WHY VENTILATE?

Broadly speaking, patients that have difficulty ventilating and/or difficulty oxygenating could benefit from mechanical ventilation. Hypoventilation for the purposes of this article is defined as partial pressure of carbon dioxide (PaCO₂) in arterial blood that is greater than 60 mmHg. Oxygenation difficulties may exist when the partial pressure of oxygen (PaO₂) in arterial blood falls below 60 mmHg on room air, and especially if it remains low despite oxygen supplementation.

Under anaesthesia with volatile agents or injectable agents (especially if potent opioids such as fentanyl or remifentanil are administered by infusion), hypoventilation occurs when spontaneous breathing becomes excessively depressed. In addition, the administration of neuromuscular blockers will abolish the patient's ability to breathe spontaneously.

In the intensive care setting, mechanical ventilation of the lungs may be required to support patients with a variety of conditions (eg, pulmonary parenchymal diseases such as acute respiratory distress syndrome or pulmonary contusions) or problems that affect ventilation (such as cervical spinal injuries interfering with phrenic nerve – and therefore diaphragmatic – activity, or neuromuscular disorders such as tetanus or botulism). Some patients, such as young foals with pneumonia, may require ventilatory support because they are in danger of respiratory failure due to the enormous energy cost of breathing and their rapidly fatigued respiratory muscles.

Indications for ventilation

- Failure to ventilate (PaCO₂ > 60 mmHg)
- Failure to oxygenate (PaO₂ < 60 mmHg)
- Respiratory muscle fatigue
The requirements for ventilatory support differ between anaesthetised and intensive care patients. The latter usually demand long-term support and the use of humidified gases; consideration should also be given to the potential risk of oxygen toxicity. The ability to ventilate a patient’s lungs also has a place in emergency and resuscitation medicine – for example, oxygen demand valves and self-inflating bags with one-way valves to prevent rebreathing are readily available for use in this context.

PHYSIOLOGICAL ASPECTS OF MECHANICAL VENTILATION

To produce lung inflation, a ventilator either intermittently forces air into the lungs down a positive pressure gradient, or allows air to be sucked into the lungs down a negative pressure gradient by intermittently expanding the thoracic cage in a local subatmospheric pressure environment. As most ventilators in use today are positive pressure ventilators, artificial ventilation of the lungs has virtually become synonymous with the term intermittent positive pressure ventilation (IPPV). Intermittent negative pressure ventilators, such as the iron lung and chest cuirass, and positive–negative pressure ventilators, such as high frequency oscillators, will not be discussed further in this article.

HARMFUL EFFECTS OF IPPV

Reduced cardiac output

During normal inspiration, the negative pressure generated within the thorax results in air being sucked into the lungs and helps to augment the venous return to the heart from the great veins – the so-called ‘thoracic pump’. Increasing the positive pressure within the chest during inspiration can reduce the venous return to the heart and, thereby, reduce the cardiac output. During lung inflation under positive pressure, the pulmonary capillaries are compressed. This initially encourages their drainage to the left atrium, and so left heart venous return and cardiac output are momentarily increased. However, the increase in intrathoracic pressure effectively increases pulmonary arterial resistance, which, combined with a decrease in right heart venous return, reduces pulmonary blood flow and eventually venous return to the left atrium – and, hence, reduces systemic cardiac output towards the end of inspiration. Also, the rise in intrathoracic pressure essentially leads to cardiac tamponade, affecting the right side in particular, which further limits cardiac filling and, therefore, output.

Patients with poorly compensated cardiac disease (especially right heart failure) may not tolerate IPPV well and so caution is advised in such cases.

With IPPV, the increase in central venous pressure secondary to hindered venous return to the heart results in increased intracranial pressure and, therefore, reduced cerebral perfusion pressure. However, changes in PaCO₂ in the blood also have consequences for cerebral perfusion, and can affect sympathetic tone and therefore cardiac output – see later. Cerebral perfusion pressure depends on the difference between mean arterial blood pressure and intracranial pressure.

Uneven ventilation

In many recumbent/anaesthetised patients, including those with healthy lungs, the distribution of the inflating gas within the lungs is not uniform – and, hence, the ‘normal’ relationship of pulmonary ventilation to pulmonary perfusion is disturbed.

Insufficient ventilation of adequately perfused parts of the lungs represents a situation of increased physiological shunt (venous right-to-left shunt). When pulmonary venous blood from normal and ‘shunt’ areas of the lungs mixes (‘venous admixture’) as it returns towards the heart, some desaturation can occur, which, if severe enough, may lead to systemic arterial hypoxaemia. Although hypoxic pulmonary vasoconstriction helps to minimise such shunts in conscious healthy patients, many anaesthetic agents (particularly volatile gases) depress this protective mechanism.

Adequate ventilation of insufficiently perfused parts of the lungs represents a situation of physiological dead space (‘wasted ventilation’), which can result in inadequate excretion of carbon dioxide and, hence, hypercapnia.

If the lungs are diseased so that different lobes have different compliances and resistances to expansion, uneven distribution of ventilation is even more likely.

Lung damage

When IPPV is properly conducted, the probability of alveolar rupture in healthy lungs is quite small. (For comparison, each act of coughing produces an intrapulmonary pressure that may be as high as 80 to 90 cmH₂O, while straining at defection may produce even higher pressures.)

Experimentally, the pressure required to rupture exposed and unsupported lungs of various mammals has been found to be about 40 to 80 cmH₂O, while a pressure of 80 to 140 cmH₂O is required when the lungs are supported by the thoracic cage and abdominal musculature of the living animal. Following on from this, the maximum safe intrapulmonary pressure in the intact mammal with healthy lungs was determined to be about 70 cmH₂O (note that it is hard to exceed 40 to 60 cmH₂O by squeezing the thin black rubber bag of an anaesthetic breathing system). In experimental animals, rupture was found to occur into the mediastinum (pneumomediastinum), with subsequent passage of air up the fascial planes into the neck region (subcutaneous emphysema), and into the retroperitoneal space (pneumoretroperitoneum). At higher pressures, air emboli and intrapulmonary haemorrhages were also documented.

Many older texts refer to lung damage by barotrauma (excessive pressure), but this term has become less popular in favour of the term volutrauma, as it is now considered that high inspiratory pressures are probably not directly to blame; instead the secondary effects of over-inflation (increased volume) are more important. As our understanding of pulmonary structure and function has increased, the ability to cause ‘cellular damage’ to the lungs by applying IPPV has been recognised. Ventilator-induced lung injury includes components of mechanical damage (baro/volutrauma and atelectrauma) and inflammatory injury (biotrauma) – see box on page 188.

For long-term ventilation and even in spontaneously breathing animals, the inspired fraction of oxygen must be considered due to the risk of developing oxygen toxicity. High concentrations of oxygen (>60 per cent) in the inspired gases result in damage to the lungs caused by the production of oxygen-derived free radicals. Alveolar cellular injury can begin within six hours, and progress on longer exposures. In experimental studies, animals exposed to 100 per cent oxygen died within 48 to 72 hours (average 54 hours).

Although lung damage rarely occurs in healthy lungs until inspiratory pressures exceed 40 cmH₂O, it is wise not to exceed peak inspiratory pressures of 15 to 25 cmH₂O in order to minimise adverse cardiovascular effects and to prevent injury to the lungs (eg, in the face of pre-existing but as yet undiagnosed pulmonary disease).
Components of ventilator-induced lung injury

Volutrauma
Volutrauma describes the overdistension of ‘normal’ alveoli such that alveolar wall stress failure may occur. Overdistension of alveoli also results in associated capillaries being overstretched (and potentially also suffering stress failure).

Atelectotrauma
Atelectotrauma describes the shear stresses acting on the walls of ‘unstable’ alveoli, which tend to collapse fully during expiration only to be ‘snapped open’ again during the next inspiration. Alveoli can exist in this state when the functional residual capacity (FRC) of the lung (ie, the volume of gas remaining in the lungs at the end of a normal exhalation, which is important for continued gaseous exchange during the end expiratory pause) has fallen below the closing capacity of the lung (ie, the lung volume at which small airways and alveoli start to collapse). This situation may occur under anaesthesia (when FRC reduces), in disease and in neonates, which have a relatively high closing capacity compared with their FRC.

Biotrauma
Stretching/shearing forces on the alveolar walls and vascular endothelium of alveolar capillaries (ie, both volutrauma and atelectotrauma) can result in alveolar epithelial cell damage with consequent reduction in surfactant production, increased epithelial–endothelial barrier permeability with consequent alveolar flooding, and biotrauma (the activation of an inflammatory response within the lung). This inflammatory response can even result in pathology akin to acute respiratory distress syndrome (ARDS), otherwise known as ‘shock-lung’.

Another problem associated with lung ventilation, especially if cool, dry gases are supplied for more than a few hours, is loss of heat and moisture from the respiratory tract. Mucus secretions become more viscous and are cleared less effectively because ciliary function is depressed by cooling. Secretions and microbes can therefore accumulate in the lower respiratory tract and encourage infections to develop. Uncleared secretions may also interfere with gaseous exchange.

Blood gas and acid-base disturbances
Inadequate ventilation results in carbon dioxide retention, the development of hypercapnia and a fall in blood pH (respiratory acidosis). Hypercapnia, in turn, leads to an increase in sympathetic tone and, hence, an increase in circulating catecholamines. While this may improve arterial blood pressure and cardiac output, catecholamines are also arrhythmogenic, especially in the presence of halothane.

High arterial PaCO₂ can cause acute cerebral vaso-dilation, which can raise intracranial pressure and may reduce cerebral perfusion. Excessively high PaCO₂ in the blood (eg, ≥98 mmHg) also produces central nervous system depression (in the same way that carbon dioxide can be used for euthanasia). Thus, the depth of anaesthesia must be closely monitored.

Overventilation results in excessive elimination of carbon dioxide, while also usually promoting oxygenation of the blood. Hypocapnia will raise the blood pH, leading to respiratory alkalosis. In addition, the reduction in sympathetic tone may augment the decrease in cardiac output due to the effects of raised intrathoracic pressure.

Intracranial and intraocular pressure alterations
IPPV results in cyclical damming back of venous return to the heart. The increase in central venous pressure can lead to increases in intracranial and intraocular pressures. Changes in arterial blood carbon dioxide tension can also affect the vascular tone in these organs and thus their intracompartmental pressures.

If the arterial PaCO₂ falls below around 20 mmHg, the cerebral blood vessels tend to vasoconstrict severely, resulting in an acute reduction in intracranial pressure. In these circumstances, cerebral perfusion tends to be reduced despite a theoretical increase in cerebral perfusion pressure because of the high cerebral vascular resistance. Cerebral hypoxia may follow and anaesthesia will seem to deepen.

Fluid imbalances
Through the influences of IPPV on cardiac output there tends to be activation of the renin–angiotensin–aldosterone and sympathetic nervous systems. Antidiuretic hormone (ADH) secretion is also increased. IPPV is documented as being one of the causes of the syndrome of inappropriate ADH (SIADH) release. Atrial natriuretic peptide (ANP) release is inhibited by reduced filling/stretching of the atria. The overall outcome is that urine output falls in the face of fluid retention, especially with longer term ventilation.

Renal and hepatic effects
The reduced cardiac output and venous congestion of abdominal viscera can compromise renal and splanchnic perfusion. IPPV is said to promote some redistribution of intrarenal blood flow and result in reduced hepatic portal blood flow, both of which may alter drug metabolism and elimination.

Meteorism
If IPPV of a patient’s lungs is delivered via a face-mask, there is always the risk of inflating the stomach – so-called ‘meteorism’. This increases the risk of gastric content reflux and aspiration, and also results in an increase in intra-abdominal pressure. The latter can have serious consequences both for the chest (through diaphragmatic ‘splinting’) and for abdominal viscera through what is described as the abdominal compartment syndrome. Gastric rupture is also a possibility.

MINIMISING THE HARMFUL EFFECTS OF IPPV

Cardiovascular effects
Normal healthy animals usually compensate for the reduction in cardiac output by increasing their sympathetic tone, resulting in an increase in heart rate and vascular tone. However, the degree of compensation possible can be reduced by:

- General anaesthesia. Baroreceptor reflexes tend to be dampened, and the ‘deeper’ the anaesthesia, the poorer the baroreflex responses;
- Hypovolaemia. Compensatory responses may already be at work (eg, hypovolaemic animals already have high sympathetic tone);
- ‘High epidural’ with local anaesthetic. The sympathetic tone is reduced to the caudal part of the animal. If the block reaches far cranially, the cardioaccelerator fibres may also be blocked, so no increase in heart rate is possible either. Animals with spinal injuries often have poor sympathetic tone to areas caudal to the injury;
- Hypothermia/pyrexia. In order to encourage heat loss, peripheral vasodilation counteracts sympathetically induced vasoconstriction.
Monitor anaesthetic depth closely when instituting IPPV in patients receiving volatile anaesthetic agents, as the plane of anaesthesia can deepen, which can compound the decline in cardiovascular function.

Sometimes, intravenous fluid therapy and positive inotrope administration may be required to offset the cardiovascular effects of IPPV.

The magnitude of the positive pressure and the time for which it is applied to the lungs – collectively termed ‘mean intrapulmonary pressure’ – is of huge importance to the cardiovascular system. The lower the mean intrapulmonary pressure during the respiratory cycle, the less marked the cardiovascular effects will be. This mean pressure is not the arithmetic mean between the highest and lowest pressures, but is the mean of a large number of equally spaced (in time) instantaneous readings of the pressure within the lung during one respiratory cycle.

**Lung damage**

In a patient with healthy lungs, short-term mechanical ventilation during anaesthesia does not appear to induce any significant lung inflammation, even when relatively high tidal volumes are employed, with or without positive end expiratory pressure (PEEP). (Anaesthetic agents themselves may well have an immunomodulatory effect!) However, similar ventilation strategies in injured or infected lungs may augment the pre-existing inflammation. A ‘lung protective’ ventilation approach is now commonplace in human medicine, especially in emergency and intensive care settings. This favours ‘low stretch’ – that is, the delivery of smaller tidal volumes, but at a more frequent rate to achieve adequate delivery of minute ventilation. At the extreme of lung protective ventilation strategies is the use of high frequency jet ventilation or high frequency oscillation, both of which impose less shear forces on the walls of alveoli. In order to maintain adequate FRC with lung protective ventilation strategies, however, high expiratory pressures are often employed (see below).

The maintenance of an adequate FRC is important for prolonged ventilation. This is referred to as the ‘open lung’ concept and can also be important for animals under anaesthesia, in which it is achieved by delivering an occasional ‘sigh breath’ to spontaneously breathing patients (as anaesthetised animals cannot yawn or sigh).

When delivering small tidal volumes, the maintenance of an adequate FRC was thought to be ensured by certain ‘alveolar recruitment strategies’, such as:

- ‘Sigh breaths’ provided every 20 minutes at twice the normal tidal volume or by inflating the lungs up to a peak inspiratory airway pressure of 30 cmH₂O or
- ‘Vital capacity manoeuvres’ provided every 20 minutes by inflating lungs to an airway pressure of around 40 cmH₂O and holding for seven to eight seconds.

Although these manoeuvres could successfully recruit (reopen) closed alveoli, atelectasis would recur quite quickly. It was subsequently considered that, if some form of resistance was applied to exhalation so that the newly recruited alveoli would not reclose, the ‘open lung’ could be maintained.

The application of either continuous positive airway pressure (CPAP) to spontaneously breathing patients or PEEP to ventilated patients is now commonplace. These are preferable to the occasional manoeuvres outlined above for maintaining or increasing FRC, as they both result in alveoli being ‘splinted’ open, thus reducing the repetitive stretching open/collapsing closed of unstable alveoli and also the degree of atelectotrauma and biotrauma. However, applying positive pressure during the expiratory phase of ventilation does result in an increase in the mean intrapulmonary pressure, and so can be detrimental to cardiac output.

Pulmonary compliance has static and dynamic components, and varies slightly throughout the normal lung, depending on topographical location. The graph above shows a pressure–volume curve for a ‘normal’ lung. The gradient represents the dynamic compliance, and two points become apparent: the upper and lower inflection points. There is the risk of alveolar overdistension and rupture above the upper inflection point, while unstable alveoli are likely to close below the lower inflection point. In terms of ventilation strategy, the aim is to keep to the linear part of the curve. In the case of diseased lungs, these two points may lie closer together.

Protective lung ventilation strategies, especially those incorporating high frequency/oscillation, sometimes result in carbon dioxide retention (so-called permissive hypercapnia), but this is usually well tolerated by patients.

Prolonged ventilation with >60 per cent inspired oxygen leads to the development of oxygen toxic-

### Ways to minimise mean intrapulmonary pressure

- Do not maintain positive pressure for longer than is necessary to deliver an adequate tidal volume
- Aim for a relatively long expiratory period (passive deflation), with an inspiratory to expiratory ratio of 1:2 to 1:3 (or less)
- Infl ate the lungs with rapid flows of gas (but not too rapid as even distribution of gas to the lungs may be compromised)
- Minimise resistance to gas flow. Use the largest diameter endotracheal tube possible. This is particularly important during passive exhalation. Consider tube connections – try not to have too many angles/sharp bends
- Minimise the dead space in both the patient (eg, by intubating the trachea) and the apparatus, as this reduces the volume and pressure delivery requirements of the ventilator
ity. Although less of a problem for anaesthesia (where anaesthetic times are relatively short), oxygen toxicity becomes an issue in the intensive care setting where, in some patients, high inspired oxygen percentages are required for a long time to maintain arterial blood PaO2 at adequate levels for tissue oxygen delivery.

Under anaesthesia, many patients receive nearly 100 per cent oxygen (unless nitrous oxide or air are included in the fresh gases delivered from the anaesthetic machine). While this should ensure adequate oxygenation of arterial blood and can help overcome minor degrees of shunt (up to about 30 per cent of cardiac output), it may be inadequate to overcome hypoxaemia in the case of larger shunts (up to 50 per cent). This may be seen in anaesthetised horses in dorsal recumbency, for example. However, the provision of such high inspired oxygen concentrations has other consequences. It may result in the development of alveolar collapse/closure through ‘absorption atelectasis’ (also known as ‘resorption atelectasis’), which further adds to ventilation/perfusion mismatching. Thus, the inclusion of other, less soluble gases (eg, nitrogen, helium or nitrous oxide), which can help to ‘split open’ the alveoli, should be considered. Inspired oxygen of 30 per cent is usually sufficient for healthy anaesthetised small animals to offset the effect of small shunt fractions (up to about 10 per cent of cardiac output), which commonly develop, although >60 per cent inspired oxygen is more often required for anaesthetised horses, which suffer larger shunt fractions.

It has also been documented that high arterial blood PaO2 (>300 mmHg) results in cerebral vasoconstriction, thus reducing cerebral blood flow. Very high cerebral tissue PaO2 (such as is achievable under hyperbaric conditions) can result in seizures, possibly via oxidative damage of neuronal membranes. Under normobaric conditions, however, most tissues are protected from the dire consequences of ITPV on intracranial pressure – at least in the short term.

Intracranial pressure alterations

IPPV tends to increase intracranial pressure. However, mild to moderate hyperventilation (to produce hypocapnia) reduces intracranial pressure consequent to cerebral vasoconstriction in the short term (a few hours). Mild to moderate hyperventilation can be used as a strategy along with head elevation in an attempt to offset the direct consequences of IPPV on intracranial pressure – at least in the short term.

STARTING AND STOPPING IPPV

Animals do not need to be ‘paralysed’ (ie, by administering a neuromuscular blocker) to ventilate their lungs. The apnoeic threshold is the PaCO2 in arterial blood at which ventilatory efforts cease, and its distance from the normal arterial PaCO2 is relatively constant (around 5 to 9 mmHg), seemingly irrespective of species. Therefore, if mild hyperventilation is performed, and a relative hypocapnia is established, IPPV usually becomes possible without the animal fighting the ventilator because the chemoreceptors are less likely to initiate ventilation at a lower PaCO2. Also, under anaesthesia, especially with volatile agents, chemoreceptors have reduced sensitivity to the blood gases, thus further reducing the patient’s spontaneous ventilatory efforts. The ventilatory response to oxygen (driven by peripheral chemoreceptors only) is all but abolished by volatile anaesthetic agents, whereas the ventilatory response to carbon dioxide (driven by central and peripheral chemoreceptors) is dampened. Despite ventilation being easy to institute in most animals, some patients may continue to ‘fight’ the ventilator. If such cases are then to undergo a thoracotomy, where control of lung inflation becomes of paramount importance (especially when the surgeon cuts into the chest, when inflated
lungs are prone to damage), neuromuscular blockade is indicated.

Reversal of any neuromuscular blocking agents must be ensured to wean an anaesthetised patient off IPPV. This is achieved by, for example, reducing the frequency and/or tidal volume of delivered breaths to allow the arterial PaCO₂ to increase, eventually stimulating the chemoreceptors to respond. Alternatively, the sensitivity to patient-triggering may be changed, thus switching from controlled/mandatory breaths to a situation where the patient has to make an inspiratory effort before the ventilator then ‘assists’ the breath. The trigger sensitivity is further altered in a stepwise fashion until the patient needs no assistance to breathe adequately on its own. For horses, mechanical IPPV may be abruptly stopped at the end of surgery without weaning, but must be made available again subsequently (eg, using an oxygen demand valve once the horse is in the recovery box). The adequacy of spontaneous ventilation can be judged by measuring tidal volume (eg, using a Wright’s respirometer) coupled with breathing rate, and comparing post-operative values with those obtained prior to IPPV being initiated and/or by monitoring end tidal carbon dioxide values until normocapnia can be maintained. Once normocapnia is observed, a sigh breath should be given to check that the animal is making adequate tidal breaths because shallow breathing can result in ‘normal’ end tidal carbon dioxide values, but a sigh breath can reveal carbon dioxide retention.

Weaning intensive care patients off ventilators can sometimes be very difficult. Pre-existing diseases and the presenting problem(s) can influence the ease of weaning, as can other factors such as nutritional status, the development of so-called ‘polyneuropathy of critical illness’ and even respiratory muscle myopathy.

**MONITORING IPPV**

The peak inspiratory pressure, rate and tidal volume should be noted. The tidal volume delivered by the ventilator may not correspond exactly to what enters the patient’s lungs, as some volume is lost (eg, when the tubing distends or via small leaks). However, a Wright’s respirometer can be used to measure tidal volume by positioning it as close to the endotracheal tube adapter as possible. Depending on its orientation, it can measure either the inhaled or exhaled tidal volume (the latter tends to be slightly less than the former).

The overall efficacy of ventilation can be measured non-invasively by capnography. In small animals, the end tidal PaCO₂ is normally very similar to the arterial blood PaCO₂ but, if blood gas analysis is available, it is good practice to occasionally verify the capnography values. As mentioned, in horses, large differences can develop between the end tidal and arterial blood PaCO₂. Therefore, arterial blood gas analyses are more regularly performed (about every half hour) to aid monitoring. Normocapnia should be maintained wherever possible (end tidal and/or arterial blood PaCO₂ of 35 to 45 mmHg for dogs and horses, and 32 to 35 mmHg for cats). See Bilbrough (2006) for further information.

Oxygenation status can also be assessed by arterial blood gas analysis. Pulse oximetry is a rather late indicator of trouble, but is nonetheless useful for beat-to-beat analysis.

**TYPES OF VENTILATION**

Ventilators may help a patient’s ventilation to varying degrees depending on requirements. The table below outlines the four main ‘types’ of ventilation, from spontaneous breathing, where the ventilator essentially is inactive, right through to mandatory ventilation where the ventilator provides all of the patient’s ventilatory requirements. A patient capable of spontaneous ventilation may need ventilatory support because it might only be able to provide occasional breaths for itself, and so will require the ventilator to provide the remainder of its ventilatory needs (eg, anaesthetised animal breathing very slowly might need the occasional mandatory bag-squeeze).

The common types of ventilation are:

- **Continuous Mandatory Ventilation (CMV)**. All breaths are provided by the ventilator. However, if the ventilator is insensitive to the patient’s own respiratory efforts, the patient may ‘fight the ventilator’, and if mandatory breaths are superimposed on spontaneous breaths, lung damage may occur;

- **Intermittent Mandatory Ventilation (IMV)**. Mandatory breaths are provided by the ventilator at a set minimum frequency, ensuring adequate ventilation if the patient fails to breathe adequately spontaneously. If the patient’s spontaneous ventilation rate is faster than the set minimum rate, the majority of the patient’s breaths are spontaneous, with the few mandatory ones as ‘extras’ in between. If the patient initiates a breath at the same time as the ventilator also starts an inspiration, the breath becomes ‘assisted’;

- **Synchronised Intermittent Mandatory Ventilation (SIMV)**. This aims to synchronise the delivery of a minimum rate of mandatory breaths with patient effort, so these breaths are in effect assisted. If the patient fails to trigger a breath, the ventilator delivers a mandatory breath. The patient can also breathe spontaneously between these assisted and mandatory breaths.

<table>
<thead>
<tr>
<th>Types of Ventilation</th>
<th>Definition of breath type</th>
</tr>
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<tbody>
<tr>
<td>Patient Patient Patient</td>
<td>Spontaneous</td>
</tr>
<tr>
<td>Patient Ventilator Ventilator</td>
<td>Assisted</td>
</tr>
<tr>
<td>Patient Ventilator Ventilator</td>
<td>Supported</td>
</tr>
<tr>
<td>Ventilator Ventilator</td>
<td>Mandatory (controlled)</td>
</tr>
</tbody>
</table>

- **MONITORING IPPV**

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Oxygenation status can also be assessed by arterial blood gas analysis. Pulse oximetry is a rather late indicator of trouble, but is nonetheless useful for beat-to-beat analysis.
TECHNIQUES TO AID VENTILATION/OXYGENATION

- **Pressure Support Ventilation (PSV).** Patient-triggered breaths during spontaneous breathing, including those spontaneous breaths during IMV/SIMV, can be ‘supported’ in order to reduce the work of breathing. The work of breathing may be high in patients with weak/fatigued respiratory muscles or those with artificial airways, especially if the endotracheal or tracheotomy tube is relatively narrow, thus offering a relatively high resistance. PSV involves the maintenance of a predetermined airway pressure during inspiration by gas flow from the ventilator, and is pressure-limited and patient- (ie, flow-) cycled.

Other techniques to aid both ventilation and oxygenation involve the inclusion of some resistance to exhalation in the hope of preventing atelectasis and reducing atelectotrauma and biotrauma, while aiming to maintain FRC and adequate gaseous exchange. These include:

- **Extrinsic PEEP.** This can be applied when mandatory ventilation is being provided to a patient. The increased resistance to exhalation results in the maintenance of positive pressure within the airways at the end of exhalation. Intrinsic PEEP can develop following gas-trapping if insufficient time is allowed for exhalation (see box below); here, again, positive pressure exists (and even increases) within the airways during the end expiratory pause. PEEP can help to offset the development of ventilator-induced lung injury (see page 188), but

### Breath waveforms

The shape of the inspiratory flow (related to volume) and pressure waveforms are often described, especially in the intensive care setting. Delivered breaths tend to be either volume- (flow-) controlled (and often volume-limited too), or pressure-controlled (and pressure-limited). Typical waveforms for the respective respiratory cycles are shown below. With all modes of IPPV, it is important to allow sufficient time for passive exhalation, which is an exponential process, to safeguard against ‘gas-trapping’ (sometimes referred to as ‘breath-stacking’), which can result in the development of intrinsic PEEP.

**Typical waveforms**

**Flow**

*Volume- (flow-) controlled* | *Pressure-controlled*
---|---

**Volume**

*Ascending (accelerating) ramp* | *Sinusoidal* | *Rectangular (constant)* | *Descending (decelerating) ramp"

**Pressure**

*Ascending exponential wave* | *Rectangular (square wave, representing constant pressure*

### Volume-controlled ventilation

Volume-controlled ventilation ensures that a constant tidal volume is delivered with each breath, regardless of the compliance and resistance of the patient’s respiratory system. High peak inspiratory and mean intrathoracic pressures may be attained if the respiratory system compliance is low and/or the resistance is high, and thus may risk adverse cardiovascular effects and potential lung damage.

### Pressure-controlled ventilation

The peak inspiratory pressure is chosen to be constant, which should help to avoid baro/volutrauma. In this mode, the tidal volume varies if the respiratory system compliance or resistance change, so potentially a large tidal volume can be delivered if compliance increases (eg, following relief of intra-abdominal pressure and diaphragmatic splinting in a colic surgery); alternatively, a smaller tidal volume may be delivered if the compliance fails. Small leaks and the ‘compression volume’ (ie, volume lost during positive pressure ventilation due to compression of gas within the system) are compensated for in this mode as well.
the application of PEEP increases the mean intrathoracic pressure and may also necessitate the use of higher peak inspiratory pressures, both of which can have detrimental cardiovascular side effects (see earlier);
- CPAP. This is applied throughout the entire breathing cycle and achieves the same goals as PEEP and has similar cardiovascular side effects, but is usually applied when the patient is breathing spontaneously. The pressure tends to stay at a fairly constant baseline, with a slightly more positive pressure during exhalation than inhalation;
- **Bi-level positive airway pressure (Bi-PAP).** Also known as bi-level pressure support, this is used in spontaneously breathing humans – for example, to treat obstructive sleep apnoea. It is similar to CPAP, except the inspiratory positive airway pressure is usually higher than the expiratory positive airway pressure. The use of Bi-PAP has not yet been described in veterinary medicine.

**VENTILATOR DESIGN**

When deciding how best to ventilate a patient’s lungs, it is important to know about the features and limitations of the available ventilator.

**Ventilators for intensive care**

Ventilators intended for use in an intensive care setting tend to allow greater flexibility in approach, but are often unsuited to the delivery of volatile anaesthetic agents. These intensive care type ventilators often have their own hoses, humidification devices, filters, water traps, and so on. Desirable features for ventilators in the intensive care setting include:
- The ability to ventilate a wide range of patient sizes;
- Easy to use, intuitive operator controls;
- The ability to ‘assist’ or ‘control’ ventilation;
- The ability to provide volume- or pressure-controlled ventilation;
- The ability to provide PEEP and CPAP;
- The ability to provide SIMV and PSV;
- The ability to provide varying inspired oxygen concentrations;
- The ability to provide humidified gases without changing the ventilator’s characteristics;
- The ability to minimise the risks of respiratory tract infection and cross-infection;
- Plenty of safety features (eg, high pressure/gas supply failure/power failure alarms, and so on).

**Ventilators for anaesthesia**

Conversely, ventilators designed for anaesthesia are often not readily adaptable for long-term use in an intensive care setting. They are often designed to attach to the bag port of most anaesthetic breathing systems.

Suitable anaesthetic breathing systems include the Jackson Rees modified Ayre’s T-piece and the Bain (non-rebreathing) systems and the circle (rebreathing) system. Magill and Lack systems are less efficient non-rebreathing systems during IPPV than during spontaneous ventilation, and their use for IPPV runs the risk of causing the patient to rebreathe carbon dioxide. Although this can be minimised by doubling the fresh gas flow chosen for spontaneous breathing and ensuring a long end expiratory pause for the system to be purged of carbon dioxide, these systems are generally not recommended for prolonged IPPV.

The T-piece and Bain, however, are more efficient during IPPV than spontaneous ventilation, and the fresh gas flow can be halved from that required for normal spontaneous breathing without risk to the patient, providing:
- There is a relatively long end expiratory pause and the ventilation frequency is not too high so that carbon dioxide can be purged from the system in between breaths by the fresh gas flow; or
- The patient’s lungs are purposely hyperventilated, but permissive rebreathing is allowed so that the animal does not become hypocapnic. Capnography and/or blood gas monitoring becomes almost essential in this situation.

When ventilators were first used, it was often difficult not to hyperventilate patients, which can still be a problem with smaller patients. Many anaesthetic machines have a yoke for a carbon dioxide cylinder. One of the reasons for this was so that the anaesthetist would be able to include 4 per cent carbon dioxide in the inspired gas mixture in order to offset the hypocapnia caused by hyperventilation (which is inevitable during IPPV).

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**CORRECTIONS AND CLARIFICATIONS**

**Avoidance of medicines residues in milk: an update**

*In Practice*, March 2007, pp 147-150

In the section on ‘Antibiotic screening tests’, on page 149, it was incorrectly stated that all the tests for detecting residues in milk are intended for use on bulk tank milk samples only. The exception to this is the Delvo SP NT test, which has been developed for use on individual cows or bulk milk. Many farmers still continue to use the Delvo SP test on-farm as they may not be aware of the availability of the NT test. The majority of milk buyers have switched over to the Delvo SP NT test for bulk milk sampling. The error is regretted.

**Practical guide to fluid therapy in neonatal foals**

*In Practice*, March 2007, pp 130-137

The picture caption on page 136 should have read ‘(above) Blood can be allowed to settle to separate plasma and red blood cells. (right) Plasma can then be decanted into a separate bag ready for use. (far right) Ideally, commercially available plasma should be used’, and not as stated. The error is regretted.
Practical guide to fluid therapy in neonatal foals

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