory pressure

- denotes an airway pressure that is kept above atmospheric pressure at the end of the expiratory cycle
PEEP

Respiratory effects

- Increase FRC
- Opening of previously closed alveoli
- Transfer of oedema fluid to interstitial compartment
- Reduces airway resistance
  (NB inverse relationship between lung volume and airway resistance)
- Only slight increase in dead space/tidal volume ratio
- Barotrauma if PEEP > 20cmH2O
PEEP

- **Cardiovascular effects**
  - Pulmonary shunt is decreased
  - Decrease in cardiac output thus increase O2 extraction by tissues and a fall in mixed venous P O2)
  - Overall minimal rise in PaO2
  - In LV failure, PEEP squeezes the heart. Decreases diastolic stretch in disease will improve cardiac output
  - Adverse effects dramatically exacerbated if hypovolaemic
PEEP

- Using PEEP
  - Keep PEEP < 10cm H2O
  - Maintain preload
    - normovolaemia
    - adequate vascular resistance
  - Monitor
    - SpO2
    - PAO2 - PaO2 difference
    - Delta down in the arterial pressure trace
Monitoring PEEP

- **SpO2**
  
  - Determined by respiratory function and cardiac output
Monitoring PEEP

- $P_{A\text{O}_2} - P_{a\text{O}_2}$ Difference
Monitoring PEEP

Ideal Alveolar-arterial O$_2$ Difference

O$_2$-CO$_2$ diagram

i = hypothetical composition of alveolar gas and end-capillary blood when no V/Q inequality is present
Monitoring PEEP

Ideal Alveolar-arterial O_2 Difference

O2-CO2 diagram

- “i” is the hypothetical composition of alveolar gas and end-capillary blood when no ventilation-perfusion inequality is present.
- As inequality develops, the arterial “a” and alveolar “A” points diverge along their respective R lines (respiratory exchange ratio).
- The mixed alveolar-arterial PO2 difference is the horizontal distance between the points.
- In practice the ideal Alveolar – arterial O2 difference is calculated by using the alveolar gas equation which relates the Alveolar P02 to the composition of the inspired gas, respiratory exchange ratio and the PCO2 (arterial PCO2 is taken to be the same as Alveolar PCO2 since the line along which point I moves is nearly horizontal).
- This ideal A-a O2 difference is caused by units between i and v, that is, those with low ventilation-perfusion ratios.
Monitoring PEEP

- Arterial pressure wave form
  Delta down or the wavering A-line sign
Arterial pressure wave form

PEEP 7cm

Note: Variation in peak systolic pressure synchronous with ventilator rate: the wavering A-line sign

PEEP + Hypovolaemia or PEEP 15cm or Over-inflation
Methods to Assure Oxygenation in patients requiring ventilatory support

- Intubate, commence ventilation with FiO2 100% and progressively wean to non-toxic levels <50%, maintaining SPO2 > 90%.
- In the diseased lung note that inhaled anaesthetic agents can worsen V/Q abnormalities thus delay weaning from high inspired FiO2
- Provided no hypovolaemia or hypothermia and haemodynamically stable, introduce “best PEEP” (that which increases SPO2 with minimal depression of Cardiac Output)
- Trial different settings to optimise patient oxygenation, oxygen delivery and minimise depression of cardiac output
Learning to use a Ventilator

- Read the machine manual!
- Chose in which mode you wish to use the ventilator
  - Direct patient ventilator
  - Anaesthetic machine ventilator
  - Using a direct patient ventilator to run as an anaesthetic machine ventilator
    - Bag in the bottle (2 separate circuits)
    - Connect ventilator by a long 22mm corrugated tubing to the connection site of the rebreathing bag in the anaesthetic machine’s patient breathing circuit (circle, Bain or T piece)
- Connect a 2L rebreathing bag to the patient end of the breathing circuit instead of a real patient
- If using an anaesthetic machine, close the pop-off valve
Learning to use a Ventilator

- If your machine gives you various ventilation options for either controlled ventilation or assisted ventilation (where the patient initiates the breath) including IPPV, IMV, PEEP, SIMV CPAP, select IPPV to start with.
- Set the ventilator settings where possible to match a hypothetical dog (e.g., tidal volume 10ml/kg, RR 15, I:E 1:3, Max inspiratory pressure 20cm H20). For controls with no scale, just set midway.
- Turn on the supply gas and switch on the electrical supply.
- If alarms sound, change the alarm limits to accept the settings you have chosen to use.
- Adjust the various dials to see what effects occur on the “patient” being ventilated.
Indications for IPPV

- primary ventilatory failure
  - anaesthetic overdose
  - tick or snake envenomation
- inadequate ventilation secondary to disease
  - obesity
  - sepsis, SIRS, MOPS
- neuromuscular blockade
- open thoracic cavity
- thoracoscopy
- control of intracranial pressure
- acute lung pathology
  - pulmonary oedema
  - ARDS & IRDS
  - infection
  - severe aspiration/chemical pneumonitis
  - trauma: pulmonary contusion
- hypotensive anaesthesia
Outcomes of Artificial Ventilation

- Neuromuscular disease patients:
  50% successfully weaned and of these 50% discharged alive

- Hyoxaemic respiratory failure (lung disease)
  25% successfully weaned and 15% discharged alive

- Australian snake envenomated patients (at STAH)
  Approximately 80-90% successfully weaned and discharged
The End

South Tamworth Animal Hospital
Some Useful Additional Information
Uneven time constants of ventilation
Regional Differences In Ventilation: The effect of lung volume

At low lung volumes, the distribution of ventilation is reversed, the upper regions ventilate better than the lower regions.
The Henderson-Hasselbalch equation

\[ \text{pH} = \text{pK}_A + \log \frac{[\text{HCO}_3^-]}{[\text{CO}_2]} \]

\[ \text{pH} = 6.1 + \log [\text{HCO}_3^-]_{\text{mmol/L}}^{0.03 \times \text{PaCO}_2} \]

\[ \text{pH} = 6.1 + \log \frac{\text{kidney}}{\text{lung}} \]
Figure 30-1. Titration curve for bicarbonate buffer system showing the pH of extracellular fluid when the percentages of buffer in the form of $\text{HCO}_3^-$ and $\text{CO}_2$ (or $\text{H}_2\text{CO}_3$) are altered.
Figure 30-2. Change in extracellular fluid pH caused by increased or decreased rate of alveolar ventilation, expressed as times normal.
Figure 30–11. Acid-base nomogram showing arterial blood pH, arterial plasma $\text{HCO}_3^-$, and $\text{PCO}_2$ values. The central open circle shows the approximate limits for acid-base status in normal people. The shaded areas in the nomogram show the approximate limits for the normal compensations caused by simple metabolic and respiratory disorders. For values lying outside the shaded areas, one should suspect a mixed acid-base disorder. (Adapted from Cogan MG, Rector FC, Jr.: Acid-Base Disorders in the Kidney, 3rd ed. Philadelphia: W. B. Saunders Co., 1986.)