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RESPIRATORY AND CARDIOVASCULAR SUPPORT

Anthea Raisis & Gabrielle Musk

INTRODUCTION

Maintenance of normal oxygenation, ventilation and perfusion (the ABC of resuscitation) is essential in the neurological patient to prevent secondary neurological injury or exacerbation of the underlying condition. In addition, correction of hypoxaemia, hypercapnia and poor perfusion are the most important strategies for reducing intracranial pressure (ICP). The type and extent of supportive care required will depend on the cause of respiratory and/or cardiovascular impairment and the severity of disruption to normal oxygenation, ventilation and perfusion. Techniques for maintaining normal respiratory and cardiovascular function and, therefore, adequate oxygen delivery to the tissues are outlined in this chapter.

PROVIDING AN ARTIFICIAL AIRWAY

The indications for providing an artificial airway are listed below:

- Laryngeal paresis or paralysis, which can be caused by cranial nerve deficits associated with disorders of the brainstem or generalized neuromuscular disease.
- Laryngeal spasm (e.g. tetanus).
- An inability to protect the airway adequately (e.g. severe depression/recumbency associated with intracranial disease).
- Mechanical ventilation.

Treatment methods for acquiring and maintaining a patent airway

Oral endotracheal intubation

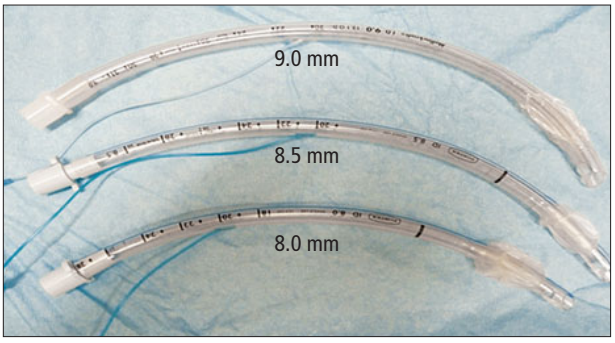
Specific indications

- Emergency management of upper airway obstruction (e.g. laryngeal spasm, laryngeal paralysis).
- Protection of airway in an obtunded/unconscious animal.
- Animals that need mechanical ventilatory support.

Precautions

Oral endotracheal intubation in most animals will require sedation or anaesthesia. Details of how to sedate/anaesthetize a neurological patient safely are descibed in Chapter 29.

Selection of an appropriately sized endotracheal tube (ETT) is essential (**19**). Resistance to breathing is markedly increased as the diameter of the ETT decreases. If the diameter of the tube is halved, resistance increases 16-fold. To minimize resistance to breathing, the largest diameter tube that can be easily and safely passed should be used. If the ETT is too long, resistance to expiration will also increase (doubling the length will double the resistance to flow) and the risk of excessive



▶ **19 Selection of an appropriate endotracheal tube for each animal is required to ensure that resistance to breathing is not excessive. When preparing for intubation, select the most appropriate endotracheal tube size in addition to tubes 0.5–1.0 mm above and below that size as shown here.**

equipment dead space or inadvertent endobronchial intubation increases (**20**).

The increased work of breathing associated with increased resistance can be overcome by mechanical ventilation. The potential for this increased work to reduce the adequacy of ventilation and cause hypercapnia will be greatest in any animal expected to breathe spontaneously or when attempting to wean an animal off the ventilator. An excessively long ETT will also increase the apparatus dead space, leading to rebreathing of expired carbon dioxide (CO_2). This also increases the risk of hypercapnia and increased ICP in the neurological patient.

As oral intubation bypasses the nasal cavity, which normally humidifies inspired air, the airways are predisposed to desiccation. Humidification of gases is essential to minimize drying of lower airways and should always be used in patients that are ventilated for anything other than a short period of time.

Inflation of the ETT cuff is required to prevent aspiration of saliva or gastric contents. The cuff should be inflated just enough to create a seal. If excessive or prolonged inflation pressure is created, the risk of pressure necrosis of the tracheal mucosa increases. The pressure of the small arterioles in the tracheal mucosa is very low, so the pressure within the cuff should not exceed 25 mmHg. Careful inflation of the cuff is required to minimize damage to the mucosa. During prolonged intubation, occasional repositioning of the endotracheal tube may help reduce mucosal ischaemia; however, repositioning the ETT increases the risk of aspiration of secretions that have accumulated above the cuff. If the tube is repositioned, the pharynx and oesophagus should be suctioned prior to cuff deflation to reduce the risk of secretions entering the airway.

Tracheostomy

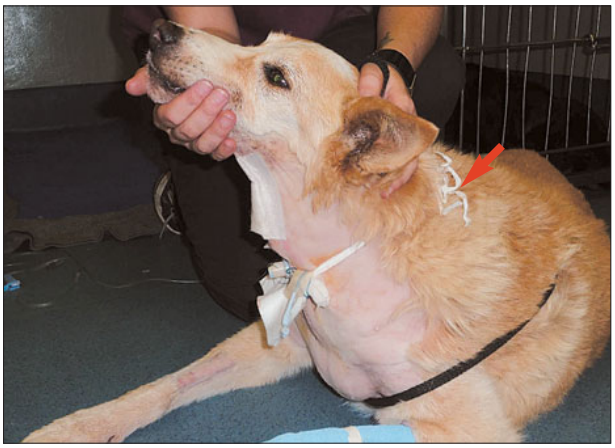
Specific indications

- Chronic management of airway dysfunction in conscious patients (e.g. animals with tetanus, laryngeal paresis).
- To reduce the amount of sedation/anaesthesia required to immobilize animals requiring mechanical ventilation.
- Severe laryngeal trauma.

Precautions

The tracheostomy tube must be secured adequately in place to prevent accidental removal and loss of the airway. It is essential that the tube can still be removed rapidly should obstruction occur. Tying the tube in place is preferred over suturing (**21**).

▲ **20** **Endotracheal tubes and other apparatus (such as the capnograph sampling connector; arrow) extending beyond the level of the animal's incisors may increase apparatus dead space, resistance to expiration and the work of breathing. Always pre-measure the endotracheal tube before placement.**



▲ **21** **A tracheostomy tube is used to provide a patent airway in animals with severe compromise of the upper respiratory tract. The tube should be tied in place (arrow) to allow rapid removal should obstruction of the tube occur.**

The accumulation of secretions may occlude the tube. When mechanical ventilation is not required and the risk of aspiration is considered minimal, it is recommended that an uncuffed tube is used (**22**). It is also recommended that the diameter of the tube is one half the diameter of the trachea, so the animal can breathe around the tube for short periods should the tube become occluded.

Care of the tracheostomy site is labour intensive. Animals need constant monitoring, as occlusion of the tube can result in death within minutes. Regular suctioning is required to prevent accumulation of secretions. The tube should be replaced 1–2 times a day.

Intratracheal catheter

If the upper airway is completely obstructed, an intratracheal catheter or needle (large dog: 16 g; small to medium dog: 18 g) can be placed (**23**). Oxygen can be insufflated via these catheters/needles for <5 minutes.

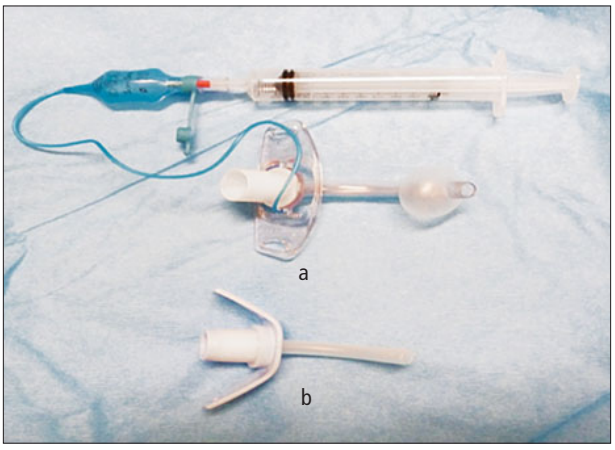
Specific indications

- Obstructed airway and impending respiratory arrest.
- Short-term oxygenation while oral intubation or tracheostomy is performed.

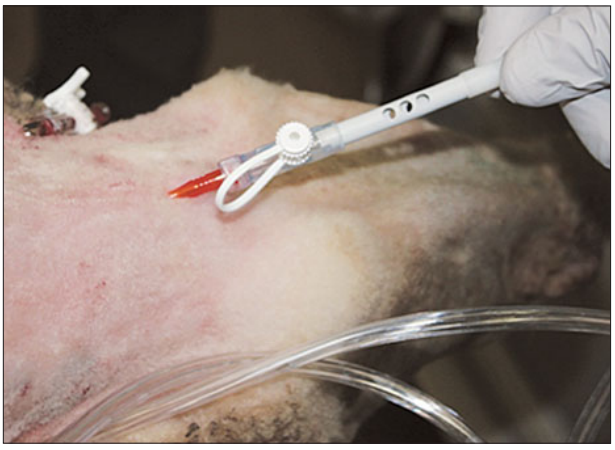
Precautions

This method of oxygenation is for short-term use only (<5 minutes) and must be used with conservative oxygen flow rates. When the airway is completely obstructed, oxygen insufflated into the lung via a narrow needle or catheter cannot be expired, therefore there is a risk of rupturing the lung, resulting in a pneumothorax. To determine suitable flow rates, the volume of the animal's lungs must be considered. For example: a 20 kg dog would have an approximate tidal volume of 200 ml (based on 10 ml/kg). Therefore, an oxygen flow of 1 litre $minute^{-1}$ would fill the tidal volume of the lungs in 12 seconds, while an oxygen flow rate of 200 ml/minute would take 1 minute.

This method does not allow for mechanical ventilation of the animal, and hypercapnia will occur. Intubation should be performed promptly and mechanical ventilation instigated as soon as a patent airway is available.



▲ 22 **A cuffed tracheostomy tube (a) is required when mechanical ventilation is being performed. However, when the animal is conscious and spontaneously breathing, an uncuffed tracheostomy tube (b) is preferred.**



▲ 23 **An intratracheal catheter, as shown here, can be used to provide short-term (<5 minutes) oxygen supplementation in animals that are completely obstructed.**

BREATHING

Adequate oxygenation (arterial oxygen partial pressure [PaO_2] ≥80 mmHg [10.7 kPa]; saturation of haemoglobin with oxygen [SpO_2] ≥95%) and ventilation (arterial carbon dioxide partial pressure [$PaCO_2$] 35–40 mmHg [4.7–5.3 kPa]; end-tidal carbon dioxide partial pressure [$P'ETCO_2$] 30–35 mmHg [4–4.7 kPa]) are required to maintain cerebral oxygen delivery and prevent increases in ICP.

Oxygen supplementation

Indications

Oxygen supplementation is indicated if there is a critical decrease in oxygen delivery to the brain. Oxygen delivery (DO_2) is a product of cardiac output (CO) and oxygen carrying capacity (CaO_2) measured as the oxygen content of arterial blood:

$$DO_2 = CO \times CaO_2$$

The oxygen content of arterial blood is a combination of oxygen bound to haemoglobin (haemoglobin × SpO_2 × 1.34) and that dissolved in the plasma (0.003 × PaO_2). As the majority of oxygen is carried in the blood bound to haemoglobin, decreases in oxyhaemoglobin saturation and haemoglobin concentration have the greatest influence on oxygen delivery to tissues, including the brain. Decreases in oxygen saturation of haemoglobin may occur due to decreased inspired oxygen percentage, decreased ventilation or decreased transfer of oxygen across the alveoli, or ventilation/perfusion mismatch or right-to-left shunting of pulmonary blood flow. A decrease in haemoglobin concentration (anaemia) will occur due to blood loss or red cell destruction.

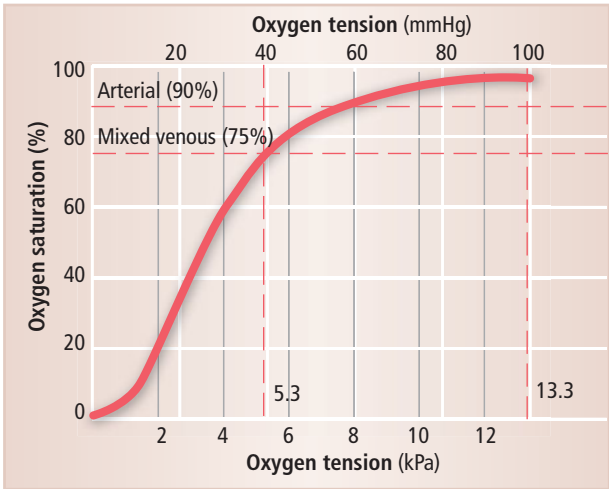
Hypoxaemia

Definition

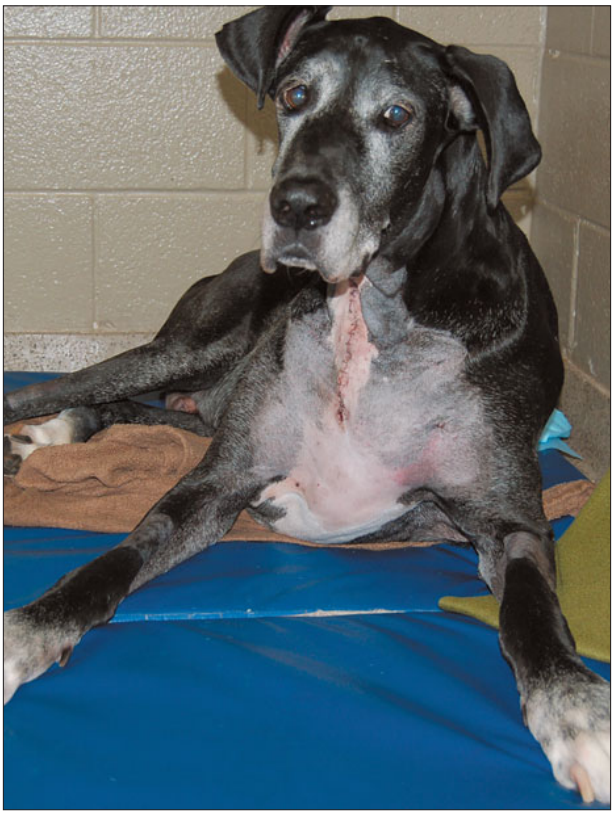
Although only a small amount of oxygen is carried dissolved in blood, assessment of oxygenation is frequently performed by measurement of PaO_2. Severe hypoxaemia is defined as a PaO_2 of <60 mmHg (8 kPa). This is equivalent to an SpO_2 of 90% (**24**). SpO_2 is measured using pulse oximetry (see below). For animals with intracranial disease, it is recommended that the PaO_2 is maintained at >80 mmHg (10.7 kPa) to prevent increases in ICP.

Common causes of hypoxaemia in animals with neurological disease

- Aspiration pneumonia. Animals with CN deficits associated with brainstem or neuromuscular disease are predisposed to regurgitation and aspiration due to impairment of upper respiratory tract (URT) function. Recumbency and severe depression also predispose the animal to aspiration.
- Atelectasis (**25**). Recumbency, immobility and a high inspired oxygen concentration all predispose to alveolar collapse. This creates a mismatch in ventilation and perfusion and results in shunting of blood through the lungs. The shunt prevents oxygenation of the blood as it bypasses ventilated alveoli.
- Acute lung injury (ALI). Lung injury secondary to systemic inflammation, prolonged exposure to high inspired oxygen concentrations and trauma due to high ventilation pressures all contribute to the incidence and severity of ALI. These processes cause the release of inflammatory mediators within the lung, which stimulate a vicious cycle of pathological changes within the alveoli and surrounding lung tissues. The net result is interference with pulmonary gas exchange. ALI is tentatively diagnosed when the ratio of arterial oxygen tension

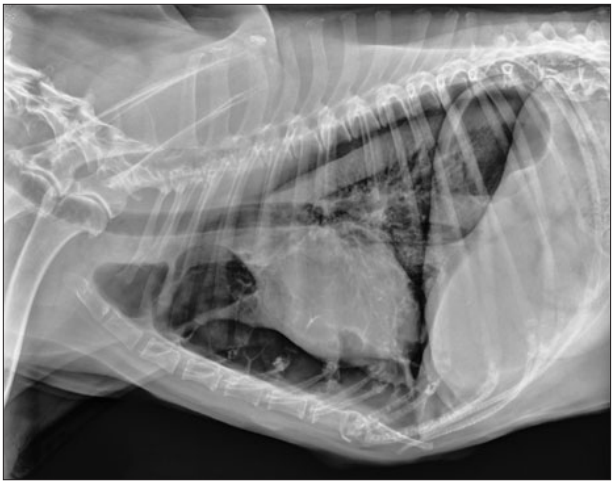


▲ **24 Oxygen–haemoglobin dissociation curve.**



◀ **25 Recumbency and immobility are very common causes of atelectasis and hypoxaemia in the neurological patient.**

▼ **26 Pneumothorax secondary to chest trauma, as shown in this lateral radiograph, can contribute to hypoxaemia in animals with concurrent head trauma. (Photo courtesy Shannon Holmes)**



to fractional inspired oxygen concentration (PaO_2:FiO_2) is <300. Supportive care of ALI is based on supplemental oxygenation via mask, nasal catheters or oxygen cage.

- Acute respiratory distress syndrome (ARDS). ARDS occurs as the pathological changes in ALI progress and cause greater interference with gas exchange. ARDS is defined as a PaO_2:FiO_2 ratio of <200. These patients require ventilatory support.
- Pulmonary contusions or pneumothorax (**26**) in animals with concurrent chest trauma.
- Neurogenic or non-cardiogenic pulmonary oedema occurs in response to increased sympathetic nervous system stimulation and blood pressure (BP) secondary to brainstem ischaemia or compression. URT obstruction may also cause pulmonary oedema if the negative pressures generated during inspiration damage the alveolar membrane, causing fluid to flood the alveoli.
- Hypoventilation. In an animal breathing room air, hypoventilation causes increased alveolar CO_2, which dilutes alveolar oxygen and results in less oxygen being available to diffuse into the arterial blood. Restoring normal CO_2 values by the use of ventilation should also correct the hypoxaemia. If the hypoxaemia is not corrected with ventilation and the return of CO_2 to normal, then other causes of hypoxaemia need to be investigated.

Management

Hypoxaemia is managed by treating the underlying cause when possible and providing supplemental oxygen (see below) until the cause of the hypoxaemia has been corrected.

Anaemia

The clinical significance of anaemia varies according to whether the anaemia is acute or chronic:

- Acute anaemia (e.g. haemorrhage): clinical signs will usually occur if the PCV (packed cell volume) is <0.30 l/l (30%) in dogs and <0.25 l/l (25%) in cats.
- Chronic anaemia (e.g. haemolytic anaemia): clinical signs will generally be apparent when the PCV is <0.20 l/l (20%) in dogs and <0.15 l/l (15%) in cats.

Causes of anaemia in neurological disease

The most common cause is haemorrhage secondary to trauma or surgical blood loss.

Management

The definitive treatment for reduced tissue oxygenation due to anaemia is whole blood or red blood cell (RBC) transfusion. For further information on blood transfusion see Chapter 31.

If there is a delay in administering blood or RBCs, oxygen supplementation can be useful. While oxygen supplementation in these cases may only provide extremely small amounts of dissolved oxygen in the blood, this can be life saving in the short term.

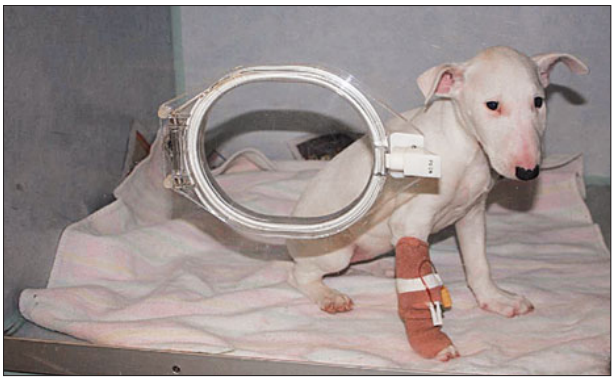
Treatment methods for supplementing oxygen

Increasing the FiO_2

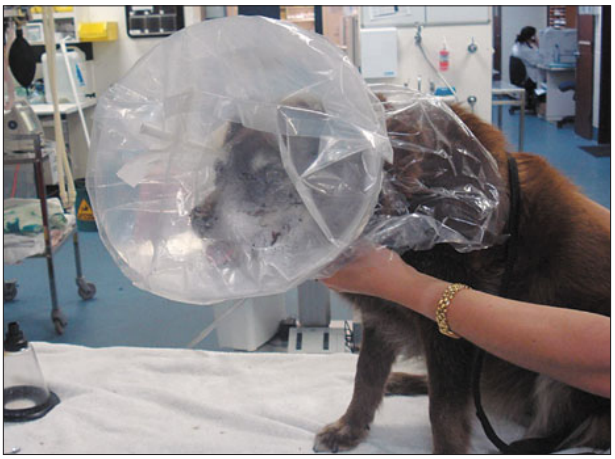
The aim of techniques that increase the inspired percentage of oxygen is to use the lowest FiO_2 that will maintain the PaO_2 at between 80 and 100 mmHg (10.7–13.3 kPa). As haemoglobin is fully saturated when the PaO_2 is approximately 100 mmHg (13.3 kPa), further increases in arterial oxygen tension produce very little increase in the amount of oxygen carried by the blood. In addition, there is an increased risk of lung damage as exposure to higher than normal levels of alveolar oxygen triggers pathological changes within the pulmonary tissues (oxygen toxicity). The higher the alveolar oxygen tension and the longer the duration of treatment, the greater the risk of irreversible alveolar damage.

Increased inspired oxygen can be delivered by a number of methods, including oxygen cage, head collar, mask or nasal catheter.

- **Oxygen cage.** Cages are useful for initial stabilization of small dogs and cats in respiratory distress while minimizing the stress of handling (**27**). The advantage of using an oxygen cage is that an FiO_2 of >50% can be achieved when the cage remains closed. The disadvantages include limited access to the patient if continuous oxygen support is required. In addition, patients are also at risk of hyperthermia and should be monitored carefully.
- **Head collar.** Head collars can be used for larger animals where size prevents the use of oxygen cages. The use of head collars covered with plastic film can provide a personalized oxygen cage. Oxygen is delivered via tubing attached on the inside of the head collar (**28**). Head collars have the advantage of providing a high FiO_2 while still allowing access to the rest of the animal. The disadvantages include:
  - The risk of jugular occlusion and increased ICP if the collar is too tight.
  - Hypercapnia can occur if the collar is tight, the oxygen flow is too low or the supply tubing becomes disconnected, allowing CO_2 to build up within the enclosed head collar. Hypoxaemia will also develop if the oxygen tubing detaches. Flow rates equivalent to those used in non-rebreathing anaesthetic systems (200–300 ml/kg/minute) are probably adequate.
  - Hyperthermia can also occur with this technique as the animal is rebreathing expired warm and humidified air.



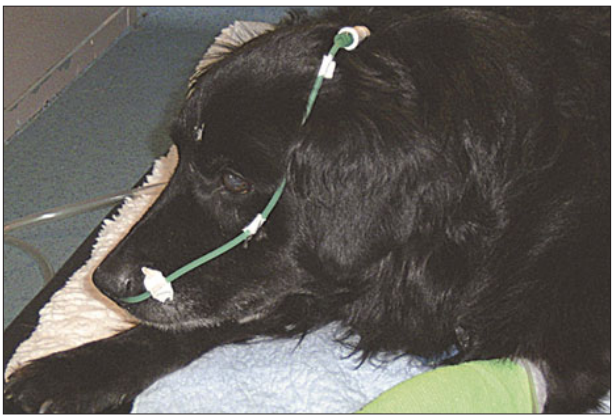
▶ **27 An oxygen cage can be used to provide oxygen supplementation in small- to medium-sized animals.**



▲ 28 **A head collar covered in plastic film can be used to provide oxygen supplementation to larger animals when other methods are not feasible. Care should be taken to avoid jugular vein compression and overheating.**

▲ 29 **Face masks can be used to provide oxygen during initial stabilization or prior to induction of anaesthesia.**

▲ 30 **'Flow-by' oxygen therapy can be used to provide short-term oxygen supplementation to animals that will not tolerate a face mask.**

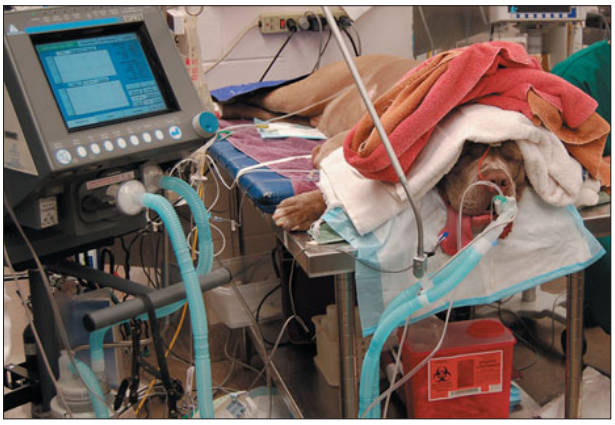


▲ 31 **Nasal catheters can be used to provide continuous oxygen supplementation and also allow continuous access. They should be avoided in animals with increased intracranial pressure, coagulopathy and nasal fractures.**

- **Face mask.** Face masks are generally used for initial stabilization only, particularly in animals not amenable to oxygen cages due to their size (**29**). The advantage of using face masks for oxygen supplementation is that they are easily accessible and allow for prompt administration of oxygen in an emergency setting. One of the disadvantages is that animals in respiratory distress may not tolerate a mask held over their face. In these cases 'flow-by' oxygenation can be used (**30**). A tight-fitting mask can also exacerbate hyperthermia, as rebreathing of expired humidified gases occurs. An open mask or flow-by technique provides limited increase in FiO_2.
- **Nasal catheter**. An intranasal catheter is inserted into the ventral nasal meatus, directing the catheter ventromedially (**31**). The tip can be positioned just inside the nostril or in the nasopharynx.

To position the catheter tip in the nasopharynx, the catheter should be pre-measured to the level of the medial canthus to ensure correct positioning. If the tip is inserted too far, it risks being introduced into the oesophagus, causing aerophagia and gastric dilation. Most animals will tolerate an oxygen flow of 100 ml/kg/minute/nostril. Higher flows may cause increased irritation, sneezing or removal of the catheter by the patient. If flows >100 ml/kg/minute are required to achieve adequate oxygenation, a catheter can be placed in each nostril, allowing a total oxygen flow of up to 200 ml/kg/minute to be delivered comfortably. Nasal catheters have the advantage of allowing long-term supplementation of oxygen in animals with respiratory disease and continuous access to the patient. Furthermore, there is no risk of hyperthermia or hypercapnia. The disadvantages include sneezing associated with placement of nasal catheters, which can be detrimental if there is: (1) increased ICP, as sneezing will cause further increases, which may result in fatal herniation of the brainstem, or (2) coagulopathy, as sneezing may cause bleeding from the nostril or haemorrhage into the eye or brain. This procedure should also be avoided in the head-trauma patient, when fractures within the nasal cavity can lead to misplacement of the catheter within neural tissue. Finally, there is a limited increase in FiO_2 of 0.3–0.4 (30–40% inspired oxygen concentration) observed with this method.

▲ **32** **Endotracheal intubation and delivery of high inspired oxygen concentration may be needed in animals that do not improve with other methods of oxygen supplementation.**



▲ **33** **Mechanical ventilation is commonly used to 'breathe' for patients that have inadequate spontaneous ventilation or are unresponsive to other methods of oxygen supplementation.**

Endotracheal intubation

Endotracheal intubation and connection to a breathing system may be necessary when other methods of increasing FiO_2 fail to achieve adequate arterial oxygen tension or if the animal becomes increasingly depressed (**32**). Intubation must be performed before the animal becomes unconscious. This will require careful administration of intravenous anaesthetic agents. If intubation is delayed until the animal becomes unconscious because of the pathological process, cardiopulmonary arrest is highly likely.

Endotracheal intubation has the advantage of allowing connection to a breathing system and the delivery of 100% oxygen. The clinician can therefore be more confident of the delivered FiO_2. Disadvantages include: (1) maintenance of oral endotracheal tubes generally requires some degree of sedation or anaesthesia and (2) the depressant effects of sedative and anaesthetic agents will also necessitate some form of positive pressure ventilation, particularly in animals with intracranial disease where maintenance of normal $PaCO_2$ is essential.

Assisted ventilation is indicated when increasing FiO_2 fails to correct hypoxaemia (**33**). Details of ventilation techniques are described in the next section.

VENTILATION

The aim of mechanical ventilation is to recruit and stabilize alveoli and deliver oxygen to and remove carbon dioxide from them without causing lung injury. Careful monitoring of ventilation to ensure normocapnia and satisfactory oxygenation is essential to guide management strategies. Furthermore, the haemodynamic consequences of mechanical ventilation should be continuously monitored. During spontaneous ventilation, a negative pressure is created within the thoracic cavity to draw air into the lungs. During mechanical ventilation, a positive pressure is generated to push gas into the lungs. This positive pressure compresses blood vessels within the chest, especially in the low-pressure venous circulation and right side of the heart. The net effect is a decrease in cardiac output. Ideally, cardiac output should be monitored in ventilated animals, but arterial BP measurement is a common surrogate measure in a clinical setting.

Mechanical ventilation is indicated in animals with respiratory failure. Respiratory failure can be divided into two types:

- **Hypercapnic ventilatory failure** (inadequate alveolar ventilation = hypoventilation): generally considered to be present when the $PaCO_2$ is >50 mmHg (6.7 kPa). Ventilatory failure should also be considered in animals with normal $PaCO_2$ if there is: (1) clinical evidence of increased respiratory work and impending fatigue (high respiratory rate; change in respiratory pattern; increased inspiratory effort; erratic changes in tidal volume and/or respiratory frequency), or (2) inadequate respiratory compensation for metabolic acidosis. In animals with intracranial disease, any increase in CO_2 may have detrimental effects on ICP. Mechanical ventilation is required in any animal with intracranial disease if the $PaCO_2$ is >40 mmHg (5.3 kPa). The causes of ventilatory failure in animals with neurological disease include:
  - Neuromuscular weakness: tetrodotoxin, snake envenomation, tick paralysis, botulism, polyradiculoneuritis, myasthenia gravis (MG).
  - Cervical spinal cord injury.
  - Intracranial disease with involvement of respiratory centres (i.e. caudal fossa pathology).
  - Drug-induced CNS depression: anaesthesia, opioid overdose.
- **Hypoxaemic respiratory failure** (inadequate oxygen exchange): generally defined as arterial oxygen tension <60 mmHg (8 kPa) when the FiO_2 is >0.5.

Treatment methods for providing ventilation

The most common type of ventilation used routinely in animals is intermittent positive pressure ventilation (IPPV). The factor determining the termination of inspiration and the onset of expiration is most often volume, pressure or time. Positive end-expiratory pressure (PEEP) and continuous positive airway pressure (CPAP) are also used, particularly in animals with pulmonary pathology. *Table 6* gives a summary of the modes of ventilation that can be used in neurological patients.

Table 6 **Modes of mechanical ventilation**

MODE	MECHANISM	USES/ADVANTAGES	DISADVANTAGES/CAUTIONS
Volume-cycled IPPV	Inspiration terminated when pre-set volume delivered. Delivers desired V_T regardless of lung compliance	Preferred use in normal lungs	Increases risk of lung injury in animals with altered lung compliance
Pressure-cycled IPPV	Inspiration is terminated when a pre-set pressure is delivered. V_T will therefore be determined by lung and chest wall compliance	Preferred method for animals with altered lung compliance or very small patients	Poor airway, lung or chest wall compliance will require high inflation pressures to achieve an adequate minute volume

(Continued)

Table 6 **Modes of mechanical ventilation** (*continued*)

MODE	MECHANISM	USES/ADVANTAGES	DISADVANTAGES/CAUTIONS
Time-cycled IPPV	Inspiration is terminated when a set inspiration time has passed. V_T is determined by lung and chest wall compliance	Infrequently used in current clinical practice	PIP is determined by lung and chest wall compliance. Decreases in compliance will cause increases in PIP and increase risk of injury
OTHER DEFINITIONS			
PEEP	Maintains positive pressure at the end of expiration during controlled ventilation, preventing complete alveolar collapse at the part of the respiratory cycle when airway pressure is usually 0 cmH_2O	Minimizes atelectasis and associated ventilation/perfusion mismatch in normal lungs. Minimizes volutrauma in lung pathology	High PEEP may compromise venous return and cardiac output. Decreased venous return can lead to increased cerebral blood volume and increased ICP
CPAP	Technique used in spontaneously breathing animals to prevent alveolar collapse at the end of expiration and increase functional residual capacity	Can help stabilize open alveoli and prevent alveolar collapse. May improve oxygenation in spontaneously breathing animals and negate need for IPPV	Animals may not tolerate nasal catheters or prongs. Higher than normal pressure at the end of expiration may compromise venous return and cardiac output

V_T = tidal volume; PEEP = positive end-expiratory pressure; CPAP = continuous positive airway pressure; ICP = intracranial pressure; PIP = peak inspiratory pressure; IPPV = intermittent positive pressure ventilation.

A wide range of other ventilation strategies are used to promote ventilation and oxygenation, minimize lung injury and prevent haemodynamic compromise. Techniques including high-frequency ventilation, inverse ratio ventilation, biphasic airway pressure and extracorporeal membrane oxygenation have been used to reduce airway pressures in human patients with severe pulmonary disease. These techniques are limited to specialist centres with specialist equipment, a thorough understanding of which is essential before embarking on either short- or long-term management. A detailed description of the equipment and techniques is beyond the scope of this book. For more information, the reader is referred to the Further reading list, p. 623.

Adverse effects of ventilation

Mechanical ventilation may have numerous adverse effects on a variety of body systems, including the cardiovascular, respiratory, neurological, renal and gastrointestinal systems. A summary of these effects and how to minimize them is given in *Table* 7. Before ventilation of any animal is undertaken it is essential to understand the potential adverse effects and how to avoid them.

Guidelines for use of mechanical ventilation

The aim of ventilation is to optimize oxygenation and ventilation, while minimizing adverse effects on cardiovascular, pulmonary and neurological functions.

Different ventilatory strategies are used to manage different types of respiratory failure. The ventilation strategy may need to be further altered if the animal has concurrent intracranial disease. Guidelines for use of ventilation in these situations are described below (see also *Table 8*, page 46).

Table 7 **Adverse effects of mechanical ventilation**

SYSTEM	EFFECT	METHODS OF MINIMIZING ADVERSE EFFECTS
Cardiovascular	Positive airway pressure during IPPV interferes with venous return and CO during inspiration. CPAP and PEEP interfere with venous return and CO during expiration. PEEP in combination with IPPV interferes with venous return throughout the respiratory cycle. Decreased CO leads to decreased oxygen delivery to the tissues and may offset improvement in oxygen content	• To minimize the detrimental effects on cardiovascular performance it is important to ensure adequate blood volume and minimize PIP, the amount of time spent in inspiration (inspiratory time) and PEEP. • Monitor blood pressure and CVP during ventilation. • Monitor ICP in animals with intracranial disease.
Pulmonary	Barotrauma: excessive airway pressure causes physical disruption of lung tissue and extra-alveolar air (e.g. pneumothorax). Volutrauma: overdistension of alveoli causes increased permeability, oedema and inflammation. Repetitive stretching and recoil of alveoli induces inflammatory and structural changes, leading to increased permeability of alveoli and oedema formation. Inflammation associated with ventilator-induced lung injury may be an important inciting factor for development of SIRS and MOF	• Use the minimum V_T and PIP necessary to achieve adequate oxygenation and ventilation. • Lung disease: lower V_T, higher RR and PEEP. Slight hypoventilation and permissive hypercapnia ($PaCO_2$ 45–60 mmHg). *Note:* Permissive hypercapnia must be avoided in patients with intracranial disease.
Renal function	Decreased urine output due to release of ADH and decreasing ANP production causing fluid retention. Decreased GFR and intrarenal blood flow redistribution may interfere with the renal elimination of drugs	• Use the minimum V_T and PIP necessary to achieve adequate oxygenation and ventilation.
Gastrointestinal system	Increased incidence of gastric ulceration and liver dysfunction. Decreased portal blood flow may also reduce the elimination of drugs that undergo hepatic metabolism	• Use the minimum V_T and PIP necessary to achieve adequate oxygenation and ventilation.
Central nervous system	Interference with venous return can lead to increased cerebral venous blood volume and ICP	• Use the minimum V_T and PIP necessary to achieve adequate oxygenation and ventilation. • Neuromuscular relaxation in anaesthetized animals can help reduce the PIP required to ventilate to normocapnia. • Use PEEP very carefully in animals with intracranial disease. • Monitor CVP and ICP in animals with intracranial disease.

V_T = tidal volume; RR = respiratory rate; PIP = peak inspiratory pressure; PEEP = positive end-expiratory pressure; CPAP = continuous positive airway pressure; ICP = intracranial pressure; CVP = central venous pressure; CO = cardiac output; GFR = glomerular filtration rate; SIRS = systemic inflammatory response syndrome; MOF = multiple organ failure; ADH = antidiuretic hormone; ANP = atrial natriuretic peptide; IPPV = intermittent positive pressure ventilation.

Table 8 **Guidelines for mechanical ventilation**

	NORMAL LUNGS	ABNORMAL LUNGS	INTRACRANIAL DISEASE
Preferred ventilator type	Pressure or volume cycled	Pressure cycled	Pressure cycled
Settings			
V_T	10–20 ml/kg	<6 ml/kg	Settings are a compromise between pre-existing lung condition and need to maintain normocapnia and prevent increased ICP
RR	Comparable to resting respiratory rate	Adequate to maintain appropriate $F'EtCO_2$	
I:E ratio	1:3	1:3	
PIP	10–20 cmH_2O will adequately ventilate most animals with normal lungs; cats will sometimes require <10 cmH_2O due to higher chest compliance	Higher PIP required due to reduced compliance (see Further reading)	Use the minimal PIP required to achieve normoxia and normocapnia, but limit adverse effects on venous drainage and ICP
PEEP	0–5 cmH_2O	Start at 3 cmH_2O and increase up to 10 cmH_2O as required to achieve adequate oxygenation without decreasing oxygen delivery	Avoid/use carefully in animals with intracranial hypertension

V_T = tidal volume; RR = respiratory rate; I:E ratio = inspiratory:expiratory time ratio; PIP = peak inspiratory pressure; PEEP = positive end-expiratory pressure; ICP = intracranial pressure; $F'ECO_2$. = Fractional concentration of carbon dioxide in mixed expired air

Mechanical ventilation in cases of ventilatory failure

In animals with ventilatory failure, peak inspiratory pressure (PIP) is adjusted until the minimum pressure that maintains adequate ventilation and oxygenation is obtained. For normal chests, a PIP of 8–10 cmH_2O in dogs and slightly less in cats will adequately ventilate most animals in normal body condition. Higher pressures may be required in obese animals and animals with barrel chests.

The majority of these animals will have normal lungs at least initially, so only conservative PEEP may be required (up to 3 cmH_2O). This will help prevent the development and progression of atelectasis, especially if a high FiO_2 is used. PEEP must be used with extreme care in animals with increased ICP.

Mechanical ventilation of animals with lung pathology in the absence of intracranial disease

Smaller tidal volumes (5–6 ml/kg) and higher respiratory rates are recommended in animals with diseased lungs to prevent overdistension of normal lung tissue. Overdistension of normal lungs occurs as gas delivered during the inspiratory phase follows the pathway of least resistance and distributes to areas of lung that are more compliant (i.e. normal lung tissue is preferentially ventilated compared with less compliant diseased tissue). Overdistension of normal lung contributes to ventilator-induced lung injury (VILI) and triggers a cascade of worsening pulmonary function and the development of multiple organ failure. Unfortunately, this type of ventilation strategy is unable to maintain normal CO_2. However, in animals with normal intracranial compliance the increases in PaO_2 are generally tolerated.

PEEP is particularly useful in animals with reduced lung compliance, as there is a tendency for lungs to collapse during expiration. In these conditions, PEEP prevents alveolar collapse and atelectasis. By preventing alveolar collapse, PEEP prevents the cyclic collapse and re-opening of alveoli that contribute to VILI. By minimizing atelectasis, PEEP also reduces physiological dead space and improves pulmonary gas exchange.

Adverse effects of PEEP includes interference with normal venous return during the expiratory phase of ventilation. To minimize this effect, the amount of PEEP

applied is adjusted to optimize arterial oxygenation while maintaining normal cardiac output (and oxygen delivery). Clinically, where measurement of cardiac output is not available, the effect of IPPV on arterial BP and central venous pressure (CVP) can be used to assess the impact on cardiovascular function.

Mechanical ventilation of animals with lung pathology in the presence of intracranial disease

Ventilation of animals with both lung and intracranial disease presents a unique challenge, as many of the ventilator strategies designed to minimize VILI have a detrimental effect on ICP.

The low tidal volume ventilation strategies recommended in animals with lung pathology cause permissive hypercapnia and are harmful in animals with intracranial disease, as a marked increase in ICP accompanies increasing $PaCO_2$. Increasing mean airway pressure also has the potential to increase ICP due to detrimental effects on venous return increasing cerebral blood volume. The level of PIP required to ventilate these animals adequately may be reduced by use of non-depolarizing neuromuscular blockers, which increase compliance of the chest wall. Venous return is optimized by increasing expiratory time and, thus, the time for blood to return to the heart. PEEP should be avoided or used extremely carefully in animals with intracranial disease. (For details of neuromuscular blockers see Chapter 29.)

Increases in ICP negate any improvement in oxygenation by reducing cerebral perfusion pressure (CPP). Furthermore, increased ICP increases the risk of herniation and death. The use of ICP monitoring may be worthwhile in animals with concurrent intracranial disease and lung disease warranting mechanical ventilation to ensure ventilation strategies do not exacerbate intracranial hypertension. (For more details on ventilation in ARDS see Further reading.)

Ventilation of animals with pulmonary disease and concurrent CNS disease requires intensive monitoring of pulmonary, cardiovascular and neurological functions. This is limited to referral hospitals.

CARE OF THE VENTILATED ANIMAL

Appropriate monitoring and supportive care of the ventilated patient are required to minimize adverse effects of chronic intubation, ventilation and recumbency. This section discusses some important aspects of this care. *Table 9* gives a summary of the general nursing requirements of these animals.

Table 9 **General nursing of the ventilated patient**

PROCEDURE	RATIONALE
Lubricate eyes	Paralysed animals are unable to close eyelids and are predisposed to corneal ulcers if eyes are allowed to dry out
Moisten and reposition tongue every 1–2 hours	Prevents desiccation, circulatory stasis and swelling. Wrapping the tongue in a swab soaked with water can be useful for preventing desiccation
Reposition endotracheal tube every 2 hours	Prevents local tracheal necrosis at site of inflated cuff. Do not inflate the cuff of the ETT excessively. The cuff should be inflated until there is no audible leak during inspiration. High-volume low-pressure cuffs are preferred. Ties used to secure the ETT in place should also be repositioned every 2–4 hours to prevent circulatory stasis of muzzle
Swab pharynx and suction stomach/ oesophagus every 2 hours	Helps minimize regurgitation and aspiration. Even when the cuff of the ETT is inflated, fluid can accumulate proximal to the cuff and enter the airway when the cuff is deflated. It is important that animals with intracranial disease are adequately sedated or anaesthetized during this procedure, as coughing will cause a marked increased in ICP

(Continued)

Table 9 **General nursing of the ventilated patient** (*continued*)

PROCEDURE	RATIONALE
Positioning	Maintain in sternal recumbency if possible, as this optimizes pulmonary and cardiovascular functions. If positioned in lateral recumbency, turning every 2 hours may help prevent atelectasis and compression of dependent muscle groups
Head elevation	In animals with intracranial disease the head should be supported so that it is level with or slightly above the heart in order to encourage venous drainage. Excessive elevation of the head will impair perfusion. Care must be taken to avoid jugular venous occlusion
Environmental temperature control	Monitor body temperature and warm as needed using circulating warm air, blankets, heating pads or hot water bottles. Care must be taken to avoid discomfort or burns from the heat source
Clean and dry	Incontinence pads can be used, but must be changed regularly; alternatively, place an indwelling urinary catheter

Sedation/analgesia

Most animals with respiratory disease will require anaesthesia to allow endotracheal intubation and ventilation. Some degree of sedation or anaesthesia will also be required in animals with neuromuscular disease, particularly during recovery when animals will attempt to move, but are still too weak to maintain their own spontaneous ventilation or protect their upper airway. If anaesthesia is not required to maintain immobility (e.g. animal paralysed by snake envenomation), it is important to remember that these animals are conscious and aware of environmental stimuli, including noise and touch. Some form of analgesia/sedation is recommended to minimize the stress associated with handling. Agents used to sedate/anaesthetize animals requiring ventilation are discussed in Chapter 29.

Monitoring

As mechanical ventilation may have detrimental effects on a variety of organ systems, close monitoring of ventilated animals is essential.

Cardiovascular function

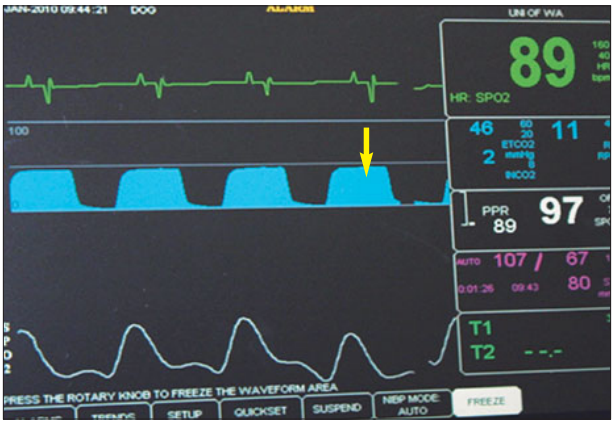
To ensure adequate circulating blood volume and BP and to minimize the detrimental effects of ventilation on cardiovascular function, measurement of arterial BP and CVP should be performed in ventilated animals. These techniques are described in detail in Monitoring the cardiovascular system, p. 56.

Pulmonary function

Adequacy of ventilation

Assessment of the adequacy of ventilation can be made by measurement of airway pressure, capnography (see p. 50) and measurement of arterial CO_2.

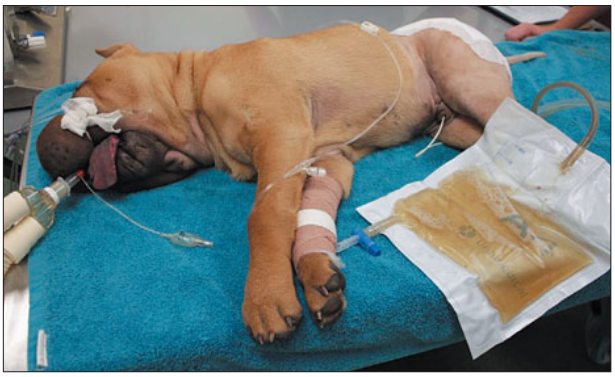
Measurement of airway pressure is essential whenever mechanical ventilation is performed, as sudden changes in pressure can have dramatic consequences. Failure to achieve a predetermined airway pressure indicates inadequate delivery of volume to the patient and may be due to insufficient gas supply, leaks in the breathing system or disconnection. Abrupt increases in airway pressure suggest sudden changes in compliance or resistance and can be caused by obstruction or kinking of tubing, acute bronchoconstriction or development of pneumothorax. Gradual increases in airway pressure suggest deterioration in pulmonary function, with associated decreases in compliance and increases in resistance.



▲ **34 Multivariable monitoring recording haemoglobin saturation and plethysmograph using pulse oximetry. The level of CO_2 expired over time is measured using capnography (blue wave form). Arrow = end-tidal CO_2.**

Adequacy of oxygenation

The amount of oxygen within the blood can be assessed by measuring oxyhaemoglobin saturation (SpO_2) with a pulse oximeter (**34**) and measuring the partial pressure of oxygen in arterial blood (PaO_2). Calculation of oxygen delivery to the tissues requires measurement of both arterial oxygen content and cardiac output. Measurement of mixed venous oxygen can provide an indirect measure of adequacy of oxygen supply to the tissues. Details of these techniques are described below.



▲ **35 A closed urinary collection system allows continuous monitoring of urine output in critical patients.**

Neurological function

Neurological function is influenced by the adequacy of oxygenation, ventilation and perfusion, and a reduction in these variables can lead to neurological deterioration. Deterioration of neurological status will be observed clinically by progressive mental depression in conscious animals, dilation of pupils with absence of a PLR and development of cardiorespiratory abnormalities (e.g. cardiac arrhythmias, Cushing reflex [reflex bradycardia], abnormal breathing patterns). ICP monitoring may also be useful in animals with concurrent intracranial and pulmonary disease, particularly in those requiring ventilation.

Renal function

Measurement of urine output (see p. 60) and urine specific gravity (SG) should be performed regularly in chronically ventilated animals to monitor the effects of ventilation on renal function and to assess fluid balance. Ideally, this should be performed via a sterile indwelling urinary catheter and closed collection system (**35**). Measurement of blood urea, creatinine and electrolytes should also be performed daily to monitor for changes in renal function.

Fluid therapy *(for more details see Chapter 31)*

Once volume deficits have been corrected, crystalloid fluids are required to maintain adequate hydration. As large amounts of water can be lost through evaporation from the airways of ventilated animals, particularly when non-rebreathing systems are used, it is generally recommended that fluid administration is supplied at 1.5–2 × maintenance to prevent dehydration and maintain adequate hydration of the airway mucosa. Humidification of ventilator gases will offset this effect.

In ventilated animals with intracranial disease, fluid therapy should ideally be monitored using CVP to prevent excessive fluid administration and increases in hydrostatic pressure.

Humidification of the airway

Ventilation with dry gases causes drying of airway secretions, decreased mucociliary clearance and associated increased risk of secondary infection. Water lost from the airway will increase fluid requirements. Adequate intravenous fluid therapy is essential for maintaining airway hydration, but heat and moisture exchange (HME) devices (see Chapter 29) placed between the animal and the breathing circuit can reduce water loss by up to 80%. These devices need to be replaced every 6 hours to ensure optimal efficiency.

Ideally, humidified gases should be used if ventilation is expected to be prolonged. Even a couple of hours of ventilation with dry gases can cause significant damage to the upper airways and lungs. Humidifiers can be fitted to most ventilators and are placed in the inspiratory limb of the ventilator breathing system. Sterile saline can be instilled into the airway at the level of the tracheal bifurcation and then removed via suctioning every 1–2 hours. As this increases the risk of nosocomial infection, suctioning is only recommended when secretions are excessive. Suctioning generally requires the patient to be disconnected from the ventilator (open suctioning). This predisposes to alveolar collapse. The performance of an alveolar recruitment manoeuvre may be necessary to re-recruit collapsed alveoli. Alveolar recruitment is achieved by manually inflating the lung with a larger than normal tidal volume or PIP than that being used for ventilation, holding for a prolonged inspiratory time.

Cardiovascular support in the ventilated patient

Vasoactive agents may be indicated in animals with hypotension unresponsive to normalization of blood volume and techniques used to reduce mean airway pressure during ventilation. Conservative approaches to the correction of hypotension should be performed first (fluid therapy and minimizing the delivery of vasodilating agents such as anaesthetics) and preparations made for the administration of vasoactive drugs if indicated. Close monitoring must be performed before, during and after pharmacological management of BP abnormalities. Pharmacological manipulation of BP is described in the sections on hypotension (p. 52) and hypertension (p. 55).

Monitoring oxygenation and ventilation

Pulse oximetry

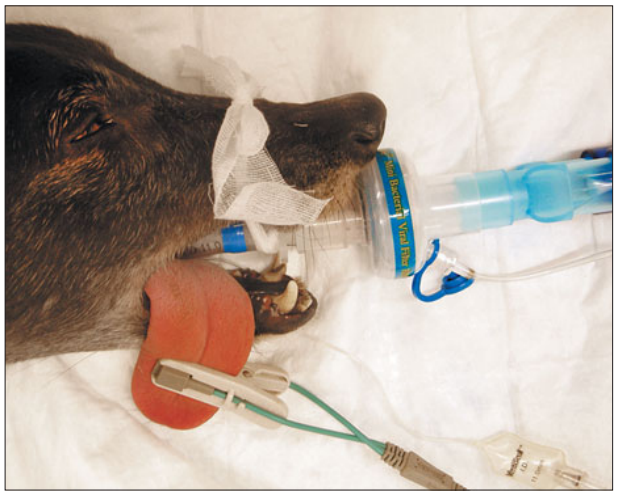
The factors that interfere with a reliable pulse oximeter reading include local vasoconstriction, interference from extraneous lighting, movement, haemoglobin abnormalities and pigment. Pulse oximetry can be used to provide a guide to oxygenation. It provides a non-invasive beat-by-beat assessment of the amount of oxygen carried by haemoglobin within the arterial blood. However, to ensure accuracy, the pulse quality must be good and reflected in the generation of a continuous plethysmogram.

Pulse oximetry measures the percentage of haemoglobin (Hb) molecules that are saturated with oxygen and the pulse rate. Due to the shape of the oxyhaemoglobin dissociation curve (see **24**), a saturation of >95% is required to ensure a PaO_2 of >80 mmHg (10.7 kPa). There are many physiological and technical factors that can interfere with the pulse oximeter, so the readings should be interpreted with a thorough understanding of the limitations of the equipment. (*Note:* Pulse oximetry does not assess the adequacy of ventiliation and severe hypercapnia can develop despite adequate oxygen saturation, especially if the patient is inspiring gases with a high FiO_2. Pulse oximetry can only be relied on to measure oxyhaemoglobin saturation and pulse rate.)

Capnography

Capnography provides a breath-by-breath assessment of the adequacy of ventilation, assuming normal cardiovascular function. This technique measures CO_2 in the expired patient gases ($P'ETCO_2$) (see **34**), which approximates the CO_2 tension in the alveoli ($PACO_2$). As alveolar gases should be in equilibrium with arterial blood, $P'ETCO_2$ can be used to approximate $PaCO_2$.

Intermittent analysis of arterial blood gas samples should also be performed to ensure capnography is providing a reliable indication of ventilation, as discrepancies between arterial and end-tidal CO_2 can occur. Differences between $PaCO_2$ and $P'ETCO_2$ are caused by reduced pulmonary blood flow (and increased physiological dead space) due to pulmonary thromboembolism, air embolism and reduced cardiac output (heart failure, hypovolaemia). In small animal patients



▲ **36 A humidifying and moisture exchange (HME) device, placed between the patient and the anaesthetic circuit, prevents excessive heat and fluid loss during anaesthesia. A capnograph attached to the HME provides breath-by-breath assessment of adequacy of ventilation.**

with cardiovascular compromise, $PaCO_2$ may differ from $P'ETCO_2$ by 10–20 mmHg or more due to development of physiological dead space. Discrepancies will also occur due to increased dead-space ventilation, as less of the inspired tidal volume actually reaches the alveoli. An example of this is panting in a spontaneously breathing animal.

As capnography provides breath-by-breath information and is a non-invasive monitor, it is an important tool for assessing pulmonary and circulatory emergencies in the ventilated patient (**36**).

Arterial blood gases

Measurement of PaO_2 is the gold standard for determining the amount of oxygen within arterial blood and should be performed whenever the accuracy of pulse oximetry is in doubt. Measurement of mixed venous oxygen tension (blood collected from the right atrium via a central venous catheter) can provide an indication of the relationship between oxygen supply and demand. An increase in the difference between arterial and mixed venous oxygen tension suggests either decreased tissue perfusion or increased tissue consumption.

Calculation of oxygen delivery

Oxygen delivery to tissues is a product of cardiac output and arterial oxygen content ($DO_2 = CO \times CaO_2$). A calculation of DO_2 is the ultimate measure of adequacy of tissue oxygenation, but is not always practical. CaO_2 is easily calculated, as it is the sum of oxygen bound to haemoglobin and oxygen dissolved in the plasma ($CaO_2 = [SpO_2 \times Hb \times 1.34] + [0.003 \times PaO_2]$). Because of the requirement for specialist equipment, assessment of cardiac output is usually limited to referral institutions. As a result, cardiac output is usually extrapolated from the measurement of BP (mean arterial pressure [MAP] = cardiac output × systemic vascular resistance [SVR]). An inability to obtain direct measurement of cardiac output prevents the calculation of DO_2 in a clinical setting.

CIRCULATION: CARDIOVASCULAR SUPPORT OF THE NEUROLOGICAL PATIENT

Abnormalities of cardiovascular function can cause secondary neurological damage in animals with intracranial disease. Hypotension leads to reduced CPP, particularly in the presence of increased ICP (CPP = MAP – ICP). Hypertension has the potential to increase ICP in the presence of intracranial disease. This occurs when increases in MAP are in excess of normal autoregulation or when autoregulation is impaired. When autoregulation is impaired, any increase in MAP can cause linear increases in cerebral blood volume and ICP. Therefore, cardiovascular function needs to be monitored closely in neurological patients and hypotension and hypertension managed as required.

Circulatory shock/hypotension

Definition

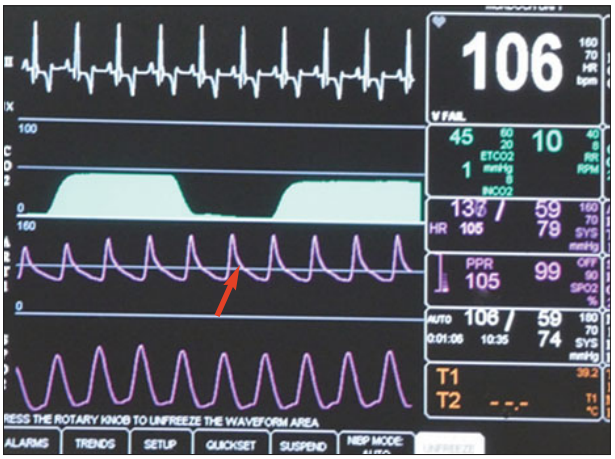
Circulatory shock causes a significant reduction in organ perfusion. During the compensatory phase, BP is maintained by the responses to reduced tissue perfusion, including increases in heart rate (**37, 38**), peripheral vasoconstriction, shifts in fluid from the interstitial space to the intravascular space and reduced urine production. Once the fluid deficit exceeds the ability of the body to compensate (decompensatory shock), decreases in BP occur (**39**).

In humans with head trauma, hypotension is defined as systolic arterial blood pressure (SAP) ≤90 mmHg. Similar limits of MAP in the trauma patient have not been established in small animals, although maintenance of CPP (MAP – ICP) at between 60 and 70 mmHg and MAP >70 mmHg is associated with a better outcome in head-trauma cases. In the absence of ICP monitoring it is recommended that the MAP should be 70–80 mmHg and the SAP should be 100 mmHg to maintain CPP in the presence of increased ICP. Further increases in CPP above 70 mmHg are not recommended, as the use of aggressive fluid therapy and vasopressors to maintain CPP above 70 mmHg is associated with systemic complications such as ARDS.

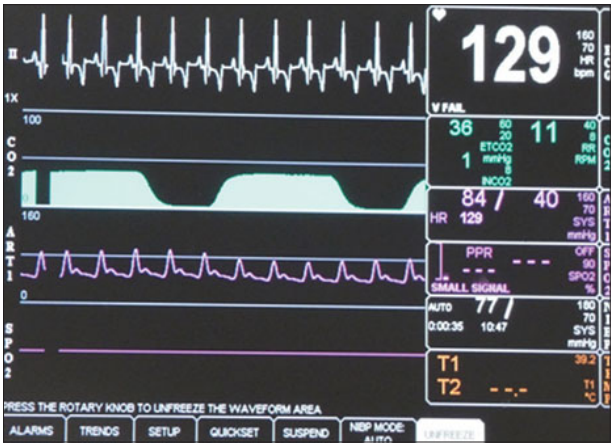
Clinical signs of circulatory shock

Instigation of appropriate therapy is dependent on early recognition of shock. This requires a good understanding of the clinical signs of circulatory shock. The clinical signs of shock will vary depending on the severity, the degree of compensation and the cause.

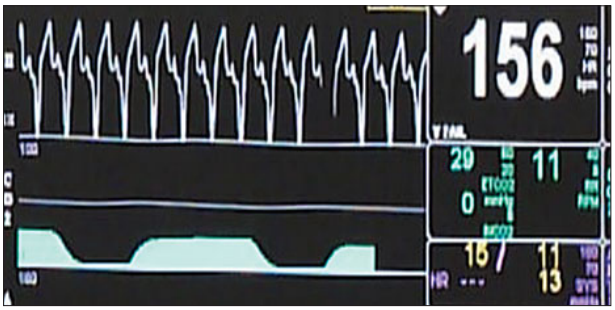
Reduction in cardiac output causes sympathetic nervous stimulation, leading to tachycardia and peripheral vasoconstriction (pale mucosal membranes, cold extremities, reduced rectal temperature). If these changes can compensate for the reduction in cardiac output, BP will be normal. Once these compensatory responses are overwhelmed, BP will decrease. It is essential that therapy is started before decompensation and decreases in BP occur in order to minimize organ damage.



▲ **37 Recording from a patient taken before blood loss. Heart rate (seen here to be 106 beats per minute) and blood pressure (wave marked with arrow), measured using an arterial catheter, are within normal ranges.**



▲ **38 When loss of blood volume is severe or prolonged, compensatory mechanisms fail and hypotension occurs, as can be seen in this recording.**



▶ **39 Electrocardiogram showing tachyarrhythmia, here associated with poor cardiac output and hypotension. In this case urgent management is required, including rapid correction of the cause of the arrhythmia.**

Management of circulatory shock

Appropriate treatment depends on the cause. Arterial BP is influenced by cardiac output and total peripheral resistance. A decrease in either cardiac oputput or total peripheral resistance will decrease BP. Cardiac output is the product of heart rate and stroke volume. Stroke volume is determined by preload (blood volume), myocardial contractility and afterload.

Hypovolaemic shock

A reduction in circulating blood volume results in a decreased amount of blood returning to the heart (preload), with a resultant decrease in stroke volume and cardiac output and, therefore, BP. The most common cause of hypovolaemic shock in the neurological patient is haemorrhage associated with trauma or surgical losses. Trauma may also cause hypovolaemia due to loss of protein-rich fluid into damaged tissues such as muscle bruises and large wounds. In immobile animals unable to access water, dehydration may lead to hypovolaemia once the fluid deficit exceeds 10% of body weight.

Fluid therapy forms the cornerstone of the management of hypovolaemia. (For details on fluid therapy see Chapter 31.)

Vasodilatory/distributive shock

Vasodilatory shock is caused by marked peripheral vasodilation, which results in relative hypovolaemia as the vascular volume increases relative to the volume of blood within the circulation. A primary cause of peripheral vasodilation and hypotension in animals with neurological disease is spinal disease that interferes with the sympathetic innervation of the splanchnic vasculature. A secondary cause is the use of anaesthetic agents that cause peripheral vasodilation (e.g. propofol, isoflurane, sevoflurane). Vasodilation associated with administration of these agents is dependent on the dose and rate of administration. The higher the dose or the faster the rate of administration, the greater the amount of vasodilation and the lower the BP. Vasodilation and hypotension will also occur in any animal that develops concurrent systemic inflammatory response syndrome (SIRS). SIRS may develop in the presence of any factor that can stimulate a widespread inflammatory response (e.g. multiple organ trauma, hypoxaemia and ventilator-induced lung injury).

In most cases, management requires treatment of the underlying cause. Symptomatic treatment includes fluid therapy and administration of vasoconstrictive agents. Drugs used to increase BP in human patients with head trauma include noradrenalin (norepinephrine) infusion, dopamine and phenylephrine. Vasopressin has also been used to increase BP in critically ill animals with SIRS. The dose rates of those agents that have been used in small animals are: noradrenalin (norepinephrine) (0.05–2 µg/kg/minute); dopamine (5–10 µg/kg/minute); vasopressin (0.5–2 IU/kg/minute) and phenylephrine (1–3 µg/kg/minute) (see Further reading).

When sedative and anaesthetic agents are responsible for the hypotension, the dose of the administered agent should be reduced and fluid therapy given to animals that do not respond to the reduction in anaesthesia.

Cardiogenic shock

Cardiogenic shock may occur due to marked changes in heart rate and rhythm, and reduced myocardial contractility. Abnormalities in cardiac rhythm include those associated with fast heart rates (tachyarrhythmias) and slow heart rates (bradyarrhythmias). Hypotension occurs secondary to fast heart rates, as there is decreased time for the heart to fill during diastole, resulting in reduced stroke volume and cardiac output. In addition, the high heart rate increases myocardial oxygen demand when oxygen delivery may be compromised. This predisposes to myocardial ischaemia, which may further exacerbate the arrhythmia. Slow heart rates cause hypotension, as cardiac output is dependent on heart rate ($CO = SV \times HR$). Poor myocardial contractility decreases the amount of blood able to be pumped by the heart and, therefore, cardiac output.

Abnormalities in cardiac rhythm

Tachyarrhythmias

Causes of tachyarrhythmias associated with neurological disease include brainstem disease/compression. Myocardial degeneration and necrosis are also reported to occur following cranial and spinal trauma in dogs. Causes associated with other systemic abnormalities include myocardial contusions secondary to thoracic trauma and myocardial ischaemia secondary to hypovolaemia (e.g. haemorrhage from trauma or surgery). Fluid, electrolyte and acid–base abnormalities may also occur secondary to inappetence, an inability to eat and drink or losses from the gastrointestinal tract or kidneys. (For more details on fluid and electrolyte abnormalities that occur commonly in neurological disease see Chapters 3 and 31.) Persistent tachycardia can also lead to myocardial ischaemia and arrhythmias, as the increase in heart rate causes both an increase in myocardial oxygen consumption and a reduction in oxygen delivery due to decreased time for the heart muscle to be perfused.

Management of arrhythmias involves the correction of all possible underlying causes. Brainstem compression and/or herniation requires specific treatment for increased ICP (see Chapter 20). Brainstem ischaemia can occur due to either increased ICP or decreased BP. Any concurrent electrolyte and acid–base abnormalities also need to be corrected. Any cause of persistent tachycardia, including pain, hypovolaemia or hypotension, needs to be corrected to minimize the risk of exacerbating or contributing to the development of tachyarrhythmias.

Specific anti-arrhythmic medication is indicated if the arrhythmia persists despite treatment of all possible causes and/or if tissue perfusion is compromised. Specific anti-arrhythmics for ventricular tachyarrhythmias include lidocaine (sodium channel blocker) and beta blockers (e.g. esmolol). Supraventricular tachyarrhythmias can be treated using beta blockers. (For dose rates commonly used in clinical cases see Further reading.)

Bradyarrhythmias

The main cause of bradycardia and bradyarrhythmias in animals with neurological disease is brainstem compression (Cushing reflex). Brainstem compression causes sympathetic stimulation, which results in a marked increase in peripheral arterial BP. Initially, the heart rate also increases; however, the marked hypertension ultimately causes a reflex decrease in heart rate due to baroreceptor stimulation. Bradycardia may also occur secondary to drug overdose (e.g. opioid administration). Severe hypothermia and electrolyte abnormalities (hyperkalaemia and hypokalaemia) may also contribute to bradycardia in any critically ill patient.

Brainstem compression requires specific treatment to reduce ICP (diuretics, hyperventilation). (For details on specific management of increased ICP see Chapter 20.) Bradycardia secondary to opioid overdose requires a reduced dose of opioids +/- administration of reversal agents such as naloxone. Specific treatment of bradycardia requires administration of anticholinergics such as atropine. As these agents can mask the signs of brainstem herniation (e.g. pupil and heart rate changes), use of these agents in animals with intracranial disease needs to be performed carefully and increased ICP as a cause of the bradycardia needs to be ruled out before these agents are administered.

Poor myocardial contractility

Decreased cardiac muscle contraction most commonly occurs in neurological patients secondary to the use of anaesthetic and sedative drugs. Development of SIRS secondary to prolonged hypotension, hypoxaemia, ventilator-induced injury or multiple organ trauma may be another cause of reduced myocardial contractility in the neurological patient.

Improving myocardial contractility requires the identification and treatment of underlying causes. For drug-induced decreases in contractility, delivery of the agent needs to be decreased or stopped if possible. Use of multimodal anaesthesia with opioid-based protocols will reduce the requirement for the more cardiovascular and respiratory depressant anaesthetic drugs. The undesirable effects of anaesthesia can also be minimized by using short-acting agents that can be titrated to effect. (For more details of methods of reducing anaesthetic-related cardiovascular depression see Chapter 29.)

Inotropes are indicated when management of the underlying cause does not improve myocardial contractility or when urgent improvement in perfusion is required. Inotropes that can be used include dopamine (2–5 μg/kg/min) or dobutamine (2–5 μg/kg/min).

Obstructive shock

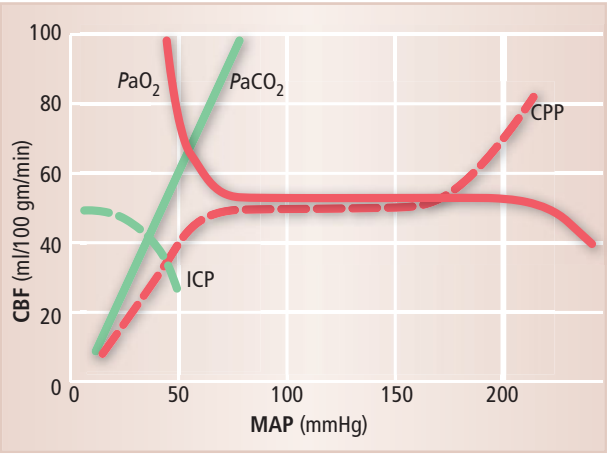
Obstructive shock occurs due to physical interference with venous return to the heart resulting in decreased preload and, therefore, stroke volume and cardiac output. In animals with trauma, obstruction to venous return may occur in association with pneumothorax. In these cases, prompt removal of the air by thoracocentesis or a chest drain will result in resolution of the shock.

Hypertension

Definition

In non-neurological patients, hypertension is defined as an MAP of >100 mmHg and a SAP of >150 mmHg. In neurological patients, increases in mean BP and, therefore, CPP outside the normal autoregulatory range of the brain cause linear increases in blood volume and ICP (**40**). In addition, abnormal brain tissue loses the ability to autoregulate. In these regions, any increase in mean BP is associated with an increase in cerebral blood volume and, potentially, an increase in ICP. Maintenance of a stable normal BP is therefore important in these animals to prevent increases in ICP.

▼ 40 Relationship between cerebral blood flow and intracranial pressure in response to changes in cerebral perfusion pressure, arterial carbon dioxide and oxygen tension. Normal pressure autoregulation of blood flow (dotted red line) maintains appropriate cerebral blood flow despite fluctuation in the mean arterial pressure. Increased $PaCO_2$ (green line) causes a global increase in cerebral blood flow that exceeds demand. Cerebral blood flow is unchanged until PaO_2 levels fall below approximately 60 mmHg, when it rises sharply.



Causes of hypertension

As described above, MAP = CO × SVR. Cardiac output is dependent on heart rate and stroke volume, which in turn are dependent on preload, contractility and afterload. Therefore, an increase in any of these factors may increase BP. The extent of the increase will depend on the degree of compensation.

Causes of hypertension in neurological patients include peripheral vasoconstriction, tachycardia and hypervolaemia (increased preload). Peripheral vasoconstriction can occur due to increased ICP and brainstem compression (Cushing reflex) in animals with intracranial disease. Peripheral vasoconstriction, tachycardia and associated hypertension may also occur secondary to sympathetic stimulation caused by pain and anxiety.

Management

Specific management of increased ICP is detailed in Chapter 20. To minimize other causes of hypertension, adequate pain management and careful administration of fluids are important. Where hypertension persists despite correction of underlying causes, specific pharmacological treatment may be required to prevent detrimental effects on ICP and neurological function.

Persistent hypertension, despite adequate analgesia, can be treated by the administration of beta receptor antagonists or calcium channel blockers. Agents that have been used in human patients with head trauma include esmolol (β blocker), labetalol (mixed α and β blocker) and nicardipine (calcium channel blocker). Use of these agents in small animal neurological patients has not been reported. Esmolol is commonly used to treat tachyarrhythmias in small animals and labetolol has been described for use in a hypertensive crisis. Nicardipine is not used clinically in small animals at present; however, amlodipine, a calcium channel blocker used in small animals with cardiovascular disease, may be a suitable alternative. The dose rates of these agents can be found in critical care texts (see Further reading).

Sodium nitroprusside is also used in small animal patients with cardiac disease to control hypertension; however, this agent is best avoided in patients with neurological injury due to its risk of causing increases in ICP.

The use of any agent that decreases BP should be associated with close monitoring of BP to ensure excessive reduction and hypotension do not occur.

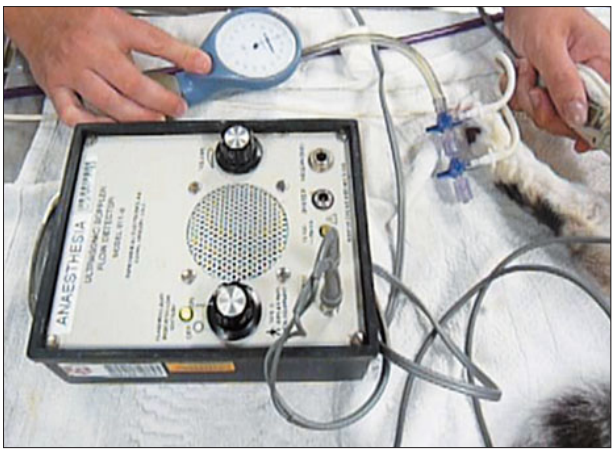
Monitoring the cardiovascular system

Arterial blood pressure

Arterial BP can be measured non-invasively using oscillometric or Doppler techniques (**41**). Oscillometric techniques (e.g. device for indirect non-invasive, automated, mean arterial pressure [DINAMAP]) provide automatic intermittent measurement of mean, systolic and diastolic pressure and pulse rate. The minimum amount of time between repeated measurements is 2 minutes. Many of the commercial systems available are unreliable in very small animals, such as cats, and in hypotensive or bradycardic patients. However, the accuracy in these situations is improving with newer equipment that is becoming more readily available. The Doppler technique is performed manually and is limited to measurement of SAP. However, this technique is more reliable in small animals and in shocked patients.

In unstable animals or chronically ventilated animals, invasive BP monitoring via an arterial catheter (**42**) is preferred over non-invasive methods. Invasive techniques allow continuous monitoring of BP. Catheters are most commonly inserted in the dorsal pedal artery in small animals. In addition, arterial blood gases can be obtained via the arterial catheter, allowing accurate assessment of pulmonary function and oxygen delivery.

▼ **41** **The Doppler technique can be very useful for measuring blood pressure, particularly in small animals.**



▶ **42** **Arterial blood pressure is commonly measured directly via a catheter placed in the dorsal pedal artery. (a) Prior to placement, the hair over the medial surface of the dorsal tarsus is clipped and the site disinfected. (b) A small amount of lidocaine (2%) can be injected subcutaneously over the artery at the intended site of catheter insertion. This is particularly useful in conscious animals to prevent discomfort and movement during insertion. It can also be useful in anaesthetized animals as the vasodilation associated with the lidocaine administration can aid catheter placement. (c) Prior to catheter placement, a small stab incision can be made in the skin. This allows the catheter to be inserted with greater control as there is no longer any skin resistance. The artery is then gently palpated at the site of insertion and the catheter inserted gently but firmly in the direction of palpation. If the artery continues to be palpable as the catheter is inserted, then it is likely that the catheter is too far lateral or medial to the artery. If the artery cannot be palpated, the catheter may be between the artery and skin. A flash of blood will enter the hub of the stylet once the arterial wall has been penetrated by the bevel of the stylet. It is essential that the catheter is not separated from the stylet until at least 5 mm have been inserted to ensure that both stylet and catheter have penetrated the wall of the artery. If this has not occurred, the catheter will not be able to be inserted. (d) Taping and/or superglue can be used to secure the catheter in place. (e) The catheter can then be connected to the fluid line to allow blood-pressure monitoring.**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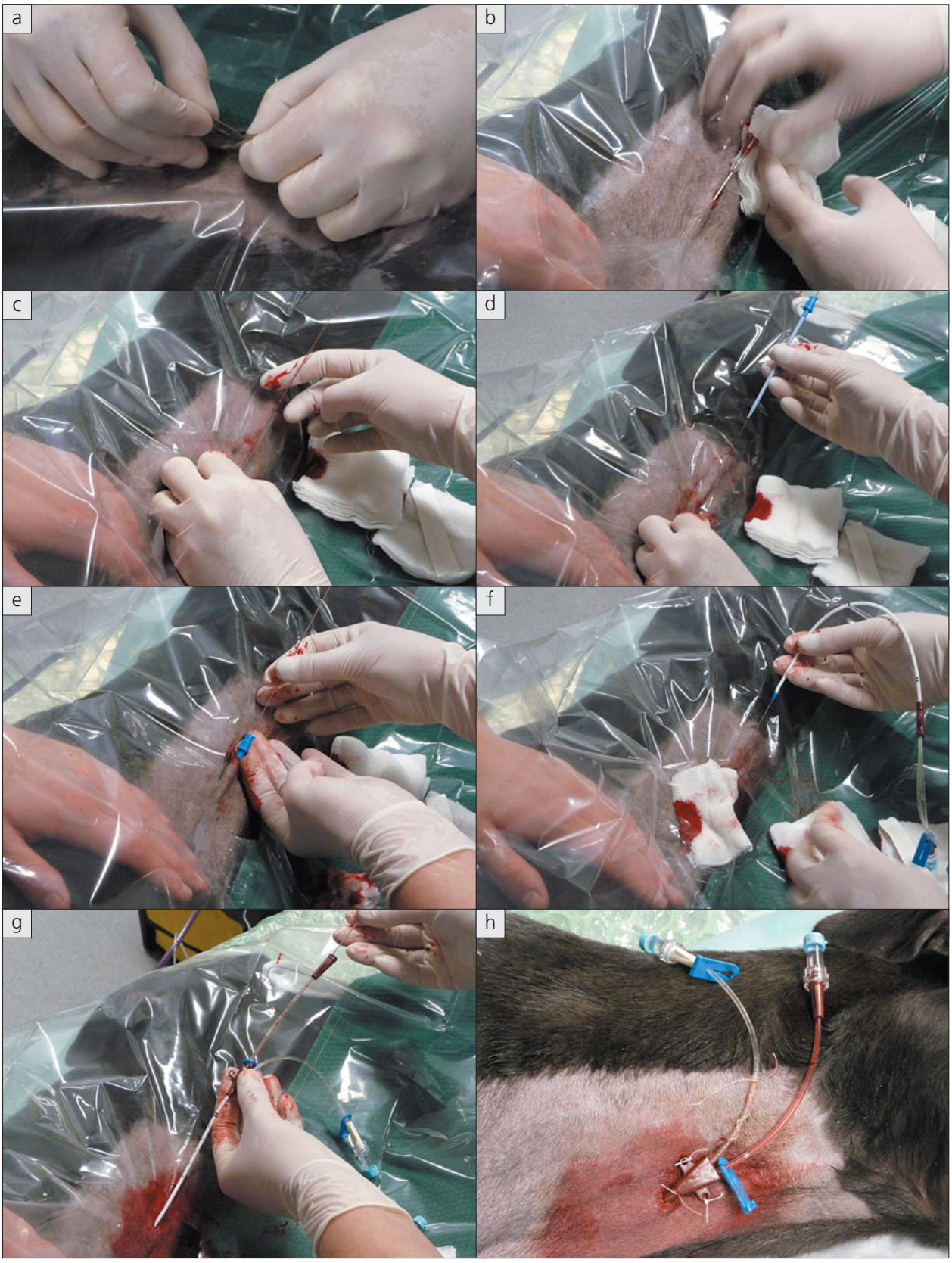
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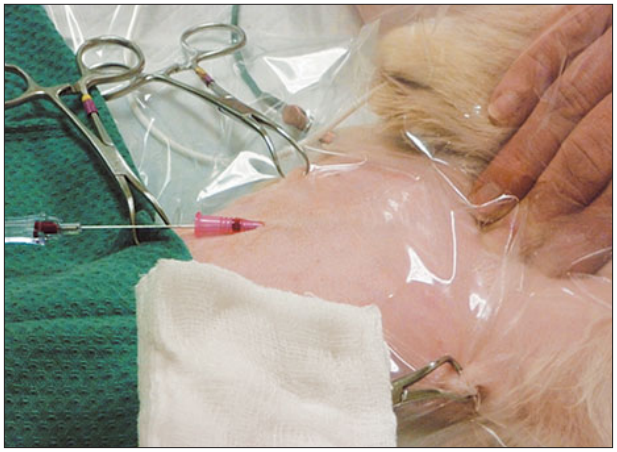
◀ **43 Insertion of a catheter into the jugular vein. Prior to placement, the dead space of the catheter is filled with saline or heparinized saline and an area over the selected jugular vein is clipped. The site is then disinfected. A roll of bandage or gauze placed under the neck can be useful for increasing visibility of the vein, but care should be taken not to occlude the dependent jugular vein in animals with intracranial disease. (a) A small stab incision can be made (carefully) in the skin over the jugular vein, as shown by tenting the skin between two fingers; this assists placement of the catheter. (b) With an assistant compressing the vein proximally, the introducer (a needle or catheter) is inserted through the stab incision into the jugular vein. (c) Once the guide wire has been placed, the needle introducer is removed, leaving the wire in place. (d) The dilator is then passed over the wire and threaded into the vein. (e) A gentle but firm twisting action will help insert the dilator through the skin and vessel wall. The dilator is them removed, again leaving the wire in place. At this stage some haemorrhage will occur at the entry site as the hole in the vessel wall is larger than the wire. Gentle pressure at the entry site using a sterile swab will reduce the amount of haemorrhage until the catheter is placed. (f) The catheter is then passed over the guide wire. During insertion of the catheter it is essential to maintain a hold on the guide wire at all times. The wire is inserted into the proximal end of the catheter and gradually passed up the catheter. (g) When the hub of the catheter is reached, the injection port is removed and the wire passed through the end of the catheter. Once the wire passes through the hub, the operator can hold the wire and pass the catheter over the wire into the vein. The wire is then removed and the injection port replaced. The catheter is flushed with saline. (h) The catheter is sutured in place. A sterile dressing is placed over the entry wound. Neck bandages should not be used in animals with intracranial disease, but can be used in other patients to help secure the catheter in place.**

Central venous pressure

CVP is measured using a fluid-filled catheter inserted into the cranial vena cava via the jugular vein (**43**). Placement of the catheter in the jugular vein may increase the risk of disturbance to venous return and increased ICP. An alternative is to place a percutaneous central catheter, which is passed into the caudal vena cava via a catheter in the medial saphenous vein (**44**). Where this is not possible, a jugular venous catheter can be placed, taking care not to occlude the jugular during placement (surgical exposure may be required) and utilizing a catheter with as small a diameter as possible to minimize interference with venous return. The use of neck bandages over these catheters is discouraged in animals with intracranial disease.

CVP in spontaneously breathing animals is normally between 0 and 10 cm H_2O. CVP is determined by the amount of blood returning to the heart and the ability of the heart to pump this blood. Therefore, measurement of CVP will help differentiate cardiac and hypovolaemic causes of hypotension and determine appropriate treatment. A low BP accompanied by a high CVP suggests that the heart is unable effectively to pump blood returning to the heart, while low BP and CVP supports a reduction in the volume of blood returning to the heart (i.e. hypovolaemia). In contrast, a high CVP and high MAP would suggest hypervolaemia.

Measurement of CVP is helpful to determine the extent of adverse effects of ventilation. A high CVP can be caused by high mean intrathoracic pressure used for ventilation. In such a case, the PIP and/or PEEP requires adjustment.



▶ **44 Central venous pressure can be measured via a catheter placed in the caudal vena cava via the medial saphenous vein.**

Urine output

Urine output provides an indirect indication of renal perfusion and therefore circulating blood volume and hydration status. It is an extremely valuable tool for assessing the adequacy of cardiovascular function in neurological patients.

Urine output is normally 1–2 ml/kg/hour in normovolaemic patients on normal maintenance fluid rates. Higher rates should be expected in normally hydrated animals receiving more than maintenance rates of fluid. Higher than normal output is also expected in animals that have received diuretics or glucocorticoids. (For more details on interpretation of changes in urine output see Chapter 31.)

Monitoring of urine output is easy, cheap and informative. An indwelling urinary catheter is inserted and a closed collection system set up. It is an extremely useful way of assessing fluid balance in unstable neurological patients, particularly those receiving diuretics, where fluid losses in the urine are higher than normal. In addition, animals with traumatic brain injury may have alterations in the reabsorption of renal sodium and water (e.g. central diabetes insipidus [DI] resulting in abnormal urine production and urine SG).