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Monitoring during anaesthesia: techniques and interpretation

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Introduction

There is evidence that safety of anaesthesia in horses should be improved. Results of confidential enquiries of perioperative fatalities reveal that the mortality rate in horses during and after anaesthesia is significantly higher than in canine or feline practice (Johnston *et al.* 1995). Factors associated with this increased risk for complications include **specific age groups** (horses age <1 month or >12 years), **position during anaesthesia** (dorsal recumbency) and **duration of anaesthesia** (anaesthesia exceeding 2 h) (Johnston *et al.* 1995).

Given these risk factors, monitoring physiological functions becomes critical to ensure immediate recognition of trends or abnormalities and provide information that can be used to modify anaesthetic management, thereby avoiding complications (**Table 1**). While it is difficult to document conclusively a positive effect by a specific monitor on outcome from anaesthesia (Ward 1997), it has been recognised in human anaesthesia that **monitors provide better safety** than simple vigilance by the anaesthetist.

Recommendations from the **American College of Veterinary Anaesthesiologists for monitoring during anaesthesia** include measurement of blood pressure in anaesthetised horses (Anon 1995). Experimental and clinical investigations have revealed that **post anaesthetic myopathy is directly related to hypotension** occurring during anaesthesia and that mean arterial blood pressure <70 mmHg for 15 mins is associated with increased incidence of myopathy (Grandy *et al.* 1987; Cribb 1988a; Richey *et al.* 1990). Furthermore, identification of hypotension and appropriate treatment to improve cardiovascular function significantly decreased the severity of post anaesthetic myopathies occurring at one hospital (Young and Taylor 1993).

Acute deterioration of cardiovascular function occurs occasionally in anaesthetised horses (Whitton and Trim 1985; Kellagher and Watney 1986). Therefore, an audible monitor of peripheral blood flow (Doppler flow probe over an artery) or continuous measurement of blood pressure (from a catheter in an artery) should be used to provide an early warning of such changes and to increase the likelihood of successful resuscitation.

A retrospective study investigating the prevalence of intraoperative complications in horses undergoing ophthalmic surgery provided evidence of the **value of measuring anaesthetic gases** (C. M. Trim, unpublished data). In that study, use of the gas analyser was associated with significantly fewer horses moving in response to surgery. Assuming that the risk of a surgical accident is increased when a horse moves during surgery, use of the gas analyser to monitor anaesthetic depth may well have a positive effect on outcome. Additionally, **measurement of anaesthetic gas concentrations should help prevent unintentional deep anaesthesia.**

Monitoring is most effective when response to the information obtained occurs in a timely and appropriate manner. Information obtained during anaesthesia in horses must be interpreted not only using acceptable ranges of values, for heart rate or blood pressure for example, but also in the light of the anaesthetic drug combinations used.

Different drugs produce different sets of 'normal' values. For example, a brisk palpebral reflex is 'normal' during **ketamine** anaesthesia, but the same reflex observed during **thiopentone** anaesthesia indicates a light plane of anaesthesia and the need for administration of additional drugs. Similarly, values for heart rate and blood pressure may be different in horses anaesthetised with **ketamine compared with halothane or isoflurane.** A decrease in mean arterial pressure (MAP) to 70 mmHg is expected during halothane anaesthesia approximately 20–40 mins after induction of anaesthesia, whereas a decrease in MAP to values below 80 mmHg during total i.v. anaesthesia with ketamine should be considered abnormal (**Figs 1** and **2**).

TABLE 1: Summary of commonly used monitors of anaesthetised horses

Essential monitoring
• Observation of movement, reflexes, eye position, gum colour, CRT, depth and character of breathing
• Counting pulse and respiratory rate, character and rhythm of pulse
• Measurement of blood pressure
• Temperature
Additional useful monitoring
• Expired anaesthetic gas
• Electrocardiogram
• Capnography
• Oxygen saturation (pulse oximeter)
• Arterial pH and blood gases

CRT = capillary refill time.

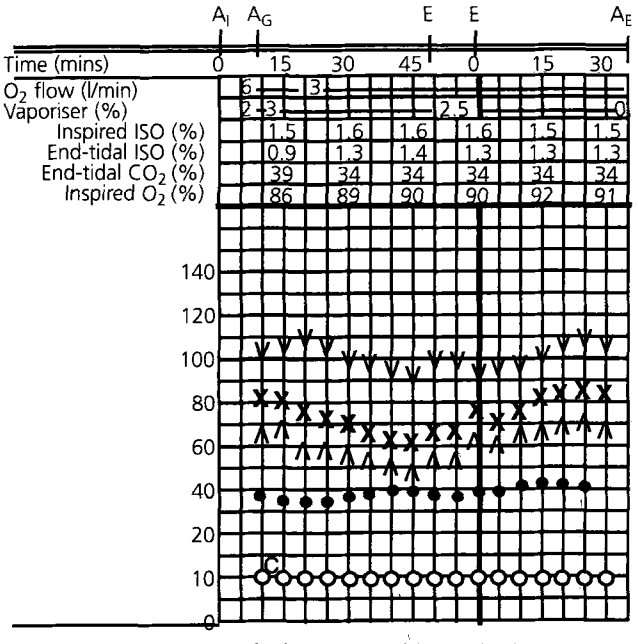

Fig 1: Anaesthetic record of a 3-year-old, 373 kg bwt Quarter Horse gelding during exploration and removal of a foreign body in the region of the pastern. *The gelding was sedated with detomidine 0.01 mg/kg bwt i.v. and anaesthesia induced 8 mins later by gravity infusion of a mixture of 5% guaifenesin and 0.2% thiopentone to a total dose of 600 ml (guaifenesin 80 mg/kg bwt and thiopentone 3.2 mg/kg bwt). Anaesthesia was maintained with isoflurane and controlled ventilation, with the gelding in dorsal recumbency.*

Measurement of end-tidal isoflurane concentration confirmed that after initial stabilisation, a constant concentration of isoflurane was maintained in the horse. Note *that the inspired concentration of isoflurane was approximately half the concentration set on the vaporiser. Acetated Ringer's solution was infused during anaesthesia. The mean arterial pressure (MAP) progressively decreased as the vasoconstrictive effects of detomidine dissipated. Administration of ephedrine 0.06 mg/kg bwt i.v. in divided doses (E, E) increased arterial pressure to above 70 mmHg. Xylazine 0.2 mg/kg bwt i.v. was given after isoflurane was discontinued. Recovery from anaesthesia was rapid and smooth. A_I = Anes induction; A_G = begin inhalant; A_E = extubation; V = systolic arterial pressure; X = MAP; Λ = diastolic arterial pressure; ● = heart rate (beats/min); ○ = controlled ventilation (breaths/min).*

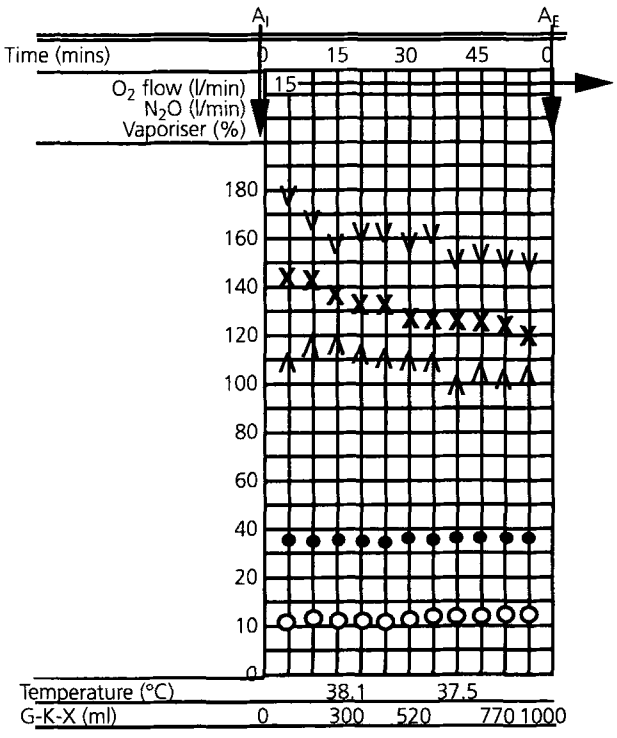

Fig 2: Anaesthetic record of a 15-year-old, 466 kg bwt Quarter Horse mare during lavage of a digital flexor tendon sheath and drain placement. *The mare was sedated with xylazine 0.95 mg/kg bwt and butorphanol 0.02 mg/kg bwt i.v. and anaesthesia induced 5 mins later by i.v. injection of ketamine 2.2 mg/kg bwt. Anaesthesia was maintained by infusing the combination of 1 litre 5% guaifenesin (G), 1300 mg ketamine (K) and 650 mg xylazine (X) at a rate of 2 ml/kg bwt/h. The trachea was intubated and O_2 15 l/min insufflated into the endotracheal tube. Blood pressure was monitored at the tail by oscillometry (DINAMAP). Anaesthesia was continued for 62 mins during which the whole litre was infused. The mare stood at the first attempt 45 mins later. The high mean arterial pressures (MAP) measured in this horse are expected during anaesthesia with this drug combination. Heart rates and respiratory rates were within the expected ranges of 30–40 beats/min and 10–20 breaths/min, respectively. A_I = Anes induction; A_E = extubation; V = systolic arterial pressure; X = MAP; Λ = diastolic arterial pressure; ● = heart rate (beats/min); ○ = spontaneous ventilation (breaths/min).*

In this report, the depth of anaesthesia, cardiovascular and respiratory status are discussed in relation to methods of monitoring endpoints.

Depth of anaesthesia

Movement of the head or limb in response to a noxious stimulus is a sign of inadequate anaesthesia in horses for all anaesthetic agents. In contrast, eye movements, reflexes, respiratory rate, heart rate and arterial blood pressure vary with different anaesthetic agents with changes in depth of anaesthesia.

For instance, **eye movements** occurring during anaesthesia are similar with thiopentone and the inhalation agents; the eye rotates into a rostroventral position during a moderate depth of anaesthesia that is sufficient for surgery. At this level of anaesthesia, the palpebral reflex is weak and nystagmus is absent (**Table 2**).

The development of a brisk palpebral reflex and the appearance of nystagmus are associated with lightening of anaesthesia, and the eye may also rotate dorsocaudally. **Lacrimation** may occur during light anaesthesia. During deep anaesthesia with inhalation agents **small oscillations of the eye resembling nystagmus** may occur, but these oscillations have variable amplitudes. These movements **should not** be used as an indication to increase administration of anaesthetic agent if the palpebral reflex is absent and the eye is in a central position.

In contrast, **ketamine anaesthesia** causes the eye to be either centrally placed or directed slightly rostroventral, the palpebral reflex is brisk and small amplitude nystagmus is often present (**Table 2**). As the blood concentration of ketamine decreases, the eye may rotate into a more rostral position and

TABLE 2: Signs associated with different depths of anaesthesia produced by thiopentone-guaifenesin (T-G) or ketamine ± guaifenesin (K ± G)

	T-G			K ± G		
	Light	Medium	Deep	Light	Medium	Deep
Eye position	C or DC					
	DC	RV	C	RV	More C	C
Nystagmus	+	-	-	+	+	-
Palpebral reflex	++	+	-	++	++	+
Sigh	+	-	-	+	-	-
Swallow	+	-	-	+	+	-

C = Central; DC = dorsocaudal; RV = rostroventral.

TABLE 3: End-tidal concentrations (%) of inhalation agents producing light to moderately deep planes of anaesthesia. The lowest values given may be insufficient to provide anaesthesia for surgery, except when detomidine or opioids have been administered for premedication or in systemically ill horses. Typically, a vaporiser setting of up to twice these values is required to achieve these end-tidal values during maintenance of anaesthesia

	Depth of anaesthesia		
Anaesthetic agent	Light	Moderate	Moderately
Halothane	0.9%	1.1%	1.35%
Isoflurane	1.3%	1.56%	1.95%
Sevoflurane	2.3%	2.8%	3.45%

the amplitude of nystagmus increases. Approximately 12 (10–20) mins after injection of ketamine, when the anaesthetic plane is light, **the horse may sigh**. As consciousness returns, the horse may **move its ear** in response to noise and shortly thereafter may move a leg or lift the head.

Measurements of heart rate, blood pressure and respiratory rates are not reliable guides to the depth of anaesthesia as horses may move during anaesthesia before changes in heart rate or arterial pressure occur. Even a constant depth of anaesthesia with halothane or isoflurane may be associated with a progressive increase in MAP in the first 2–3 h of anaesthesia (Steffey *et al.* 1987a).

Hypertension may be caused by increased arterial carbon dioxide tension ($PaCO_2$) from **hypoventilation** or by a **loose cuff**, or **too small a cuff**, causing an error in blood pressure measurement.

Conversely, **increasing the depth of anaesthesia with halothane, isoflurane** or **sevoflurane** consistently produces decreases in arterial pressure and cardiac output without changes in heart rates (Steffey and Howland 1980; Aida *et al.* 1996). Increasing depth of anaesthesia with halothane may cause an increase in respiratory rate, whereas the respiratory rate is usually decreased during deep anaesthesia with isoflurane (Steffey and Howland 1980).

A low respiratory rate may also result from the inclusion of an opioid, such as butorphanol, in the anaesthetic protocol.

Anaesthetic agent concentration

An anaesthetic gas analyser can be used to measure the anaesthetic gas concentration in inspired and expired gas (**Fig 3**). The anaesthetic gas concentration at the end of exhalation (alveolar or end-tidal concentration) is a reasonable approximation of the brain anaesthetic concentration. **The concentration required to produce anaesthesia is different for each of the inhalant anaesthetic agents** and is quantified as the minimum alveolar concentration (MAC) that will prevent 50% of a group of horses responding to a painful stimulus.

The MAC values for halothane, isoflurane and **sevoflurane** are 0.9, 1.3 and 2.3%, respectively (Steffey *et al.* 1977; Aida *et al.* 1994). Consequently, a higher concentration of isoflurane than halothane, and an even higher concentration of sevoflurane, is required to produce anaesthesia.

Anaesthetic requirements are less in very young foals, with MAC values of 0.7% for halothane and 0.9% for isoflurane (Dunlop 1994).

End-tidal concentrations exceeding MAC values (usually 1.2–1.5 times MAC) will be required to prevent movement in response to surgery (**Table 3**). Higher concentrations of inhalation agents cause unacceptable cardiovascular depression and a balanced technique utilising either an opioid or an α_2-sedative for analgesia is advisable. Therefore, **when a gas analyser is available, the vaporiser setting and O_2 flow rate are adjusted to maintain the end-tidal concentration between 1 and 1.5x MAC**, and assessment of depth of anaesthesia from observations of vital signs and cardiovascular responses can be used as a guide for adjusting the vaporiser or administration of supplemental i.v. anaesthetic agents.

A gas analyser is **particularly useful in equine anaesthesia when O_2 flows of ≤4 l/min** are used with a circle circuit for maintenance of anaesthesia. The uptake of anaesthetic agent by the horse results in exhalation of gas with a low anaesthetic concentration, causing the anaesthetic concentration in the rebreathing bag to be approximately 50–60% of the concentration set on the vaporiser (**Fig 1**). **A gas analyser therefore allows accurate determination of the true inspired concentration of anaesthetic agent.**

Not all analysers measuring halogenated anaesthetic agents by infrared absorption spectrometry can be used for horses. The Datex Normac/Capnomac, for example, will measure **exhaled methane** and record the concentration as halothane (Taylor 1990). Measurements made by analysers that use higher wavelengths of infrared light should be unaffected by methane (Moens *et al.* 1991).

Electrophysiological monitoring of the brain and spinal cord

In human anaesthesia, intraoperative monitoring of the brain and spinal cord is used to reduce morbidity associated with operations involving the central nervous system or its blood supply. There have been several investigations in horses evaluating **electroencephalographic** (**EEG**) changes with changes in depth of anaesthesia as determined by clinical

signs or end-tidal concentrations of halothane (Otto and Short 1991; Ekstrom *et al.* 1993; Johnson *et al.* 1994). The **EEG** may be influenced by a variety of factors occurring during anaesthesia, including cerebrocortical depression, hypotension, hypoxaemia and hypercapnia. A computerised EEG technique, power spectrum analysis described by 80 or 95% spectral edge frequency, has potential application for monitoring depth of anaesthesia (Otto and Short 1991; Johnson *et al.* 1994; Otto *et al.* 1996).

Cardiovascular system

Considerable information on adequacy of cardiovascular function can be obtained by:

- Assessing colour of gums.
- Capillary refill time (CRT).
- Heart rate.
- Palpation of peripheral pulse strength and rhythm.

Tissue perfusion is decreased when the gums are pale, rather than pink, or the CRT exceeds 1.5 secs. Heart rate is a major determinant of cardiac output and a rate <24 beats/min during anaesthesia is usually associated with low cardiac output. In contrast, significant changes in blood pressure occur frequently during inhalation anaesthesia without a corresponding change in heart rate (**Fig 1**).

A **MAP of ≤65 mmHg (hypotension)** is associated with a significant decrease in tissue perfusion. **Potential consequences of hypotension** include myopathy, renal failure or blindness after anaesthesia. Mean pressure as low as 55 mmHg may precede cardiac arrest.

Measurement of blood pressure allows recognition of hypotension and institution of appropriate treatment, such as decreasing anaesthetic depth or i.v. administration of fluids or a vasoactive drug such as ephedrine, dopamine or dobutamine (**Fig 1**). When MAP exceeds 70 mmHg, palpation of the strength of the peripheral pulse and observation of oral membrane colour and CRT should be used to assess adequacy of peripheral perfusion and cardiac output.

For example, an increase in blood pressure during anaesthesia in response to a surgical stimulus is frequently associated with blanching of oral membranes, indicating that the increase in pressure was caused by peripheral vasoconstriction. Cardiac output may be unchanged or even decreased during such responses, and decreased tissue perfusion may occur (Wagner *et al.* 1996).

Arterial blood pressure is lower during anaesthesia in foals than in **mature** horses. In a study of healthy foals age 5 or 6 days anaesthetised with isoflurane, MAP was 58 mmHg. When the same foals were re-anaesthetised at age 4–6 weeks, the average MAP had increased to 80 mmHg, and there was a corresponding decrease in cardiac index (Hodgson *et al.* 1990). In a study of foals anaesthetised at equivalent depths of anaesthesia with halothane or isoflurane, there were minimal differences in arterial pressure and peripheral resistance; however, cardiac index was higher during isoflurane anaesthesia (Dunlop *et al.* 1990). Increasing depth of anaesthesia resulted in decreases in arterial pressure and peripheral resistance, and increases in heart rate.

Palpation of a facial artery may provide a rough estimate of arterial blood pressure, although **vasodilation occurring during inhalation anaesthesia** results in a large diameter artery that may give the impression of a strong pulse even when pressure is low. **Conversely**, anaesthesia caused by xylazine or detomidine with ketamine is characterised by peripheral vasoconstriction and the pulse may feel thready even though the arterial pressure is high (**Fig 2**).

In general, palpation of a strong pulse within a turgid artery is an indication of adequate blood pressure, whereas arterial pressure is low when digital palpation collapses the artery. More

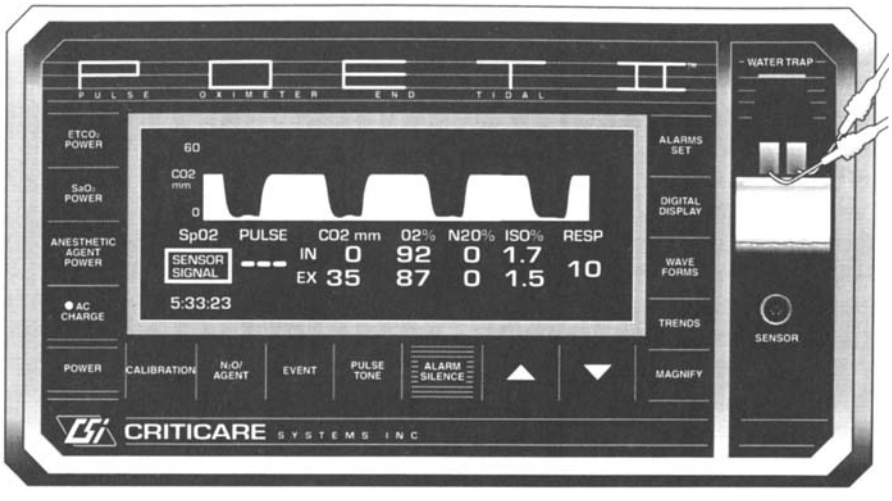

Fig 3: This gas analyser (POET II)[1] aspirates a continuous flow (150 ml/min) of gas from an adapter with a side port inserted between the endotracheal tube and the anaesthetic circuit. The monitor is currently measuring an inspiratory isoflurane concentration of 1.7% and an end-tidal concentration of 1.5%. Other information displayed is that the inspired O_2 concentration is 92%, respiratory rate is 10 breaths/min (the horse is on controlled ventilation) and the end-tidal CO_2 concentration is 35 mmHg. The pulse oximeter capability is not in use.

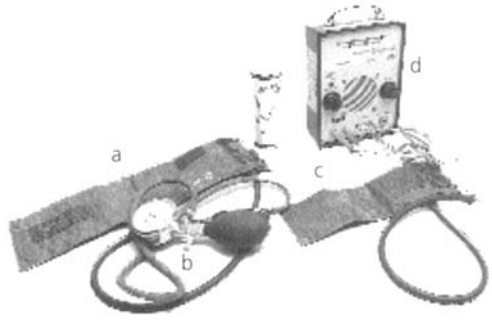

Fig 4: Indirect measurement of blood pressure using doppler ultrasound technique (Model 811)². a = Cuff; b = manometer; c = probe; d = audio monitor.

accurate monitoring of blood pressure provides information that can be used to minimise deviations from normal physiological status and thereby reduce morbidity and mortality.

Indirect measurement of blood pressure

Doppler ultrasound

The 2 most commonly used noninvasive methods of measuring arterial blood pressure are **Doppler-shifted ultrasound** and **oscillometry**.

Indirect measurement of blood pressure using the **Doppler ultrasound technique** is achieved by wrapping a cuff snugly around the base of the tail with the cuff air bladder centred over the ventral surface of the tail. A probe covered with contact gel is taped distal to the cuff on the ventral midline groove over the coccygeal artery (**Fig 4**). The probe emits ultrasound waves and detects the signal reflected by the moving arterial wall. The receiver detects the difference in frequency between transmitted and reflected ultrasound waves (Doppler shift) and produces an audible swooshing sound. The cuff, which is connected to an anaeroid manometer, is manually inflated to above systolic pressure so that no arterial motion occurs and no sound is heard. The pressure in the cuff is gradually released until the first sounds of blood flow are detected at systolic pressure. As additional pressure is released from the cuff, diastolic pressure is heard as a change in character of sound from a 1- or 2-beat sound to a multiple beat sound, or as a muffling of sound or growl. This will occur 20–40 mmHg below systolic pressure. **Mean pressure can be calculated** as one-third of the pulse pressure (systolic - diastolic) plus diastolic pressure.

Readings may be recorded as **coccygeal uncorrected values** or, when the horse is in dorsal recumbency, **corrected to heart level by subtracting 10 mmHg for every 13.6 cm difference** in distance between the cuff and the point of the shoulder. The sounds associated with diastolic pressure are not obvious in all horses and the determination of diastolic pressure is subjective without the experience of previous comparisons with values obtained by direct arterial pressure measurement.

Although monitoring blood pressure has proven to be highly valuable in improving the quality of anaesthetic management, errors in measurement occur. A **cuff that is attached too loosely or slips down the tail** and becomes loose during positioning of the horse will result in an erroneously high value. The **width of the air bladder within the cuff is important for accuracy**; a bladder that is too narrow will overestimate blood pressure and one which is too wide will underestimate it. In an investigation of the influence of the ratio of cuff bladder width to tail circumference, **a ratio of 0.48** underestimated systolic pressure and overestimated diastolic pressure by approximately 9% (Parry *et al.* 1982). In mature Thoroughbreds, this value corresponded to a bladder width of 10.4 cm. Measurements of systolic arterial pressure in horses anaesthetised in dorsal recumbency with halothane using a cuff bladder to tail circumference ratio of 0.41 had an error range of ± 20 mmHg in 5% of horses (Bailey *et al.* 1994).

Oscillometry

The DINAMAP is a commonly used monitor utilising the oscillometric method of blood pressure measurement. A cuff is wrapped around the tail of mature horses or around the hindlimb in foals near the metatarsal artery. When the cuff is placed around the tail, it should be several inches from the base of the tail (6–10 cm in a mature horse), where the tail diameter is constant for the length of the cuff. The cuff should not be wrapped tightly. The cuff is automatically inflated to a high pressure and, as pressure is released from the cuff, pressure changes occurring within the cuff as a result of adjacent arterial pulsations are detected by a transducer within the monitor. Values for systolic, diastolic and mean arterial pressures and heart rate are displayed digitally and the monitor can be programmed to automatically measure at a specific time interval.

Early investigations of the **DINAMAP** confirmed accurate and clinically useful values for arterial pressure using a **cuff width to tail circumference ratio of 0.24 in ponies** (Geddes *et al.* 1977) and **0.25 or 0.35 in horses** (Latshaw *et al.* 1979; Muir *et al.* 1983). Arterial pressure values obtained with the DINAMAP were not accurate at heart rates <25 beats/min. This author's experience using a Model 8300 **DINAMAP** and a cuff width to tail circumference ratio of 0.35 to 0.40 (child or small adult cuff for a mature horse depending on tail thickness and the amount of hair) has been that the mean arterial blood pressure value obtained from this monitor is usually the same as that obtained by direct blood pressure measurement. Occasionally, the DINAMAP records pressures 10–20 mmHg higher than the actual MAP. This monitor senses pressure changes within the cuff and any movement of the extremity, for example, during preparation of the skin of the abdomen or a hindlimb for surgery, will prevent the monitor from obtaining a measurement. Additionally, this monitor is quite expensive.

There is no question that measurement of blood pressure by indirect means provides useful information in most horses. That these methods produce erroneous values in even a small number of horses is of concern and lends weight to the recommendation that blood pressure be measured by direct means whenever possible.

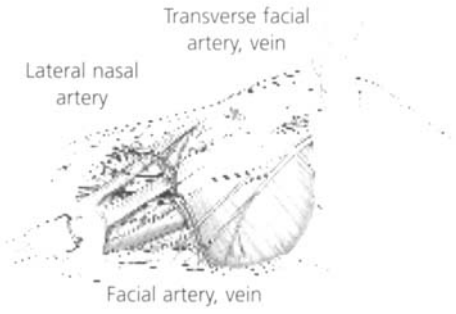

Fig 5: Peripheral artery sites for direct measurement of blood pressure. The lateral nasal, facial or transverse facial arteries on the side of the head are most easily catheterised in horses in lateral recumbency. The facial artery where it curves around the jaw is usually catheterised in horses lying in dorsal recumbency.

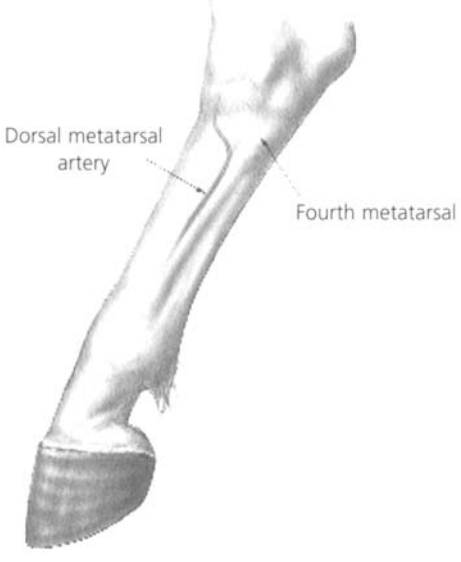

Fig 6: The dorsal metatarsal artery on the lateral side of the hindlimb is used for monitoring blood pressure in horses in lateral recumbency undergoing surgery of the head.

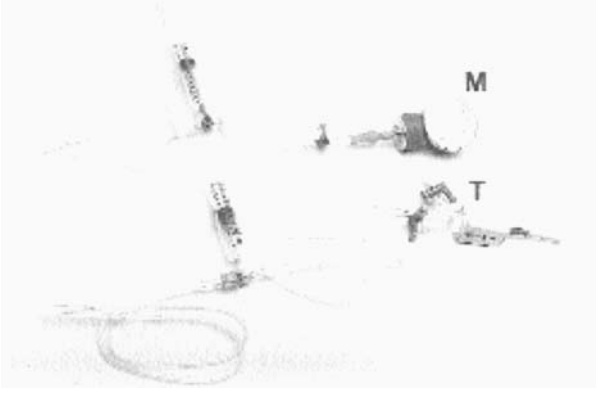

Fig 7: Arterial pressure can be measured with an anaeroid manometer (M) or an electrical transducer (T). An optional addition (not shown) is a continuous flushing device attached to a bag of saline pressurised to 200 mmHg that will deliver 2–4 ml saline/h.

Direct measurement of blood pressure

Direct measurement of blood pressure is achieved by insertion of a 20 or 22 gauge catheter or Butterfly needle into a peripheral artery, commonly the facial, lateral nasal, transverse facial, or metatarsal arteries (**Figs 5** and **6**). The hair should be clipped and the skin over the artery prepared aseptically, with care taken to avoid letting solutions enter the eyes.

With practice, the time taken to insert a catheter and connect equipment to measure blood pressure is short. Pressure is transmitted through saline-filled tubing and measured by either an anaeroid manometer or an electrical pressure transducer (**Fig 7**). When measuring pressure with the anaeroid manometer, the tubing between the central stopcock and the 20 gauge needle should be filled with **heparinised saline**. When the needle is inserted in the arterial catheter, blood entering the tubing should be flushed back into the artery. **An air gap should always be maintained in part of the tubing next to the manometer to maintain sterility and to prevent wetting of the manometer.** Alternatively, a rubber diaphragm interface can be inserted next to the manometer.

The needle of the anaeroid manometer deflects slightly with each beat and the value at the upper deflection of the needle is slightly less than the values obtained by direct measurement (Riebold and Evans 1985). For accurate measurement, the air-saline junction in the tubing connected to the manometer, or the electrical transducer, are **zero reference points** and should be placed level with the right atrium or the point of the shoulder when the horse is in dorsal recumbency or level with the sternum or spine when in lateral recumbency.

The manometer costs very little but provides only MAP values. The initial cost of an electrocardiogram (ECG) and blood pressure monitor can be high; however, the **electrical transducer provides more information.** Digital values for systolic, diastolic and MAPs, heart rate, and a waveform can be observed on the oscilloscope (**Fig 8**).

Important advantages of direct measurement of blood pressure are the reliability of measurement and the ability to observe the pressure continuously and immediately detect an abnormality.

Electrocardiogram

Electrocardiography is used to identify abnormal cardiac rhythm detected before or during anaesthesia. A frequently used monitor lead is the **'base-apex' lead**. The **right arm electrode** is clipped on the neck in the right jugular furrow and the **left arm electrode** is clipped at the apex of the heart over the left 5th intercostal space several inches from midline. The **left leg electrode** on the neck or shoulder. Lead I is selected on the electrocardiograph (**Figs 8** and **9**).

Good electrical contact must be achieved with alcohol or electrode gel. The lead to the left arm electrode should be passed between the forelimbs to avoid a wandering up-and-down baseline caused by respiratory movement.

There is a high incidence of sinus arrhythmia and first

and second degree atrioventricular (AV) heart block in conscious unsedated horses (Robertson 1990). In contrast, **dysrhythmias during anaesthesia are uncommon**. Premedication with detomidine, or supplemental i.v. injections of xylazine during anaesthesia, may cause **second degree AV block** to develop. The **sudden appearance of this dysrhythmia** during anaesthesia on any other occasion, particularly in a foal with a ruptured bladder or a horse with acute or chronic infection, **is cause for concern** as this abnormal rhythm may progress within a few minutes to **advanced heart block** (P waves only, no ventricular complexes) and **cardiac arrest**. In some horses, occasional isolated atrial premature complexes can be heard and observed on the ECG. These are usually well tolerated. **Atrial fibrillation** and **ventricular premature complexes** occur rarely and may require specific treatment if associated with hypotension (Muir and McGuirk 1984).

Respiratory system

Visual observation of respiratory rate and depth of breathing is a basic estimate of adequacy of breathing. In general, a spontaneous rate of ≤6 breaths/min constitutes respiratory depression. Respiratory rates of ≥10 breaths/min may provide adequate ventilation or the breaths may be shallow resulting in **hypoventilation**. **Measurement of the partial pressure of carbon dioxide** ($PaCO_2$) in a sample of arterial blood is **the best monitor of ventilation**. Capnography indirectly estimates $PaCO_2$ by measuring the concentration of CO_2 in expired alveolar gas. **Capnography** is also useful for diagnosis of mechanical problems in anaesthetic circuits, airway obstruction and cardiogenic shock.

Increased $PaCO_2$ (hypercapnia) is a direct consequence of **hypoventilation** and commonly occurs during anaesthesia. Normal values for $PaCO_2$ in conscious unsedated horses and ponies vary between 5.0 and 6.1 kPa (38–46 mmHg) (Steffey *et al.* 1987b; Clarke *et al.* 1991; Wagner *et al.* 1991; Wan *et al.* 1992). $PaCO_2$ values >8 kPa (60 mmHg) are indicative of significant respiratory depression.

Hypercapnia may cause stimulation of the sympathetic nervous system, increased blood pressure and cardiac output (Wagner *et al.* 1990; Khanna *et al.* 1995). Adverse effects of hypercapnia are observed in some horses as tachycardia of 60–70 beats/min, or hypotension caused by **decreased myocardial contractility**. These abnormalities are corrected within 5–10 mins by initiating controlled ventilation. More frequently, the effects of hypoventilation during inhalation anaesthesia are manifested as an inadequate depth of anaesthesia despite a vaporiser setting that should provide a sufficient depth of anaesthesia. In these horses, controlled ventilation expands the lungs, improving uptake of anaesthetic agent and resulting in increased depth of anaesthesia.

Arterial oxygenation can be monitored by measurement of the partial pressure of O_2 in a sample of arterial blood (PaO_2) or indirectly by attaching a sensor to the tongue, for example, and measuring **O_2 saturation of arterial blood**

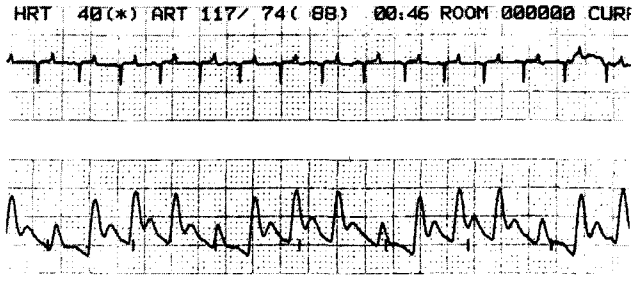

Fig 8: Normal base-apex ECG Lead 1 and arterial blood pressure recording. This horse has a systolic pressure of 117 mmHg, diastolic pressure of 74 mmHg and mean pressure of 88 mmHg. *The notch on the downslope of the trace is associated with closure of the aortic valve. The effect of controlled ventilation can be observed on the recording as rhythmic decreases in arterial pressure from decreased venous return caused by increased intrathoracic pressure.*

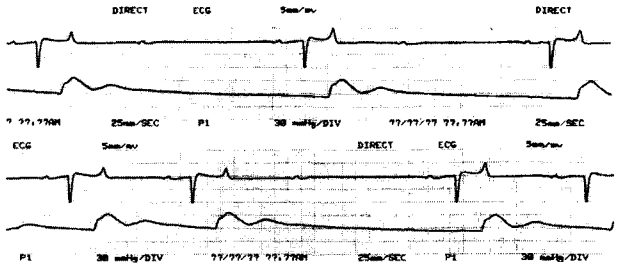

Fig 9: Second degree atrioventricular heart block. Note that P waves without ventricular complexes have no accompanying blood pressure pulse.

(**pulse oximetry**). PaO_2 values are influenced by the:

- Inspired O_2 percentage.
- Adequacy of ventilation.
- Cardiac output.
- Blood pressure.

A PaO_2 of 12–14.6 kPa (90–110 mmHg) is normal in conscious, unsedated horses at sea level; values <8 kPa (60 mmHg) constitute hypoxaemia. **Arterial O_2 saturation (SaO_2)** is the percentage of haemoglobin saturated with O_2. The relationship between PaO_2 and SaO_2 is not linear because haemoglobin changes its affinity for O_2 at increasing levels of saturation, and the association is further altered by pH and temperature of the blood. **The following values are of importance:**

- Arterial blood: PaO_2 12 kPa (90 mmHg) = 97% SaO_2.
- Arterial hypoxaemia: PaO_2 8 kPa (60 mmHg) = 90% SaO_2.
- Venous blood: PaO_2 5.3 kPa (40 mmHg) = 75% SaO_2.

Oxygen delivery to tissues is defined by the O_2 content (O_2 combined with haemoglobin and dissolved in plasma) and cardiac output, although O_2 delivery to an individual organ is influenced by the blood flow to that specific organ. **Hypoxia** is inadequate tissue oxygenation and may be caused by low arterial O_2 content or by inadequate blood flow.

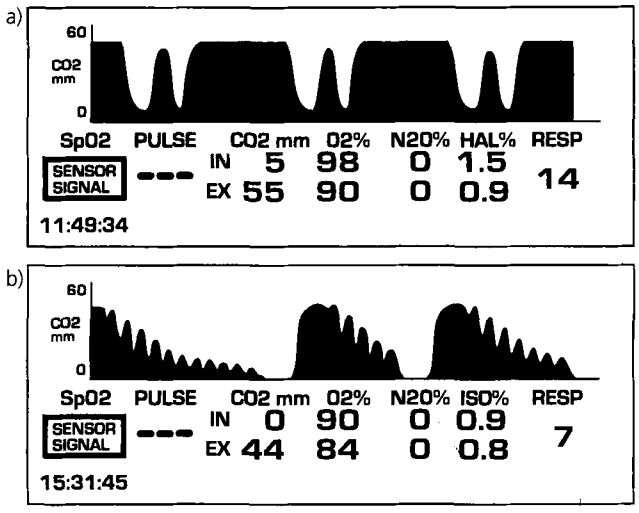

Fig 10: Line drawings from the capnograph. These breathing patterns may be observed in spontaneously breathing horses anaesthetised with halothane or isoflurane. a) *This horse was anaesthetised with halothane at an end-tidal concentration of 0.9% and was breathing in a biphasic pattern at 14 breaths/min. End-tidal CO_2 concentration was 55 mmHg and the monitor shows a small amount of CO_2 rebreathing.* ***b)*** *This horse was breathing at 7 breaths/min resulting in an end-tidal CO_2 concentration of 44 mmHg. The recording shows that inspiratory time was slow and the effects of heart beats on adjacent lung can be seen.*

TABLE 4: Mean ± s.d. (range) $PaCO_2$ (Model 170)[3] and end-tidal CO_2 (POET II or POET IQ)[1] taken at the same time within the first hour of anaesthesia from 125 horses anaesthetised in dorsal recumbency with isoflurane for colic surgery (C. M. Trim, unpublished data, 1995–1997)

$PaCO_2$	End-tidal CO_2
6.2 ± 1.3 kPa	4.5 ± 0.9 kPa
46.8 ± 9.4 mmHg	33.5 ± 7.0 mmHg
(30.8–89.5 mmHg)	(23–64 mmHg)

A **horse with a low haemoglobin level will have low blood O_2 content** despite PaO_2 and SaO_2 being within their normal ranges. Anaesthetic management of anaemic horses should therefore include administration of 100% inspired O_2 and cardiovascular support. **Hypoxaemia frequently develops in horses during general anaesthesia as a result of severe lung collapse. Patients at greatest risk are foals** with pneumonia and horses with abdominal distension from pregnancy or colic. Cyanosis may be suspected, but is usually not obvious in horses. Monitoring by blood gas analysis or pulse oximetry will confirm low PaO_2 or SaO_2.

Capnography

Measurement of exhaled CO_2 provides a noninvasive method of monitoring continuously alveolar and arterial PCO_2. Gas is aspirated from the endotracheal tube or Y-piece (Matthews *et al.* 1990) and the capnometer measures CO_2 concentration by infrared absorption. Gases leaving the analyser should be directed back into the anaesthetic circuit or into the scavenging system.

The capnometer provides breath-by-breath numerical values for CO_2 concentration and some monitors display the CO_2 waveform (capnograph). The upward slope of the waveform represents expiration and the highest value is the end-tidal CO_2 ($ETCO_2$). The downward slope occurs during inspiration and the inspiratory baseline should be zero (**Figs 3** and **10**).

Significant correlation between $ETCO_2$ and $PaCO_2$ has been recorded in anaesthetised horses (Meyer and Short 1985; Cribb 1988b; Moens 1989; Geiser and Rohrbach 1992), with a stronger correlation identified during halothane compared with isoflurane anaesthesia (Meyer and Short 1985; Cribb 1988b). The $PaCO_2$ is higher than the $ETCO_2$ as a result of differences between ventilation and perfusion within the lungs. The average difference in healthy mature horses is 1.6 kPa (12 mmHg), range 0–4.3 kPa (0–32 mmHg), during **halothane** (Cribb 1988b; Moens 1989) and 1.9 kPa (14 mmHg) during **isoflurane** anaesthesia (Cribb 1988b). No significant increase in $PaCO_2$-$ETCO_2$ difference was recorded with increased duration of anaesthesia (Cribb 1988b; Moens 1989).

In one study of 110 horses, the $PaCO_2$-$ETCO_2$ difference was **greater in heavier horses** and was increased when horses were in dorsal compared with lateral recumbency (Moens 1989). A mean $PaCO_2$-$ETCO_2$ difference of 1.8 ± 0.9 kPa (13.4 ± 6.9 mmHg; range 0–37.5 mmHg) was measured in 125 horses anaesthetised with isoflurane in dorsal recumbency for colic surgery (**Table 4**). Foals anaesthetised with isoflurane and breathing spontaneously had a mean $PaCO_2$-$ETCO_2$ difference in the first hour of anaesthesia of 0.9 kPa (7 mmHg) which increased over 90 mins anaesthesia to 1.7 kPa (13 mmHg), coincident with an increase in $PaCO_2$ (Geiser and Rohrbach 1992). The authors were unable to predict $PaCO_2$ accurately from $ETCO_2$ and **emphasised the limitations of capnometry in spontaneously breathing anaesthetised foals** (Geiser and Rohrbach 1992).

$ETCO_2$ values exceeding 6.7 kPa (50 mmHg) represent increased $PaCO_2$. However, because of the large alveolar to arterial difference that exists in some horses, when $ETCO_2$ is normal $PaCO_2$ may be normal or increased; and when $ETCO_2$ is low, $PaCO_2$ may be low, normal or increased. Therefore, at least one direct measurement of $PaCO_2$ is advisable when the $ETCO_2$ value is normal or low, particularly in horses anaesthetised for colic surgery or in foals.

Changes in $ETCO_2$ or waveform are useful indicators of significant alteration in physiological status or equipment malfunction (**Table 5**). Bumps and dips in the expiratory plateau may be caused by spontaneous respiratory efforts, heartbeats and movements of the surgeon.

Failure of the capnograph to return to zero during inspiration indicates rebreathing of CO_2. This may be caused by:

- Excessive apparatus deadspace (endotracheal tube extending out of the horse's mouth).
- Exhausted soda lime.
- Expiratory valve of the circle stuck in an open position.

A slanted or prolonged inspiratory downslope or expiratory upslope may be caused by:

- Slow inspiratory time.
- Lung disease.
- Obstruction or crack in the sampling line.
- Leak around the connection to the endotracheal tube or circle circuit.
- Excessively slow sampling of gases.

A water trap is attached to the sampling tubing to condense moisture. Water entering the monitor will cause unpredictable values.

Pulse oximetry

Pulse oximetry is a noninvasive method of continuously measuring haemoglobin O_2 saturation (SpO_2). The sensor consists of light-emitting diodes (LEDs) that emit light in the red (660 nm) and infrared (940 nm) wavelengths and a photodetector that measures the amount of light that has been transmitted through tissues (Tremper and Barker 1990). **The principles of measurement are based on the different light absorption spectra of oxyhaemoglobin and reduced haemoglobin, and the detection of a pulsatile signal.**

Pulse oximeters display a digital record of pulse rate, with an audible beep, and some monitors display the O_2 saturation waveform. **A limit for acceptable saturation can be entered into the monitor, allowing an alarm to sound when lower values are sensed.** The **pulse rate** displayed on the oximeter must correspond to the rate obtained by palpation or ECG before the measurement can be assumed to be accurate. The shape of the sensor, thickness of tissue placed within the sensor, presence of pigment and hair and movement of the patient can be responsible for the oximeter failing to measure O_2 saturation.

Different monitors, types of sensors and alternative sites for measurement have been evaluated in horses (Whitehair *et al.* 1990; Chaffin *et al.* 1996). The **Ohmeda Biox 3700** and **Physio-Control Lifestat 1600 pulse oximeters** were evaluated in mature horses using the human earlobe probe (Whitehair *et al.* 1990). Measurements were obtained from the **tongue** and the **ear**, with the most accurate measurements obtained from the tongue; the oximeters failed to detect a pulse at the nostril, lip or vulva.

The results revealed that both oximeters tended to underestimate saturation by 3.7% with 95% of O_2 saturation values within 1% above or 8% below SaO_2 (Whitehair *et al.* 1990). The **Nellcor N-200 pulse oximeter** was evaluated in anaesthetised foals using a fingertip probe (Durasensor DS-100A) (Chaffin *et al.* 1996). Attachment of the probe to the tongue or ear of foals slightly underestimated SaO_2 within the range of SaO_2 80–100%.

Reflectance pulse oximeters detect changes in absorption of light reflected from tissues, rather than transmitted through tissues as just described (Watney *et al.* 1993; Chaffin *et al.* 1996). Attachment of a reflectance probe designed for the human forehead to the ventral surface of the base of the tail in foals had 100% sensitivity for detecting SaO_2 <90%, but consistently underestimated the actual value (Chaffin *et al.* 1996). Therefore, this probe-site combination will incorrectly identify some foals as being hypoxaemic.

Pulse oximetry is a useful monitor for horses receiving **total i.v. anaesthesia**. Low arterial O_2 saturation is an indication for O_2 supplementation, if it is not supplied already. If the horse is being supplemented with O_2 by nasal insufflation, low arterial O_2 saturation indicates that the trachea should be intubated and 100% O_2 administered via demand valve or an anaesthesia machine.

TABLE 5: Conditions affecting end-tidal CO_2

No CO_2
Oesophageal intubation
Endotracheal tube kinked
Sampling line disconnect from endotracheal tube
Apnoea
Increased $ETCO_2$
Sudden
Circle one-way valve stuck open
Injection sodium bicarbonate
Increase in cardiac output
Gradual
Hypoventilation
Increased metabolism (lighter anaesthesia)
Exhausted soda lime
Decreased $ETCO_2$
Sudden
Decreased blood pressure or cardiac output
Cardiac arrest
Endotracheal tube obstruction or disconnect
Break in sampling line
Gradual
Hyperventilation
Decreased metabolism (deeper anaesthesia)
Decreased pulmonary perfusion

pH and blood gases

Analysis of pH, $PaCO_2$ and PaO_2 can provide useful information about respiratory and cardiovascular function and metabolic status. The results for PO_2 should be **corrected for the difference between the temperature** of the blood gas machine and the **horse's rectal or blood temperature** using the temperature correction factors for human blood (Fedde 1991). For research purposes, calibration of the PO_2 electrode for each horse is also recommended to avoid a 0–8% inaccuracy (Fedde 1991).

Normal values for $PaCO_2$ and PaO_2 have been given earlier in this report. The normal ranges for pH, pCO_2, HCO_3 and base excess are influenced by diet. Although healthy horses usually maintain a mild metabolic alkalosis with pH >7.4, HCO_3 >25 mmol/l and positive base excess, **horses on a high-grain diet or lower-quality hay may have nutritionally induced metabolic acidosis** (Baker *et al.* 1992).

Temperature

In the normal animal **body heat is distributed unevenly,** with the core temperature being 2–4°C higher than the periphery (Sessler 1997). **General anaesthesia inhibits vasoconstriction, allowing generalised redistribution of body heat.** An additional decrease in body temperature occurs as heat is lost to the environment. Furthermore, **anaesthetics inhibit thermoregulation, vasoconstriction** and **shivering, thereby decreasing the thresholds for cold responses** (Sessler 1997). A decrease in temperature of 1–3°C below normal has been demonstrated to provide substantial protection against cerebral ischaemia and hypoxaemia in anaesthetised dogs (Wass *et al.* 1995). However, **perioperative hypothermia is associated with:**

- Decreased resistance to infection.
- Increased incidence of surgical-wound infection.
- Increased post operative protein catabolism (Carli *et al.* 1991; Sheffield *et al.* 1994; Kurz *et al.* 1996).
- Weakness and ataxia during recovery from anaesthesia.

Hypothermia (temperature reduced to 35.5°C [96°F]), may develop in foals and in mature horses anaesthetised in a cool environment. **Administration of unwarmed i.v. fluid contributes substantially to the decrease in body temperature** (Sessler 1997). Rectal temperature should be monitored at 15 min intervals during inhalation anaesthesia. Active skin warming of the limbs may be the most effective method of preventing heat loss (Cabell *et al.* 1997).

Blood glucose

Foals may develop hypoglycaemia during or after anaesthesia with an inhalation agent (Adams and Trim 1990). Although xylazine causes hyperglycaemia in mature horses, this effect is not observed in foals age <1 month (Robertson *et al.* 1990). Clinical signs of hypoglycaemia may not be obvious during anaesthesia. **Hypoglycaemia may contribute** to hypotension and later cause a prolonged recovery from anaesthesia, depression and weakness. **Routine monitoring** should include measurement of blood glucose in foals at approximately 1 h intervals during anaesthesia. Blood glucose can be determined rapidly using reagent strips and a glucometer. Administration of 5% dextrose in water can be infused as part of intraoperative fluid therapy at a rate of 2–5 ml/kg bwt/h to maintain blood glucose between 5.5 and 11.0 mmol/l (1 and 2 g/l).

Manufacturers' addresses

[1]Criticare Systems Inc., Waukesha, Wisconsin, USA.
[2]Park Electronics, Aloha, Oregon, USA.
[3]CIBA Corning, Medfield, Massachusetts, USA.

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