ANAESTHESIA

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HISTORY OF ANAESTHESIA

The search for relief of pain would seem to be as long as the history of man. There is evidence of the use of various substances to relieve pain or cause sleep throughout recorded history. The earliest records of the use of natural substances such as *opium* and *alcohol* to relieve pain date back to thousands of years BC. In the 800's, the use of a *'soporific sponge'* is described. (1) This was a sponge soaked in a mixture of substances eg *opium, hyoscyamus, mulberry juice, hemlock, madragora and ivy.* The sponge was applied to the nose of the patient prior to surgery. With the ability to extract and identify the compounds in these plants, we can now provide justification for the inclusion of most of these substances in such recipes.

- Opium from the sap of the ripe poppy capsule contains many alkaloids, some of which act on the central nervous system to cause *analgesia.* These include *morphine*, codeine, thebaine, papavertum
- Alcohol induces *stupor*, *insensitivity to pain*, *sleep*
- Mandragora = *Solanacea* Contains substances with emetic, purgative, *narcotic* properties
- Henbane - toxic to hens ∴the bane of hens. Contains alkaloids hyoscyamine and atropine. Hyoscyamine has *sedative* effects
- Hemp = *Cannibis sativa*. Contains a narcotic resin which is *stupefying*
- Hyoscyamus contains hyoscine (*sedative*)
- Hemlock = *Conium maculatum* contains coniine, a respiratory depressant, astringent. Also acts on the spinal cord → convulsions. Reason for inclusion????

It is interesting to note that morphine, which is extracted from opium, is still the mainstay of treatment of severe pain in people today.

There were some surprisingly early discoveries by scientists and physicians whilst experimenting. *Ether*, named 'sweet vitriol', was discovered in the 13th century but was not used as an anaesthetic until 1846 (Jackson and Morton in the USA and Liston in England). Vesalius, in 1542, gave a rather gruesome account of the performance of a *tracheostomy* in a pig to allow *artificial ventilation*. This procedure was adopted as a therapeutic measure in cases of asphyxia and drowning in the latter half of the 17th century, 'often with dire results'. Sir Christopher Wren first experimented with *intra-venous injections* in 1656, and James Elsholtz gave the first *IV anaesthetic* in 1665 using opiates. (Keys 1945)

In 1771, there were two independent discoveries of *oxygen* by Joseph Priestly in England and Carl Wilhelm Scheele of Sweden. Priestly discovered *nitrous oxide* in 1772. It was introduced into medical practice in 1799 by Sir Humphrey Davies. In 1806, Friedrich Serturner extracted *morphine* from opium. In 1831, there were three independent discoveries of chloroform, Guthrie (USA), Soubieran (France) and Liebig (Germany). (Keys 1945)

The introduction of anaesthesia into human surgery was hard won. In the 1840's, there were demonstrations of painful procedures carried out under the influence of nitrous oxide and ether on both sides of the Atlantic Ocean. Successful demonstrations were, at best, regarded with suspicion, failures ridiculed. The reluctance to accept the need for anaesthesia may have been in part due to the fact that the popularity of a surgeon depended on the speed with which he performed his 'craft'. Anaesthesia would eventually lead to a change in expectations placed on surgeons. Also, there was a perception that God had bestowed us with pain and suffering, and that the pious would face it with fortitude. This particularly applied to the exclusively male surgeons' attitude to childbirth. (Youngson 1979)

Coincident with the slowly changing knowledge and attitudes to anaesthesia was the equally slow recognition of the need for asepsis in surgery. Many successful surgical operations and assisted labours ended days later with the patient succumbing to infection. Surgeons operated in gowns encrusted with the filth of many operations, and would boast of the length of time one gown would last uncleaned. Sir James Simpson of the Royal Infirmary, Edinburgh, played a pivotal role in both improving the environment of hospital

ward and surgery theatres and the acceptance of anaesthesia for obstetric procedures. (Youngson 1979)

Possibly the single most significant factor in the eventual acceptance of anaesthesia for obstetric procedures was the successful administration by John Snow of chloroform to HM Queen Victoria for the birth of Prince Leopold in 1853. From this time onwards, the administration of ether, chloroform and nitrous oxide, in conjunction with the recognition of the role of asepsis in surgery and obstetrics allowed a revolution in surgery. (Youngson 1979)

In 1847, the first chloroform anaesthetic was performed in animals by Flourens in France. M and D Shepherd, at the same time as JG Wright, introduced thiopentone as an anaesthetic in veterinary practice in 1937. Dr LW Hall of Cambridge first used halothane in veterinary practice in 1956. Thiopentone is still commonly used for induction of anaesthesia in cats and dogs. Halothane is still in use in veterinary anaesthesia, although it has largely been replaced by isoflurane.

It is usual for veterinary anaesthesia to follow trends in human anaesthesia, albeit at a little distance. The initial cost of new agents and equipment can delay acceptance by veterinarians.

CLASSIFICATIONS AND DEFINITIONS

There are many reasons for administering anaesthetics to veterinary patients. Commonly, we view these from three different perspectives.

- *Patient*   The patient needs to be *asleep* and to be free from pain. As pain is a conscious perception, what we really mean by *free from pain* is that the patient must be *free from the adverse effects of noxious stimuli (antinociception)*. Untreated pain can result in the patient entering a pathological, catabolic state, which results in weight loss, delayed healing and even death. This was first recognised by Hans Seyle in 1936.
- *Surgeon*   The surgeon needs the patient to be *still* and the muscles to be *relaxed* so that the body can be manipulated easily.
- *Anaesthetist* The anaesthetist needs an anaesthetic agent that *is easy to deliver*, *reliable* and rapidly *reversible*.

Simply, the *ideal anaesthetic agent* will
   a)   cause sleep, antinociception and muscle relaxation
   b)   have no adverse side effects, particularly depression of the cardiovascular and respiratory systems
   c)   be totally and rapidly reversible
   d)   not cause allergic reactions
   e)   be easy to use (water soluble, small volume, no pain on injection etc)

Definitions
Analgesia is absence or relief of pain.
Anaesthesia is the absence of all sensory modalities.
Pain is "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage. Pain is always subjective" (Anon, The International Association for the Study of Pain). Pain is a conscious perception and therefore cannot be measured objectively.
Nociception is the reception, conduction and central processing of nerve signals generated by the stimulation of *nociceptors*. In the conscious patient, perception of pain is dependent on this process.
A noxious stimulus is one that is actually or potentially damaging to the body tissue. An anaesthetized patient can be adversely affected by a noxious stimulus without perceiving pain.

Components of anaesthesia

Anaesthesia is considered to be the combination of

1.   Sleep
2.   Analgesia (or, more correctly, anti-nociception) and
3.   Muscle Relaxation.

The term s*aturation anaesthesia* refers to attempting to provide sleep, analgesia and muscle relaxation with deep anaesthesia using a single anaesthetic agent. By its very nature, this process is attended by pronounced side effects of the agent used. For example, if thiopentone is the agent used, very deep anaesthesia will result in severe respiratory depression, as well as cardiovascular depression. Saturation anaesthesia is rarely the method of choice.

More common is the use of multiple drugs each chosen specifically to provide one or more of sleep, analgesia and muscle relaxation. This is termed ***balanced anaesthesia***. For example, the use of a hypnotic, a narcotic analgesic and a skeletal muscle relaxant. As smaller doses of each agent can be used, the overall side effects are usually less detrimental to the patient than with saturation anaesthesia.

Anaesthesia can be considered as representing four time periods: -

1)   Premedication
2)   Induction
3)   Maintenance
4)   Recovery

Premedication. This period is the time during which the anaesthetic regimen is planned, and must begin with a *complete clinical examination* and careful consideration of the patient's *breed*, *history* and *disease status*. Clinical *laboratory testing* and *imaging* may also be indicated. Particular attention must be paid to the *cardiovascular and respiratory systems*. Consideration must also be given to the *nature* and *duration* of the procedure to be undertaken. For example, if a healthy dog is to undergo radiography of a limb that is not painful, the analgesia component of the anaesthetic will not be as critical as, say, the muscle relaxation.

Drugs from the following groups are often used as premedicants: -

- Anxiolytics
- Sedatives
- Dissociatives

- Analgesics
- Anticholinergics
- Antibiotics

Premedicants are usually administered by subcutaneous, intramuscular or intravenous injection. It is important that the animal is in a quiet environment for sufficient time to allow the drugs to take effect (eg 20 minutes). Ideally, once the premedicant drugs have taken effect, the patient will be relaxed, possibly sleeping (but able to be roused) and pain free.

Induction

Induction refers to the time when the conscious patient enters a state of hypnosis or sleep from which it cannot spontaneously wake *ie loss of consciousness*.

Induction drugs are usually delivered by intravenous injection but can be delivered by intramuscular injection or inhalation.

Classes of drugs include: -

- Barbiturates (eg thiopentone, IV)
- Phenol derivative (eg propofol)
- Neuro-active steroids (eg alphaxalone, IV)
- Dissociative agents (eg ketamine, IV or IM)
- Opioids (eg fentanyl, IV)
- Halogenated hydrocarbons (eg halothane, inhaled)
- Halogenated ethers   (eg isoflurane, sevoflurane, inhaled)

Drugs can be delivered at a predetermined dose rate as a *bolus dose*. For example, a dose of ketamine may be calculated as a mg/kg dose rate and the whole dose injected intramuscularly. Alternatively, the drug can be delivered slowly, to effect until the desired depth of anaesthesia is reached *ie the amount of drug delivered is titrated against the effect of the drug*.

Consider the effect of 15 mg/kg thiopentone administered as a bolus dose to each of a large number of dogs. The depth of anaesthesia achieved will vary from dog to dog. It is likely that if the depth of anaesthesia of dogs could be measured, the distribution of effect relative to dog numbers would look like this: -

Still awake   ⇦   Effect   ⇨   Dead
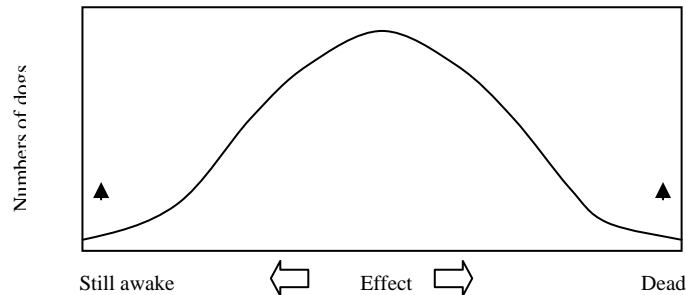
This means that some dogs would be awake and some dogs would be very deeply anaesthetized, even dead. Therefore, it would seem *desirable to administer induction agents by titration* where possible. Titration allows for the individuality of the patient and allows you to administer the appropriate amount of drug for each patient.

Maintenance.

Maintenance is the period of time when the procedure for which the patient has been anaesthetized is carried out. This means that the patient's *depth of anaesthesia must be monitored* carefully and the appropriate amount of anaesthetic agent delivered. As with induction, the delivery of drugs can be by repeated bolus doses, or by titration of dose to effect (eg variable rate of IV infusions, variable concentration of inhaled agent).

Drug groups commonly used are: -
- Steroid anaesthetic agents (eg alphaxalone, IV)
- Phenolic agent (propofol)
- Dissociative agents (eg ketamine, IV or IM)
- Opioids (eg fentanyl, IV, morphine, IV, IM, SC)
- Halogenated hydrocarbons (eg halothane, inhaled)
- Halogenated ethers   (eg isoflurane, inhaled)
- Muscle paralysing agents* (eg vecuronium, IV)

*It must be remembered that if a *skeletal muscle paralysing drug* is used, the muscles of respiration are paralysed and the patient must be *artificially ventilated* until the effect of the muscle paralysing drug has worn off and the patient is again able to spontaneously ventilate. These drugs can be administered to anaesthetized patients only.

Recovery

Following cessation of administration of anaesthetic drugs, patients recovering from anaesthesia require close supervision and may require further drug administration. For example: -
- Sedatives, Anxiolytics (eg xylazine in a horse showing excitement during recovery)
- Analgesics (eg morphine)
- Reversal agents (eg naloxone – opioids, atipamazole - medetomidine)
- Antibiotics
- Non-steroidal anti-inflammatory drugs

DRUG ADMINISTRATION

Choice of needle for injection. Within reason, the smaller the needle the better! Always ask yourself 'what size needle would I like stuck in me?' The less you hurt your patient, the less it will want to hurt you.

However, the choice of needle will be influenced by several factors. For example, the following situations require a relatively large bore needle: -

- Injection of a viscous substance
- Withdrawal of a blood sample
- Rapid injection of large volumes

Here are some suggestions of appropriate needle and catheter sizes.

Species	Route	Needle size	Catheter size
Cat	IV	26 G, ½" (13 mm)	22 G
	IM, SC	26 G, ½" (13 mm)	
Dog	IV	23 G, ¾" (19 mm) 26 G, ½" (13 mm)	22 – 18 G
	IM,SC	23 G, ¾" (19 mm) 26 G, ½" (13 mm)	
Horse & Cow	IV	18 G, 1½" (38 mm) 21 G 1½" (38 mm)	14 – 10 G, 4" (100mm)
	IM	18 G, 1½" (38 mm) 21 G 1½" (38 mm)	
	SC (eg small volume local anaesthetic bleb in the skin)	26 G, ½" (13 mm)	

Routes of administration

1. Subcutaneous injections. (SC)
The subcutaneous route for injection is very useful in *species with loose, mobile skin*, eg cats and dogs. The *absorption rate* for most drugs is about the same for SC as IM. It is desirable from the perspective of both the patient and the handler that the injection is painless if possible. Therefore, choice of *site for SC injection is critical*. The *skin on the back of the neck* in cats and dogs is the best site for SC injection as most will show little or no signs of discomfort from injection here.

2. Intramuscular injections. (IM)
Intramuscular injections can be painful in cats and dogs. (Often, the same result can be achieved with a SC injection) In large animals, premedication/sedative injections are usually given IM.
In *cats and dogs*, IM injections can be given in the quadriceps muscle group ie *cranial to the femur* or in the neck muscles (which allows good restraint). In *horses*, try the muscles of the *rump* or the *neck*. In *cattle*, the rump is best avoided as this is a valuable cut of meat. The neck muscles are commonly used.

3. Intravenous injections. (IV)
Dogs and cats.The *cephalic vein*, on the cranial aspect of the forelimb, between the elbow and the carpus is the most common site for IV injection. Alternatively, the *jugular veins, saphenous veins* (on the lateral aspect, cranial to the hock joint) and the *dorsal common digital veins* (running along the dorsum of the hind paws) can be used. In the anaesthetized patient, the veins on the underside of the tongue are very useful.
Sheep & goats   The cephalic and jugular veins
Pigs.   The *ear veins* are most commonly used for administration of anaesthetic agents.
Cattle.   The *jugular veins* and *tail vein* are easily accessible.
Horses   Drugs are usually administered via the *jugular veins*, and occasionally via the *cephalic* veins.

4. Drug administration by inhalation.
The delivery of *volatile* (eg isoflurane) or *gaseous* (eg nitrous oxide) anaesthetic agents via the patient's pulmonary ventilation is common in veterinary anaesthesia. This necessitates

the control of inhaled gases (a mixture of *carrier gases* (eg oxygen) and *anaesthetic agent*) which can be achieved by: -

- Placing the animal in an *enclosed c*hamber filled with carrier gas/anaesthetic mixture (eg rats, mice)
- Enclosing the patients nose and mouth in a *mask* through which the carrier gas/anaesthetic mixture is delivered
- Inserting *into the already anaesthetized animals' trachea a tube connected to a breathing system* containing the carrier gas/anaesthetic mixture

5. Epidural

The first epidural anaesthetic was performed in 1885 by LJ Corning in the USA who injected cocaine into the epidural space of a dog, thereby rendering the hind limbs insensitive to pain. The injection of *local anaesthetic* into the *epidural space* blocks nerves as they leave the spinal canal, thus *anaesthetising the area supplied by the particular nerves blocked*. In large animals, epidural anaesthesia in a sedated animal can eliminate the need for general anaesthesia for many procedures. In small animals, epidural anaesthesia is often used as an adjunct to light general anaesthesia. Analgesia can be obtained by epidural injection of *opioids, local anaesthetics or α_2-adrenergic agonist drugs (in ruminants)*.

ANAESTHESIA and the RESPIRATORY SYSTEM

BASIC CONCEPTS and DEFINITIONS: -

Respiratory Minute Volume (RMV) = Respiratory Rate (RR) × Tidal Volume (TV) *(l/min)* The respiratory minute volume (RMV) is the volume of gas that ventilates the whole of the respiratory tree each minute.

Alveolar Ventilation (AV) = RMV – Dead space ventilation *(l/min)* The alveolar ventilation (AV) is the volume of gas that ventilates that part of the respiratory tree that is associated with gas exchange (ie the alveoli) each minute.

Metabolic oxygen requirement of the patient (*oxygen uptake*) is the volume of oxygen used in cellular respiration per minute.   *(mls/min)*

Estimates of RMV, AV and O_2 uptake can be made using formulae based on the relationship of weight to metabolic rate. (Metabolic rate $\propto$ body surface area, Body surface area $\approx$ Wt $(kg)^{3/4}$) *Metabolic rate* and *body mass* determine oxygen requirements and therefore can be used as a basis for estimating RMV and AV.

$$O_2 \text{ uptake} \approx WT^{3/4} \times 10 \text{ ml/min}$$
$$\text{Alveolar Ventilation} \approx WT^{3/4} \times 0.16 \text{ l/min}$$
$$\text{Respiratory Minute Volume} \approx WT^{3/4} \times 0.25 \text{ l/min}$$

See Appendix 2 for a more accurate method of estimation of AV and RMV.

Revise the concepts displayed in Figure 1 below, especially inspiratory and expiratory reserve volumes. Inspiratory reserve can be decreased by such factors as abdominal contents pressing on the diaphragm (eg pregnancy), space occupying lung lesions etc.
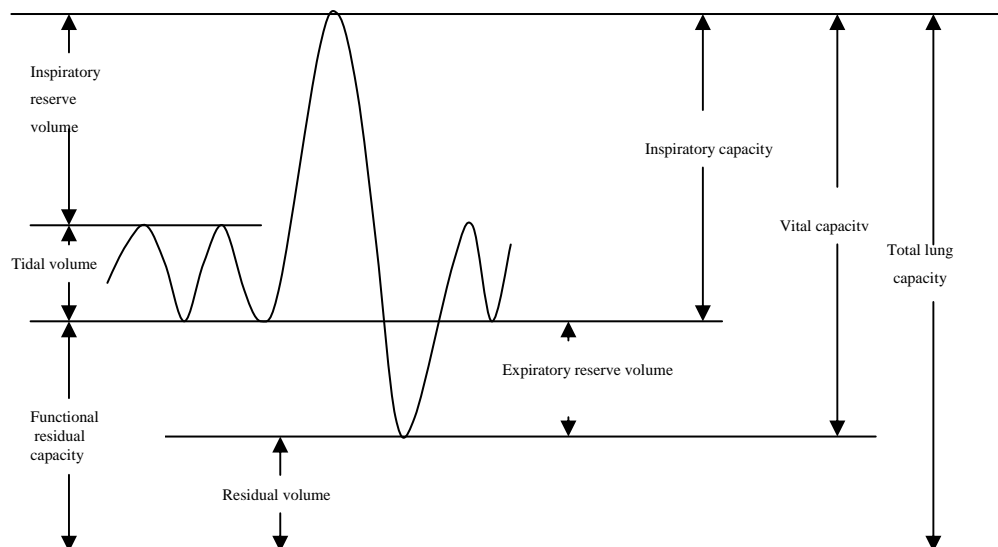
Fig.1   Static lung Volumes

PHYSIOLOGY, FUNCTIONAL ANATOMY AND CHANGES WITH ANAESTHESIA: CLINICAL IMPLICATIONS.

Overall, the functions of the respiratory system are to oxygenate haemoglobin and remove CO_2 from the blood so that, provided perfusion of the peripheral tissues is adequate, respiration at the cellular level is supported.

An understanding of the functional anatomy and physiology of the patient's respiratory system is essential for the safe practice of anaesthesia. There are many changes in the respiratory system brought about by anaesthesia that may require active management by the anaesthetist. Anaesthetized patients frequently have a tube inserted into the trachea (endotracheal or ET tube) connected to a breathing system, which may also be an anaesthetic delivery system. The anaesthetist must ensure that the system satisfies the breathing requirements of the patient and that changes due to the anaesthetic agents are accounted for.

The following is in the form of some revision of respiratory anatomy and physiology, introduction of some changes that may be brought about by anaesthesia and the clinical implications for you as the anaesthetist.

Function of the nose.

As the air passes through the nose, it is *warmed*, *humidified* and *filtered* of particulate matter. It then passes through the larynx into the trachea to the bifurcation of the bronchi.

Clinical implications: -

•	If an *endotracheal tube* is inserted, the 'air-conditioning' function of the upper airways is lost. It may be necessary to *warm, humidify and filter gases* delivered by an endotracheal tube. It is important that only medical quality gases are delivered to the patient via the endotracheal tube. The breathing of cold dry bottled gases can contribute to *dehydration* and *hypothermia* in anaesthetized patients. The anaesthetist can avoid problems by warming and humidifying the inhaled gas before it enters the patient's airways. Alternatively, the patients may need to be warmed and rehydrated in the perioperative period.

Sigh reflex

Further branching leads on to bronchi, bronchioles, then to the alveoli and the functional interface where *gas exchange* occurs between the alveoli and the blood. Not all alveoli are functional at any one time. Some alveoli will have collapsed due to surface tension. Some alveoli, particularly those in the upper segments of the lungs will be underperfused relative to ventilation and some in the dependent parts of the lungs will be overperfused relative to ventilation. This is due to gravity and is called *ventilation-perfusion mismatching.* The *sigh reflex* is necessary for the reinflation of collapsed alveoli. (ie normal, awake animals will periodically unconsciously sigh). The sigh is a response to rising carbon dioxide levels in the blood.

Clinical implications: -

•	Anaesthetized patients usually lose the 'sigh' reflex and are at risk *of increasing alveolar collapse* with time. It is advisable to give the patient a large breath periodically by squeezing the reservoir bag on the breathing system to which the patient is connected.

•	Large animals, particularly horses, have a large degree of ventilation-perfusion mismatching. This is a particular problem in horses because of the large size of the lungs. It is important to deliver an adequate concentration of oxygen (eg 30%) and to have the capacity to assist ventilation.

Dead space

The volume at inspiration of the air occupying the space in the nose, pharynx, larynx, trachea, bronchi and bronchioles which does not undergo gas exchange, is known as the *anatomical dead space.* The volume of gas in alveoli that are not perfused is known as *alveolar dead space.* Together, the anatomical dead space and the alveolar dead space make up the *physiological dead space*. Gas within this space does not take part in gas exchange. When the patient is connected to a breathing system, care must be taken that the *apparatus itself does not contribute significantly to the dead space*. Eg if a swimmer uses a snorkel, the volume of the snorkel has to be added to the tidal volume if ventilation is to be maintained adequately. The snorkel contributes dead space called the *apparatus dead space*. Similarly, the breathing system of an anaesthetic machine can contribute *apparatus dead space*. This means that the patient must breathe more deeply and/or more rapidly to achieve the same alveolar ventilation as it had before being connected to the machine.

Clinical implications: -

•	Care must be taken to ensure that *intubation of the trachea* and connection of a patient to *a breathing system* do not significantly *increase the dead space*.

•	If an *anaesthetized patient* (likely to have *depressed ventilation caused by the anaesthetic drugs*) is connected to a breathing system that *has significant apparatus dead space*, *hypoventilation and rebreathing of expired gases* is likely to result.
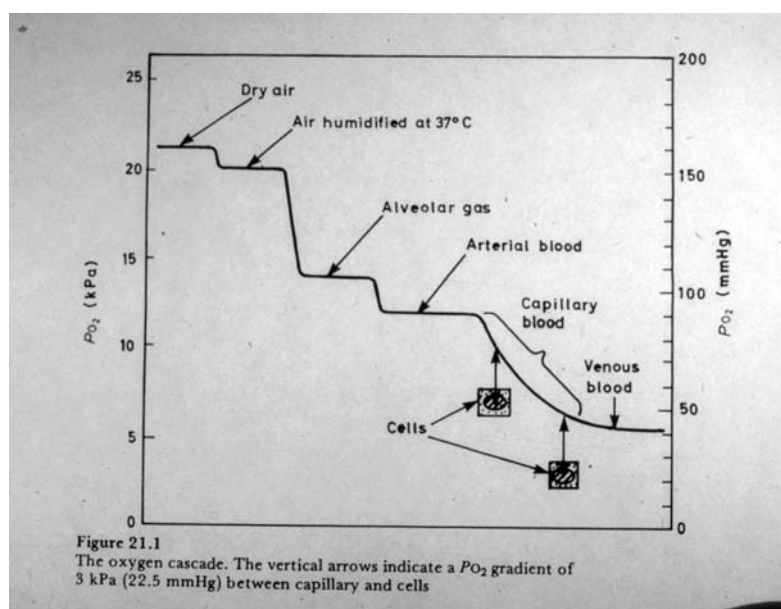
- Remember that it is Alveolar Ventilation that results in gas exchange. If the Respiratory Minute Volume decreases due to the anaesthetic agents used or positioning of the patient, the dead space component of the RMV will be unchanged – all the decrease will be due to a fall in Alveolar Ventilation.

Oxygen cascade.

Air is approximately 21% oxygen, 79% nitrogen with traces of CO_2 and other gases. *After gas exchange with the blood has taken place*, the alveoli will contain gas, which has equilibrated with the pulmonary capillary blood. This gas will be about 5 - 6 % O_2 and 4 % CO_2. When the animal expires, the last gas to leave the alveoli will not all be expelled to the atmosphere, but some will be left in the dead space. Inspired fresh gas will mix with dead space gas so that the gas in the alveoli after the next breath will contain only about 14% O_2. The lowering of O_2 concentration with mixing as gases are drawn into the respiratory tree is part of the *oxygen cascade*. The cascade proceeds further until the eventual delivery of oxygen to the tissues at a concentration of about 13% at the arterial end, with 5% at the venous end of the capillaries after gas exchange has taken place.

Clinical implications: -

- *Many anaesthetic agents cause depression of pulmonary ventilation by causing a reduced rate or tidal volume or both*. Gas exchange in the alveoli normally occurs at 13 – 14 % O_2. Depression of ventilation is likely to decrease the alveolar O_2 to a level where the oxygenation of the blood is inadequate. Therefore, once the patient is anaesthetised, *the O_2 concentration of the fresh gas delivered to the patient should contain a higher O_2 concentration, say 30%*, to ensure adequate alveolar O_2 concentration.

- Depression of ventilation will also cause an increase in alveolar CO_2 with resultant increase in blood CO_2 and lowering of pH. *The anaesthetist may need to support the ventilation during anaesthesia to prevent hypercapnia*. High blood CO_2 causes: -
  - o Vasodilation and therefore hypotension
  - o Low blood pH and consequent electrolyte changes
  - o Moderate hypercapnia is a respiratory stimulant in the conscious patient, but at high CO_2 levels, hypercapnia depresses respiration and acts as an anaesthetic in its own right.

Figure 21.1
The oxygen cascade. The vertical arrows indicate a Po_2 gradient of 3 kPa (22.5 mmHg) between capillary and cells

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Diagram from Gray, TC, Nunn, JF and JE Utting (1985) *"General Anaesthesia"* Fourth Edition, Butterworths

Airway pressures

The interpleural space is really only a potential space as a slight negative pressure holds the lungs in expansion against the parietal pleura. *The integrity of the chest wall and of the diaphragm are necessary to maintain lung expansion*. When the respiratory system is at rest, the pressure in the airways is equal to atmospheric pressure. At *inspiration*, *a larger negative pressure* is created in the pleural space by expansion of the chest wall and pulling down of the diaphragm. As the lungs expand, a *slight negative pressure is created in the airways*, and air flows in. This increasing negative pressure in the thorax also has the effect of *increasing blood flow into the thorax thereby enhancing cardiac output*.

Clinical implications: -

•   *Loss of integrity of the chest wall* (eg a penetrating chest wound) leads to *lung collapse*. The anaesthetist will need to ventilate the patient by intermittently applying *positive pressure* to the airways, thereby inflating the lungs with air under positive pressure (IPPV).

•   *Intermittent positive pressure ventilation* (IPPV) leads to a reduction of pulmonary perfusion, reduction of blood returning to the heart via the vena cavae and therefore *reduced cardiac output*. It is important to ventilate with enough pressure to adequately inflate the alveoli, but not enough to reduce venous return *(12 – 20 cm water)*

Voluntary positioning

Once a patient is anaesthetized, it can no longer voluntarily position itself. Cattle and horses rarely lie in lateral recumbency for extended periods. Once a cow or a horse lies down, the abdominal contents fall forward onto the diaphragm and severely reduce ventilation. This is not just loss of reserve, but a real reduction in tidal volume. The animal will progressively become hypoxic and hypercapnic. Similarly, a cat with a diaphragmatic hernia has severe respiratory compromise and may not voluntarily lie down as this would result in more of the abdominal contents entering the thorax. When the cat is anaesthetized, it can no longer keep itself optimally positioned for lung ventilation.
Clinical implications: -

•   By taking note of species/condition, the anaesthetist may be able *to predict and therefore minimise problems with ventilation due to positioning during anaesthesia*. Eg horses and cows should be anaesthetized for as short a period as possible and may require positive pressure ventilation. A cat with a diaphragmatic hernia should be treated with oxygen prior to induction of anaesthesia, and surgery should begin as soon as the patient is asleep to remove the abdominal contents from the thorax and allow adequate inflation of the lungs.

Resistance to airflow.

The positive and negative airway pressures generated during expiration and inspiration are very small. If the resistance of the airway or the breathing apparatus is increased, greater pressures must be generated to cause airflow and the work of breathing is increased. Whilst the patient may be able to respond to the extra demand, respiratory depression due to anaesthetic agents may make this unlikely. Increased resistance to airflow may result in eventual respiratory muscle fatigue and respiratory failure. Also, fluid is drawn into the pulmonary tissues from the micro-circulation causing pulmonary oedema, which reduces gas exchange across the alveolus.

Clinical implications: -

The anaesthetist must ensure that the breathing apparatus and endotracheal tube do not significantly increase resistance to airflow. Use the biggest endotracheal tube that will comfortably fit the trachea. This is especially important in small animals, as the increase in resistance to airflow through a tube is inversely proportional to the radius to the fourth power. A 3 mm tube will offer about three times the resistance of a 4 mm tube. This means that if a 3 mm tube is inserted instead of a 4 mm tube, the resistance to airflow will be more than 3 times as high ie for the same rate of airflow, three times as much work must be done to overcome resistance. Consideration must be given to anaesthetic apparatus resistance

to airflow. Changes in diameter, right-angle bends, branches and high flow rate relative to diameter all cause turbulent flow and therefore, increased resistance to airflow eg large animal anaesthetic machines must have wide bore tubing (5 cm) to accommodate large gas flows
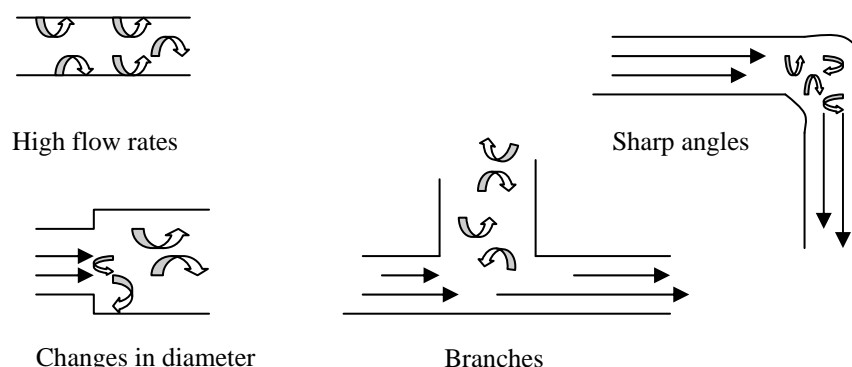

High flow rates

Sharp angles

Changes in diameter

Branches

Fig.2   Causes of turbulent flow

CONTROL OF VENTILATION.

In the normal, non-anaesthetized animal, pulmonary ventilation is precisely controlled so that blood is adequately oxygenated and CO_2 is eliminated in spite of changes in metabolic demand. There are central and peripheral mechanisms involved in this control.

The ***central chemoreceptors*** are situated on the ventral surface of the medulla, bathed in cerebro-spinal fluid. CO_2 readily diffuses into the chemoreceptor cells which are exquisitely sensitive to changes in arterial pCO_2. The response is probably due to pH change within the chemoreceptor cells as these receptors also respond to a lowering of blood pH. The central chemoreceptors are not responsive to alterations in arterial pO_2 levels. An increase in arterial pCO_2 or a drop in pH results in an increase in RMV. There are species differences in the magnitude of the response to elevated CO_2/low pH eg horses are slightly less sensitive than cats and dogs, while burrowing and diving animals are far less sensitive. Clinical implications: -

• Horses often hypoventilate or even become apnoeic during anaesthesia. Diving mammals may not breathe at all once anaesthetized. These animals may have to be ventilated manually throughout anaesthesia.

Peripheral chemoreceptors are located in the carotid and aortic bodies. These receptors are activated by low arterial pO_2, but do not play a significant part in maintaining respiratory drive until the pO_2 falls below 60 mm Hg (normal is 80 – 110 mm Hg). It is thought that the carotid body is responsible for 30 – 40% of resting respiratory drive. However, the result of activation of these receptors by hypoxaemia is an extremely powerful stimulus to breathe.

General neural traffic through the **reticular activating system (RAS)** influences the level of activity of the central respiratory control system in the brain stem. This is evidenced by the fact that pulmonary ventilation is reduced and pCO_2 is increased during sleep. Conversely, ventilation will increase as soon as an animal begins to run, before an O_2 debt or pCO_2 elevation occurs.

Clinical implications: -

• Anaesthetists may make use of the link between the RAS and respiratory drive by providing general sensory stimulation to patients to overcome apnoea during anaesthesia. Eg limb flexion.

The **apneustic and pneumotaxic centres** along with **pulmonary and airway receptors** are primarily responsible for adjusting the balance between tidal volume and respiratory rate to maintain alveolar ventilation.

Although anaesthetic drugs are thought to have little effect on the apneustic and pneumotaxic centres and pulmonary and airway receptors, there are species differences in the effects of some drugs on respiration which are difficult to explain. Eg ruminants under anaesthesia have shallow, rapid breathing while in horses, the rate is likely to reduce.
Clinical implications: -

•   The anaesthetist must be aware of the behaviour under general anaesthesia of different species with different drugs.

•   There are important species differences in airway receptors. Eg cats and pigs (and humans) have larynx's and tracheas that are very sensitive to touch, whereas a horse may not even cough if a stomach tube is accidentally passed into the trachea!
Clinical implications: -

•   Airway sensitivity can cause problems at intubation of the trachea. The chance of spasm of the larynx and consequent occlusion of the airways in cats and pigs means that these species require deep anaesthesia relative to other species, desensitisation or paralysis of the larynx prior to attempts to intubate.

The apnoeic threshold is the arterial pCO_2 at which spontaneous ventilation ceases. If the pCO_2 is reduced by 5 – 9 mm Hg from normal, the ventilatory efforts will cease in many animals.

Clinical implications: -

•   The anaesthetist can (usually) 'take over' a patient's ventilation by hyperventilating until the pCO_2 levels have dropped sufficiently.   Conversely, if the anaesthetist inadvertently hyperventilates a patient, the CO_2 levels will have to build up before spontaneous ventilation will resume.

Effect of Anaesthetic and Perianaesthetic Drugs on the Control of Respiration.
Many drugs used in the perianaesthetic period have a dose related effect on the response of the central and peripheral chemoreceptors to CO_2 and O_2 levels. In general, there is a *loss of responsiveness* and a *loss of overt signs* of changes in CO_2 and O_2 levels.

•   Most anaesthetics in current use produce a dose-dependent decrease in response to CO_2. Eg at high concentrations, halothane and isoflurane can cause almost complete abolition of response to CO_2. This results in a fall in alveolar ventilation.

•   The opioids cause a shift of the ventilatory response to carbon dioxide partial pressure curve to the right without change in slope. This means that, while there will be higher CO_2 levels, there will still be a response to further increase in levels.

ANAESTHESIA and the CARDIOVASCULAR SYSTEM

Survival during anaesthesia depends on maintenance of perfusion of the tissues, particularly the brain and the heart, with oxygenated blood. The anaesthetist must understand the structure, function and control of the cardiovascular system to be able to manage the alterations of function brought about by drugs used to anaesthetise the patient. Most of the drugs used as premedicants, analgesics or anaesthetics have some effect on the cardiovascular system.

BASIC CONCEPTS

The following table shows the *differential perfusion of peripheral tissues*.

	Vessel – rich	Muscle	Fat	Vessel - poor
% Body Mass	9	50	19	22
% CO	75	18	5.5	1.5
Perfusion relative to unit mass	122	5.3	4.2	1

Table 1. Percentage body mass and percentage cardiac output received by each of vessel-rich, muscle, fat and vessel poor tissues. (Adapted from Grey, Nunn and Utting, 1985.)

The *vessel rich group* of tissues consists of *brain, heart, kidneys, viscera and endocrine glands*. The *very high metabolic rate* of the vessel rich group means they have very high oxygen demand and are therefore *very susceptible to hypoxic damage*.
The brain alone receives 14% of the total cardiac output. If the brain is deprived of perfusion for three minutes, cell damage is almost certain. The heart receives 4% of the total circulation. The vessel rich group of tissues will be the first to equilibrate with drugs once they are picked up by the blood. There follows redistribution of drugs to the other tissues, in order of their perfusion.
The vessel poor group consists of bone, tendon, ligaments and cartilage. The extremely low metabolic rate means that these tissues are very resistant to damage due to poor perfusion, and they play little part in drug elimination from the circulation.
Blood flow depends on the pressure gradient attained as blood is ejected from the heart, viscosity of the blood and the diameter of the blood vessels, and therefore the resistance to flow.
- Blood flow (Q) is determined by blood pressure (BP) and the viscosity of the blood.
- Blood pressure is determined by cardiac output (CO) & peripheral resistance (PR)
- CO $\propto$ stroke volume (SV) and heart rate (HR)

Many anaesthetics *depress cardiac muscular activity* and/or *depress the heart rate,* thereby reducing cardiac output. Also, many drugs cause *vasodilatation* causing loss of peripheral resistance, and *drop in blood pressure*.
In addition to these changes, there are alterations in the control mechanisms that usually act to maintain the circulation. In an awake, healthy animal, a challenge to the circulation brought about eg by vasodilatation is met by an increase in heart rate and/or stroke volume so that blood pressure is maintained. The ability of an animal to respond to challenges is referred to as *cardiac reserve*. When a patient is anaesthetized, the cardiovascular system responses may be obtunded or even absent.

Control of Heart Rate and Rhythm

The intrinsic rates of contraction of the different parts of the heart vary. (Sino-atrial node > atria > ventricles.) The rate of firing of the sino-atrial node therefore dominates the lower parts of the heart and sets the pace. Contraction of the ventricles causes blood to be ejected from the heart and therefore is responsible for cardiac output. The rate of ventricular contraction is a determinant of blood flow.

The vasomotor centre in the brain stem balances autonomic tone and therefore influences heart rate. It is affected by traffic to and from other areas of the brain. For instance, as the respiratory centre rhythmically sends signals to the inspiratory muscles, the vasomotor centre is stimulated and the heart rate increases. During the expiratory pause, the heart rate declines. These changes result in a *respiratory sinus arrhythmia*. This is normal.

Some anaesthetic and premedicant *drugs affect the rate and rhythm of the heart*.

Clinical implications: -

The anaesthetist may have to *avoid certain drugs* in some cases, or *pre-empt* and *counteract* the expected side-effects. Eg: -

- The α_2-*adrenergic agonists*, particularly *xylazine*, cause *arrhythmogenic* effects including sino-atrial block, atrio-ventricular block, bradycardia, first- and second-degree heart block, atrio-ventricular dissociation and sinus dysrhythmia. What is observed is profound bradydysrhythmia. The cause of this is complex with central, then peripheral α-adrenergic effects interacting, but there is uncoupling of the normal conduction pathways in the heart. These drugs *should not be used in patients with low cardiac reserve* and only in *minimal doses* in healthy patients.
- Some of the *opioids* cause *increased vagal tone* eg fentanyl. The resulting bradycardia can be avoided by prior administration of a *vagolytic* drug eg atropine
- *Sinus arrhythmia* can be pronounced in *brachycephalic animals* eg boxers which *have high vagal tone*. These animals often are bradycardic and have a marked sinus arrhythmia at rest. Administration of acepromazine (which has some vagotonic effect) to these dogs can result in episodes of syncope, as the bradycardia becomes severe enough to result in inadequate perfusion of the brain. *Acepromazine should be avoided in boxers or administered at very low doses*.
- *Rhythm disturbances* can herald major problems during anaesthesia eg asystolic arrest or fibrillation. Administration of low doses of lignocaine, which *has membrane-stabilizing properties*, may avert further problems.

Stroke volume

Stroke volume depends on adequate filling of the ventricles and strength of myocardial contraction. Both venous return (and therefore ventricular filling) and cardiac contractility can be adversely affected during anaesthesia. Eg

- Intermittent positive pressure ventilation as described in Topic 2,
- Drugs that reduce myocardial contractility

Clinical implications: -

- IPPV must be carried out with minimum effective inflation pressures
- Many drugs used in anaesthesia cause a dose dependent decrease in myocardial contractility. These drugs must be used at low doses, and avoided in cases with low cardiac reserve. Eg halothane, thiopentone, xylazine. Drugs that increase the contractility of the heart muscle can be used to counteract the effect of these drugs. Eg dopamine, dobutamine, ephedrine

Vessel tone

Many of the drugs commonly used in anaesthesia cause vasodilatation and therefore decreased peripheral resistance. Remember the relationship: -

$$BP \propto CO \text{ and } PR$$

Thus, if peripheral resistance is decreased, cardiac output must be increased if blood pressure is to be maintained. However, if these drugs also cause a decrease in myocardial contractility or heart rate, the blood pressure will not be maintained.

Clinical implications: -

- The anaesthetist must counteract the effects of administered drugs on blood pressure. Methods include delivery of intravenous fluids, administration of pressor agents and/or positive inotropes. Some drugs are best avoided or used in very low doses in a case with a compromised cardiovascular system.
- Intravenous fluids can be delivered at 2 – 3 times maintenance levels to provide volume support.

If maintenance fluid requirements for a dog are considered to be 70 mls/kg/day, this is approximately 3 mls/kg/hr.

70 mls/kg/day $\approx$ 72 mls/kg/24hrs = 3 mls/kg/hr

It is usually appropriate to choose a fluid administration rate of 6 – 10 mls/kg/hr for an anaesthetized dog. Maintenance fluid rate for a cat is approximately 2 ml/kg/h. Thus, surgery rates of approximately 6 ml/kg/h are appropriate for a cat. For a healthy patient, isotonic electrolyte solutions will suffice.

- *Acepromazine* causes peripheral vasodilatation and therefore a drop in blood pressure. Hypovolaemia following haemorrhage or dehydration would be contraindications for the use of acepromazine (except in very low doses).

Vascular smooth muscle (skin)	α_1	vasoconstriction
Vascular smooth muscle (splanchnic area)	α_1	"
Radial muscle (iris)	α_1	mydriasis
Intestinal smooth muscle	α_1 & β_1	relaxation
Heart	β_1	↑HR and contractility
Bronchial smooth muscle	β_2	bronchodilation
Blood vessels to skeletal muscle	β_2	vasodilatation

Types of adrenoceptors in various tissues (Adapted from Vickers, Morgan and Spencer *Drugs in Anaesthetic Practice*)

From the above table, it can be seen that drugs which are principally α_1 adrenergic agonists (eg phenylephrine) will cause an increase in peripheral resistance and therefore an increase in blood pressure. β_1 adrenergic agonists will have positive inotropic and chronotropic effects.

Pressor agents/positive inotropes.	Receptors
Dopamine	D>β>α
Dobutamine	β_1>β_2>α
Adrenaline	α,β_1,β_2
Noradrenaline	α>β
Ephedrine	α>β_1>β_2
Isoprenaline	β_1>β_2
Phenylephrine	α

Drugs such as ephedrine, dobutamine and dopamine will provide pressor activity as well as positive inotropic and chronotropic effects.

DRUG HANDLING & ADMINISTRATION

Sterile Handling of Drugs

Multidose vials: -   Drugs packaged for veterinary use are often marketed in multi-dose bottles or vials with a rubber stopper through which a needle can be inserted for drug withdrawal.
- Always use a sterile needle and syringe
- Always swab the rubber top with alcoholic disinfectant before inserting the needle
- If the syringe already contains another solution, make sure that none of that solution is drawn into the multidose container. (There may be a partial vacuum in the container due to prior withdrawal of drug.)
- Remember, any contamination means the vial/bottle should be discarded.

Glass ampoules: -   Glass ampoules are meant for single use only. Breaking the top off the ampoule sometimes results in the vial shattering and cuts can result.
- Swab the neck of the ampoule with alcoholic disinfectant before attempting to crack it.
- Wrap a cotton swab around the ampoule to protect your fingers should the glass shatter
- Some vials are marked with a dot. Break away from the dot.
- Use a sterile needle and syringe
- Withdraw the required dose and discard the remaining contents.

Plastic ampoules: - These are more operator friendly than the glass equivalent.
- Swab top of ampoule
- Twist the top off the ampoule, insert sterile needle or syringe to withdraw dose. Plastic ampoules are mostly designed to fit the nozzle of a syringe.
- Discard the remaining contents.

How to Make up Drug Solutions

Drugs are often marketed as sterile dry powders, to be made up as needed. The reason for this is that these drugs are not stable in solution.
Remember: -
 1 g = 1000 mg   1 litre = 1000 ml
1 mg = 1000 μg   1 ml = 1000 μl
Dose rates are usually expressed as mg/kg or μg/kg
To make up a solution of a desired percentage: -
In relation to a solution, percentage is based on the fact that the density of water is 1 g/ml.
ie 1ml of water weighs 1 g.
Therefore, a 1% solution will contain 1g per 100g of water or 1 g/100ml
1g/100 ml = 1000 mg/100 ml = 10 mg/ml.
Example: -   In practice, you will very likely need to make up a solution of thiopentone sodium. The concentration of thiopentone sodium is customarily referred to in percentages, and it is marketed as a sterile, dry powder, 5g in a bottle.
1)   Firstly, consider how to make up a solution of 5%.
A 5% solution is 5 g/100ml. So, using sterile technique, inject 100 ml of sterile Water for Injection into the bottle.
You now have: -   5 g/100 ml = 5000 mg/100 ml = 50 mg/ml = 5%
2)   Secondly, how do you make this into a 2.5% solution? Thiopentone is very alkaline, and therefore very irritant. The higher the concentration, the more irritant the solution. A 5% solution has a pH of about 10 or 11 and is hypertonic. It is usual to use it as a 2.5% solution, which is less irritant than 5%. The bottle with 5g of dry powder will hold only 100ml and you would need to add 200ml. First, make it up to 5%. Then, withdraw 50 ml and replace this in to an empty and sterile Water for Injection bottle. Then add a further 50 ml sterile Water for Injection to each bottle. Now each bottle has 2.5 g in 100 ml ie 2.5%. It is

important to LABEL both bottles with the drug, concentration and date it was made up. (Thiopentone has limited stability once in solution and should be used within 2 weeks.)

To make up a solution to a desired concentration in mg/ml.
Example: -   The antibiotic Kefzol® is one we may use IV prior to surgery.   It is marketed as a sterile dry powder, 1 g in a 10 ml vial, to be made up into a solution of 100mg/ml. Because the volume of powder in this example is large when compared with the volume of water to be added, the addition of less than 10ml will be necessary to bring the final volume to 10 ml. Mostly, instructions needed for making up the solution are on the label of the bottle. In this case, addition of 9.6 ml of sterile Water for Injection will bring the contents to 10 ml, and the final solution will be 100mg/ml.

How to work out doses and volumes of injectable drugs.
Starting with the patient's weight and a dose rate in, say, mg/kg, you must be able to correctly draw up and administer a drug.

Example: -   Your patient is a 20 kg dog.   You require a dose of 0.05 mg/kg of acepromazine and 0.04 mg/kg atropine.

Acepromazine: -   *How many mg do I need*? 20 kg × 0.05 mg/kg = 1mg
        Check the label on the acepromazine for the concentration of drug in the bottle. Say it is 2mg/ml
        Now, ask yourself *'Do I need more or less than a ml? '(LESS)*
        Next, do a simple proportion: -

$$\text{Volume (ml)} = \text{Dose (mg)} \div \text{Concentration (mg/ml)}$$

                If 1ml contains 2 mg, then x ml contains 1 mg.
                        X ml = 1 mg/2 mg x 1 ml
                               = 0.5 ml
Atropine: -   *How many mg do I need?* 20 kg x 0.04 mg/kg = 0.8 mg
        Again, check the bottle label. Say the atropine is 0.6 mg/ml
        Again, ask yourself *'Do I need more or less than a ml?' (MORE)*
                If 1 ml contains 0.6 mg, then x ml contains 0.8 mg.
                        X ml = 0.8/0.6 x 1 ml
                               = 1⅓ ml (≈ 1.3ml)
So your syringe should contain 0.5 ml acepromazine and 1.3 ml atropine, a total volume of 1.8 ml. (These two drugs can safely be mixed in one syringe.)
Here are a few exercises. (Answers at the end of this section.)
1.   30 kg dog is to be administered 0.15 mg/kg morphine SC. The label on the glass ampoule shows that it is a solution of 15 mg/ml. What volume do you require?
2.   The muscle relaxant, Vecuronium, is marketed as a sterile, dry powder, 4 mg in a small glass ampoule, with a 1ml ampoule of sterile water as diluent. The dose rate you wish to use is 0.06 mg/kg. Your patient is an anaesthetized Rottweiler weighing 50 kg. How will you make up the vecuronium solution and what volume will you administer to your dog?
3.   You have to administer 7 mg/kg of thiopentone to a 400 kg horse. As the injection is to be given rapidly, you plan to administer the drug as a 10% solution via a 12G catheter. You have a bottle with 5 g of thiopentone as a sterile dry powder as well as 100 ml sterile Water for Injection. Describe how you will make up the thiopentone to 10%. What volume of this will you administer to the horse?   Why use a catheter instead of a needle?
4.   You are presented with a 1 kg chihuahua for premedication. This is what you plan to administer: -
        • Acepromazine   0.05 mg/kg   (Available as 2 mg/ml)
        • Atropine   0.03 mg/kg   (Available as 0.6 mg/ml)
        • Pethidine   1 mg/kg   (Available as 50 mg/ml)
        These drugs can all be mixed in the same syringe.

What volumes do you require of each of the drugs? Given that the smallest syringe you have access to is a 1 ml syringe, and that it will measure down to 0.05 ml with some accuracy, how can you accurately prepare this injection?

Intravenous injection in the dog.
Most dogs will tolerate an intravenous injection. Commonly, the cephalic vein is used. The vein is located on the cranial surface of the dog's forelimb and the skin over the vein should be clipped and cleaned as if for surgery.
The assistant should stand on the side opposite to the leg to be injected. For example, if the dog's right leg is to be used, the holder stands on the left side, with their left arm under the dog's neck, hand around the right side of the head so that the head can be gently turned away from the anaesthetist. The assistant's right hand extends over the dogs shoulders to reach the right foreleg at the level of the elbow. Starting with the thumb well medial to the angle of the elbow, the vein can be rotated laterally so that it is more prominent and more easily accessible. Keep the other fingers behind the elbow to prevent the dog pulling the leg away from the anaesthetist. Keep the thumb across the vein, occluding the flow of blood, until the anaesthetist is confident that the needle is properly inserted into the vein.
The anaesthetist.   To locate the vein, place one finger where you think the vein lies on the foreleg near the elbow, and, using the other hand, tap the vein distal to this. A fluid wave will be felt under the proximal finger if it is on the vein. Where possible, use *eccentric nozzle syringes for IV injections*. Hold the barrel of the syringe with your fingers on the sides. DO NOT have fingers under the barrel of the syringe or you will be unable to satisfactorily perform the venipuncture. Once the vein has been punctured, a flash of blood will appear in the needle hub. At this point the needle and syringe must be advanced parallel to the skin. (If you have your fingers under the syringe, you can only pass the needle deeper, through the vein and out the other side!!) Insert the needle into the vein all the way to the hub, and draw back to see a convincing flow of blood. Ask the assistant to take off the finger occluding the vein (but keep the hand behind the dogs elbow) while you inject.

Catheter placement
Where continued venous access is required, a catheter should be secured in the vein. A needle taped into the vein will likely damage the vein and not be of use should emergency access to the vein be required. Catheters are soft and pliable and do not have sharp ends.
Reasons for catheterisation of anaesthetized patients: -
♦ Venous access for drug administration (especially in emergencies)
♦ Administration of IV fluid therapy: -
    • To maintain hydration
    • To encourage urine production so that drugs and their metabolites can be excreted
    • To give volume support and therefore blood pressure support
The cephalic vein is commonly used for catheter placement. Location of vein, preparation of site and the assistants hold are as for IV injection and have already been described.
In choosing a catheter, consider both *length and diameter*. Although it is advisable to have a long catheter so that it will be unlikely to become dislodged, it is equally important not to have the tip of the catheter near a joint. If the catheter is too close to the elbow, the catheter may become occluded when the patient flexes the joint. The bigger the diameter, the better, within reason. One of the reasons for catheter placement in an anaesthetized patient is for emergency fluid administration should volume support become necessary. (eg severe haemorrhage or circulatory failure) In such an event, fluid administration must be rapid, and the rate can be limited by the bore of the catheter.
If the catheter is to be connected to fluids via an infusion set, have this ready for connection BEFORE placing the catheter.
Methods of placing and securing catheters are many and varied. You will be exposed to as many methods as there are staff members in the clinic and hospital, and then you will develop your own. Here are some ideas born of supervising many students placing catheters.

♦ The catheter and the stylette are often "stuck" together so free them up before proceeding.

♦ A common cause of failure is "burring" the tip of the catheter as it is pushed through the skin. Catheters are made of fairly fragile teflon or plastic which can buckle or tear as it is forced through the skin. Once it is torn, it cannot be inserted into the vein. Blood will flow back into the stylette, but it will not be possible to advance the catheter. To avoid this problem, take a scalpel blade and using it inverted, make a small 'nick' through the full thickness of the skin at the point of insertion of the catheter.

♦ Once the catheter is in the vein – DO NOT LET IT GO! Hold the catheter and leg firmly together.

♦ You will need to tape in the catheter. First, connect it to a sterile injection site or a fluid administration set.   Then dry EVERYTHING before taping – fingers, fur, catheter, drip line etc. (A tissue is best) Tape will not stick if anything is wet.

♦ Catheters often come apart at the junction of the catheter and the drip line – so twist them together well.

If you contaminate the catheter, discard it and select another catheter.

Intubation of the trachea. (See appendix 6)
1)   Endotracheal tubes.
For control of inspired gases, the trachea must be intubated with a tube that allows *no* gas to pass around it. This means either it must exactly fit the trachea, or it must have an inflatable cuff.
Good visibility for intubation can be obtained with the patient's head elevated, but this can result in *orthostatic* (positional) *hypotension* and reduced cerebral circulation. The reflexes that maintain cerebral blood flow regardless of position cannot be relied upon once the patient is anaesthetized. **The dog is best intubated *in lateral recumbency*, with the head and neck extended.**
2)   Intubation of the dog.
With the dog in lateral recumbency, the assistant should stand at the dog's dorsal surface with one hand behind the dog's neck and the other holding the upper lips and pulling the top jaw dorsally. This should fully extend the head and neck.
The anaesthetist should pull (gently!) the dogs tongue forward, and down over the lower incisors, thus pulling the lower jaw down and the epiglottis forward. Place the blade of the laryngoscope over the tip or just in front of the epiglottis and so that it is pulled forward and down. With the laryngoscope in place, there should be a clear view of the arytenoids. The endotracheal tube is then advanced between the arytenoids and down the trachea (some gentle twisting may be necessary). ***It is very important that you visualize the tube passing between the arytenoids and into the glottal opening. It is not sufficient simply to pass the tube over the epiglottis as this will most likely result in the tube passing down the oesophagus. Oesophageal intubation, at best, will result in the patient waking up as it will not receive any anaesthetic. More likely, the tube will press on the glottis and cause a total occlusion and death of the patient.***
3.)   Cuff inflation.
An inflatable cuff has a pilot tube with a fitting for a syringe. It is ***important <u>not</u> to over-inflate*** the cuff or pressure *necrosis of the tracheal tissues* or *occlusion of the tube* can result. To ensure correct inflation pressure, attach a syringe full of air to the pilot tube, inflate the lungs by forcing air down the endotracheal tube (by squeezing the reservoir bag) and listen for air escaping around the tube whilst inflating the cuff. Once the cuff has sealed the trachea, the leak will cease. Some pilot tubes have a valve, others have to be clamped to keep the cuff inflated.

ANSWERS TO QUESTIONS

1.
$$\text{Dose} = 30 \text{ kg} \times 0.15 \text{ mg/kg} = 4.5 \text{ mg}$$
If 1 ml contains 15 mg,
then X ml contains 4.5 mg.
$$\text{X ml} = 4.5/15 \times 1 \text{ ml} \approx 0.3 \text{ ml}$$

2.
$$\text{Dose} = 50 \text{ kg} \times 0.06 \text{ mg/kg} = 3 \text{ mg}$$
Make up 4 mg to 1 ml with sterile Water for Injection $\therefore$ 4 mg/ml
If 1 ml contains 4 mg,
then X ml contains 3 mg.
$$\text{X ml} = ¾ \times 1\text{ml} = 0.75 \text{ ml}$$

3.
$$\text{Dose} = 400 \text{ kg} \times 7 \text{ mg/kg} = 2800\text{mg}$$
Add 50 ml sterile Water for Injection to 5 g = 10% = 100 mg/ml
If 1 ml contains 100 mg,
then X ml contains 2800 mg.
$$\text{X} = 2800/100 \times 1 \text{ ml} = 28 \text{ ml}.$$
Use large bore catheter because: -
♦   the substance is HIGHLY irritant if accidentally injected perivascularly, and
♦   the volume to be delivered is huge.
♦   the drug must be administered very rapidly

4.
Acepromazine
$$1 \text{ kg} \times 0.05 \text{ mg/kg} = 0.05 \text{ mg}$$
If 1 ml contains 2 mg,
Then x ml contains 0.05 mg
$$\text{X ml} = 0.05/2 \times 1 \text{ ml} = 0.025 \text{ ml}$$
You cannot measure this accurately using a 1 ml syringe,
so dilute the acepromazine 1 in 10 using sterile Water for Injection to get 0.2 mg/ml.
Then the required dose will be in 0.25 ml.
Atropine
$$1\text{kg} \times 0.03 \text{ mg/kg} = 0.03 \text{ mg}$$
Again, dilute stock solution 1 in 10. 0.6 mg/kg becomes 0.06 mg/ml.
Required volume becomes 0.5 ml.
Pethidine
$$1 \text{ kg} \times 1 \text{ mg/kg} = 1 \text{ mg}$$
Again, dilute stock solution 1 in 10. 50 mg/ml becomes 5 mg/ml.
Required volume becomes 0.2 ml.

ANAESTHETIC MACHINES

BREATHING SYSTEMS

Introduction

An anaesthetic machine must first *satisfy the respiratory requirements* of the animal. The purpose of ventilation of the lungs is to *provide oxygen* and to remove *carbon dioxide*. A *minimum of 21% O_2* must be inhaled by an animal with *normal* ventilatory capacity. **Once an animal is anaesthetised, it cannot be assumed that ventilation is normal** and a minimum of 30% inspired O_2 is advisable to ensure adequate oxygenation. The following factors contribute to this reduction in ventilation: - reduced response to elevated partial pressure of CO_2 in the arterial blood, reduced thoracic movements (muscle relaxation), loss of the sigh reflex and loss of voluntary positioning. Reduced ventilation allows the progressive development of atelectasis (progressive alveolar collapse).

If exhaled gas remains in the breathing system to be inhaled again, the breathing system must be designed to remove CO_2 (and to add oxygen)..

Remember……
Atmospheric air is approximately 21 % oxygen and 79 % nitrogen with 0.04 % CO_2.

Anaesthetic breathing systems are classified as
- *open, semi-open, semi-closed or closed*
and
- *rebreathing* or *non-rebreathing*.

Terminology

There is some controversy in the terminology in use worldwide to describe breathing systems. The terminology adhered to in this text is as follows.

Open vs Closed

Open and Semi-Open Systems *In these systems, the patient has access to atmospheric air. You may never see anaesthesia delivered by an open or semi-open system.*

Open systems.   This would describe the induction of anaesthesia by holding a sponge soaked in chloroform some distance from the patient's nose, so that there was no restriction to inspiration of atmospheric air.
Semi-open.   If a small sieve were used to hold the sponge against the patient's nose, so that atmospheric air was drawn through the sponge by the patient's inspiratory effort, this would represent a semi-open system. Eg Schimmelbusch mask
These simple systems are unlikely to be used these days. They *cannot be used safely* with the highly volatile, highly potent and rapidly effective inhalation anaesthetics available today.

Semi-Closed And Closed Systems   These are *fully bounded systems* from which *atmospheric air is excluded*. The fresh gas is supplied from a high pressure gas cylinder via a reducing valve and a flow meter. In all anaesthetic breathing systems in common use in practice, *the inhaled gas is controlled*.
Semiclosed.   *Excess gas in the system can escape to the atmosphere* but atmospheric air cannot enter the system.
Closed.   In these systems, there is *no connection with the atmosphere*. Only the designated fresh gas enters the system, no gas leaves the system, and *carbon dioxide is chemically absorbed* within the system.

A further classification is necessary. A system can be classified as **rebreathing** or **non-rebreathing**.

In a ***non-rebreathing system***, all *gas is inhaled once only*, and then expelled to the atmosphere. All inspired gas is fresh gas. These systems are characterised by *high fresh gas flows* and are therefore always *semi-closed*. (ie some gas *must* escape to the atmosphere.)

In a ***rebreathing system***, gas can be exhaled, the *carbon dioxide removed* and the gas inhaled again. If the fresh gas flow exceeds the animal's oxygen requirements, the system will be semi-closed ie there will be a valve that allows some gas to escape into the atmosphere. If the fresh gas flow equals the animal's oxygen requirements, the system will be closed.

Systems in common use.

Non-rebreathing systems   All semi-closed (high flow)

Ayres T-piece
Bain coaxial system
Magill attachment
Lack coaxial system
Parallel Lack system

Rebreathing systems   Closed (low flow) or semi-closed (medium to high flow)

Circle absorber
Waters to-and-fro system

Oxygen Supply

Oxygen is supplied compressed in heavy metal cylinders. Gas cylinders are colour coded to indicate their contents. In Australia, the colour of medical oxygen cylinders has recently changed from *black with a white shoulder* to *all* white with a stencilled 'N' ('N' for *new)*. The pressure of the gas in the cylinder is proportional to the contents so that a pressure gauge will act as an indicator of how full the cylinder is. Cylinders are filled to a pressure of 17,600 to 19,000 kPa. A *reducing valve* on top of the cylinder brings the pressure of the gas down to a useable level. The hoses that take gas from a cylinder have attachments that are specific to each gas ie you cannot attach *eg* a nitrous oxide hose to an oxygen cylinder. ***HANDLE CYLINDERS WITH CARE. If a cylinder is knocked over and the reducing valve broken off, the cylinder may behave as a missile. ALL CYLINDERS SHOULD BE SECURED ON STABLE TROLLEYS OR CHAINED SECURELY TO A WALL.***

Flow Meters

The first step in setting up a breathing system is to deliver oxygen via a flow meter. The flow meter may be situated on the top of the cylinder, at some distance from the cylinder or as part of the breathing apparatus/anaesthetic machine. Mostly, flow meters have a ball or a bobbin floating on a column of gas flowing up a calibrated, tapered, perspex tube. By lining up the top of the bobbin or the middle of the ball with the graduations on the tube, the gas flow can be measured. Some flow meters are simple closed tubes in which a ball is supported by the pressure of the gas flowing past the base of the tube.
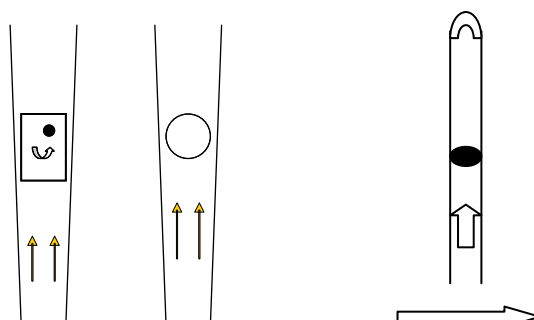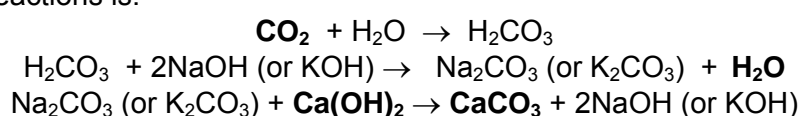
Carbon Dioxide Removal

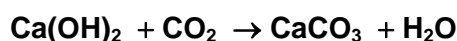There are two ways in common use to remove CO_2.

1) **High fresh gas flow used to flush the CO_2 into the atmosphere** (non-rebreathing systems which are semi-closed and have a high fresh gas flow)

2) **Absorption by chemical reaction** using a CO_2 absorbent material. (rebreathing systems which are closed or semi-closed and can be run with a low to high fresh gas flow)

CO2 absorbents: - Calcium hydroxide is the main active constituent of both soda lime and Baralyme®(about 80%). Baralyme® also contains some barium hydroxide. The more reactive hydroxides, KOH and NaOH, and water are also necessary for efficiency of the reaction with CO_2.

The series of reactions is: -

$$CO_2 + H_2O \rightarrow H_2CO_3$$
$$H_2CO_3 + 2NaOH \text{ (or KOH)} \rightarrow Na_2CO_3 \text{ (or } K_2CO_3) + H_2O$$
$$Na_2CO_3 \text{ (or } K_2CO_3) + Ca(OH)_2 \rightarrow CaCO_3 + 2NaOH \text{ (or KOH)}$$

In summary, the reaction is: -

$$Ca(OH)_2 + CO_2 \rightarrow CaCO_3 + H_2O$$

There are several ways to establish whether the absorbent is active.

1. The granules of soda lime and Baralyme® have *chemical indicators* that change colour in response to pH change. Thus, if the granules are in a clear canister, observed colour change will indicate exhaustion of the CO_2 absorbing capacity. If the granules are not changed regularly, it is possible for the indicator dye to leach out. Therefore, it is *not advisable to rely on colour change alone*.

2. The reaction described above is *exothermic*, so it should be possible, after a time, to detect an interface of temperature change within active soda lime or Baralyme®. There will be a *zone of warmth* where the reaction is taking place.

3. Attempt to crush granules of absorbent between your fingers. *Fresh granules crumble* easily. Exhausted granules are hard.

4. By far the most reliable method of establishing that the absorbent is active is to measure the CO_2 content of gases inspired once a patient is attached to the breathing circuit.

BREATHING SYSTEMS IN COMMON USE
Non-rebreathing Systems

T-Piece (Mapleson's Series E. See p 28)

There are several variations of the T-Piece, but basically, they function the same way. The patient breathes in and out of what is essentially a wide bore 'breathing' tube or *reservoir tube*. Fresh gas enters the reservoir tube at the end to which the patient is connected. The other end of the tube is open to the atmosphere. The gas expired by the patient is flushed down the tube towards the open end, and out into the atmosphere. For successful elimination of CO_2, the fresh gas flow must be high enough to ensure that the patient end of the reservoir tube contains only fresh gas by the time the patient is ready to inspire again. Remember that expired gas and fresh gas will mix because of turbulence in the tube. To be sure of elimination of CO_2, the fresh gas flow must be *2- 3 × Respiratory Minute Volume*. This means that for a 5 kg cat, the flow must be 2 - 3 × approximately 900 ml/minute = 1800 – 2700 ml/minute. Alternatively, 4 - 500 ml/kg/min.

T-pieces often have a small reservoir bag **with the end cut off** on the open end of the breathing tube. (Mapleson's Series 'F'. See p 28) This bag can be used if positive pressure ventilation is required. The anaesthetist can intermittently hold the end of the bag closed and squeeze the contents of the bag to inflate the patient's lungs. Respiratory movements can be counted by observing the bag movements if the patient is lost to view under surgical drapes. Some T-pieces have a spill valve and a small reservoir bag (intact). Gas leaves the system via the spill valve which must never be completely closed because of the very high fresh gas flow rate..

The T-Piece is often used for birds, cats, small dogs, rats, mice guinea pigs, and small native animals (animals < 7 – 10 kg).


Fresh gas delivery tube

Patient

Reservoir Tube
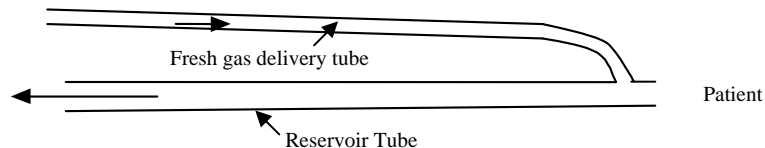
Ayres T-Piece

Remember – to avoid the patient rebreathing its expired gas, the flow rate must be at least 2 - 3 × the patient's estimated Respiratory Minute Volume, or about 500 mL/kg/min.

Bain Coaxial System

The Bain Coaxial system is functionally the same as a T-Piece, but the tube delivering fresh gas and the patient breathing tube are coaxial. As with a T-Piece, the fresh gas flow rate must be *2 - 3 × Respiratory Minute Volume.*
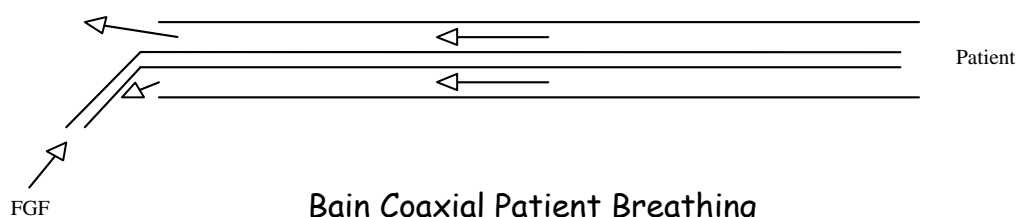
Patient

FGF   **Bain Coaxial Patient Breathing**

The Modified Bain Coaxial Systems (Mapleson's Series D. See p 28) is in common use. It has a 'spill valve' or 'pop-off valve' at the exhaust end of the breathing tube and the

reservoir bag is closed. The valve will let gas escape to the atmosphere but no gas can enter the system via this valve. It is easier to manually assist the patient's ventilation with this system as the valve can be partially closed to allow inflation of the patient's lungs. **The spill valve must never be left in the closed position.**
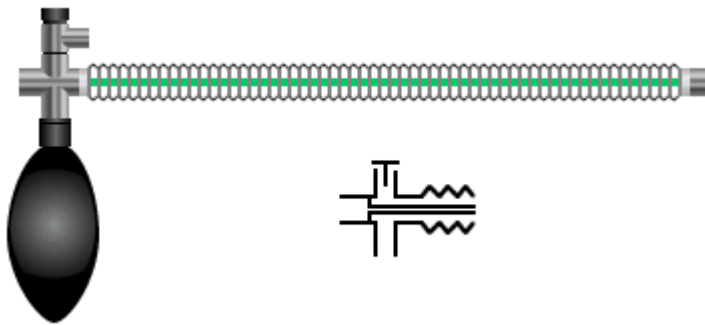

The Magill Attachment *(Mapleson's Series A. See p 28.)*

The Magill Attachment consists of a wide bore tube connected to the fresh gas outlet at one end and the patient at the other. The patient end has a one-way valve to allow gas to escape to the atmosphere without allowing atmospheric gas to enter the breathing tube (*a spill valve*)*.* Gas escapes via this valve whenever there is a slight positive pressure in the tube (during expiration and the expiratory pause, but not during inspiration). The fresh gas end of the tube has a reservoir bag. In a spontaneously breathing patient, a fresh gas flow of *80 - 90% × Respiratory Minute Volume* will result in CO_2 elimination from the breathing tube. The efficiency of this system is compromised during intermittent positive pressure ventilation (IPPV) (manual ventilation), when gas escapes from the spill valve during forced inspiration. *This is a serious problem – it is almost impossible to perform IPPV using a Magill Attachment without causing the patient to re-breathe its expired gases and therefore accumulate CO_2.*
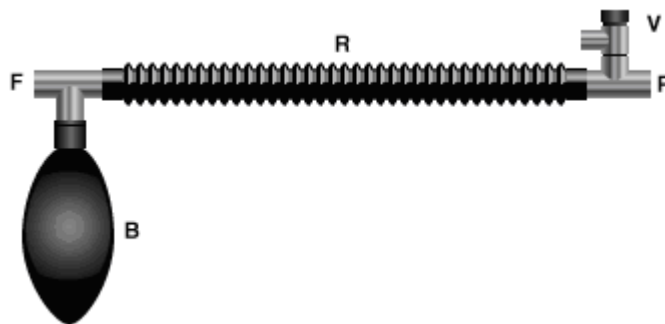

Lack Coaxial System

The Lack Coaxial System is structurally similar and functionally identical to the Magill, except the spill valve is situated at the end of the tube distal to the patient and is connected to the patient end via a narrow tube running within the larger breathing tube.
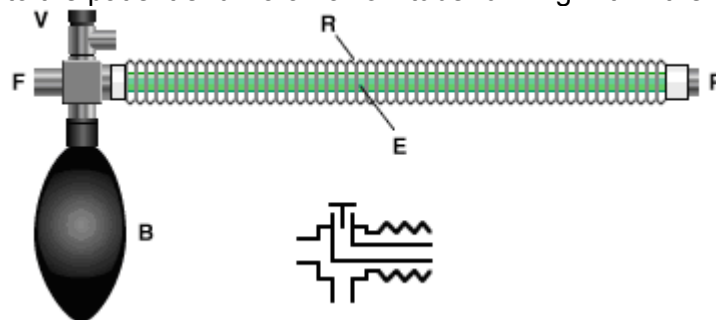
The Parallel Lack System

In the Parallel Lack System, the two tubes are not coaxial, but run side by side. A problem with co-axial systems is that the inner tube can become disconnected or damaged. Should this occur, the entire reservoir tube becomes dead-space, allowing expired CO_2 to be rebreathed. The advantage of the parallel system is that should the tubing become damaged, it will be visible and can be replaced using conventional anaesthetic tubing.
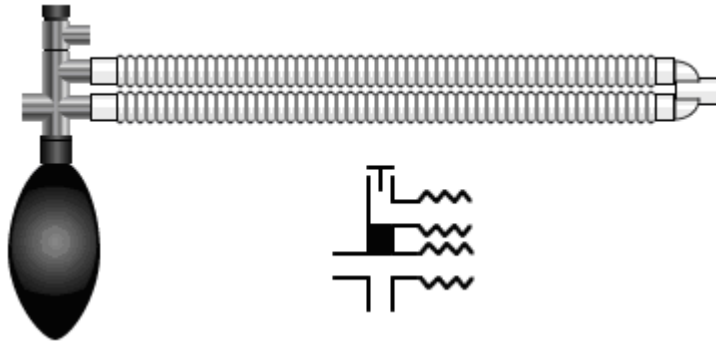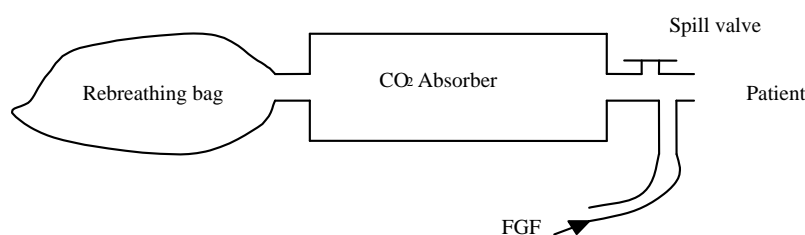
Rebreathing Systems

Remember, rebreathing systems must have some way of chemically removing CO_2 before the exhaled gas is breathed in again. The two systems described next utilise CO_2 absorbing granules eg soda lime or Baralyme®

Water's To-and-Fro

The Water's To-and-Fro system has been in use in veterinary practice since 1923. The CO_2 absorber is placed close to the patient, so the patient breathes into and out of the absorber. *It is important that the dead space volume between the patient and the absorber is kept to a minimum.* There is a spill valve (allowing gas to escape but not enter the system) situated close to the patient, and a reservoir bag at the distal end of the absorber. If the fresh gas flow rate equals the patients oxygen consumption (metabolic requirements), the spill valve can be closed, and the patient's respiratory requirements will be met until the CO_2 absorption capacity is exceeded. If the fresh gas flow rate is greater than the patient's oxygen consumption, the spill valve must be open. As the spill valve is situated close to the patient, the gas leaving the breathing system will be exhaled gas, high in CO_2.

This system has problems: -

- The absorber must be close to the patient. This may be a logistical problem in some circumstances.
- The CO_2 absorption capacity will be used up sequentially from the patient end. This means that, over time, the apparatus dead space increases ie the space around the exhausted absorber granules becomes apparatus dead space.
- CO_2 absorption is an exothermic reaction. The patient's airways will be exposed to high temperatures because the process takes place so close to the patient.
- Soda lime and Baralyme® contain KOH and NaOH. Tiny particles of these highly caustic substances may be inspired by the patient and irritate the airway mucosa.

Water's to-and-fro

The Circle Absorber

The circle absorber is the most commonly used patient breathing apparatus in veterinary practice in Australia. The essential elements of this breathing system are

- *CO_2 absorber* is connected to the patient by *breathing tubes (making a circle).*
- Two *unidirectional valves* within the circle, usually situated on the absorber canister, ensure that one arm of the circle conducts gas to the patient (inspiratory limb), and the other takes expired gas away from the patient (expiratory limb). **Gas can flow around the circle in one direction only. This is critical.**
- *Reservoir bag* situated so that it is *separated from the patient by the unidirectional valves*. This system would be too rigid without the reservoir bag, which allows the apparatus to accommodate a fluctuating volume of gas. The reservoir bag will rise and fall as the patient breathes out then in. It can be used to provide positive pressure ventilation if the patient's ventilation ceases or is inadequate.
- A *spill valve* (= Adjustable Pressure Limiting or APL valve*)* usually placed in the expiratory limb (so that the escaping gas is high in CO_2).
- *Fresh gas inlet*, usually placed in the inspiratory limb (so that fresh gas goes straight to the patient.)

To satisfy the respiratory requirements of the patient, the *fresh gas flow rate must be at least equal to the patients' metabolic oxygen requirements*. If fresh gas flow rate = metabolic oxygen requirements, the spill valve can be closed, and the breathing requirements will be satisfied until the CO_2 absorber is exhausted. If fresh gas flow rate > metabolic oxygen requirements, the spill valve *must be open* to allow excess circuit gas to escape to the atmosphere.


Circle Absorber
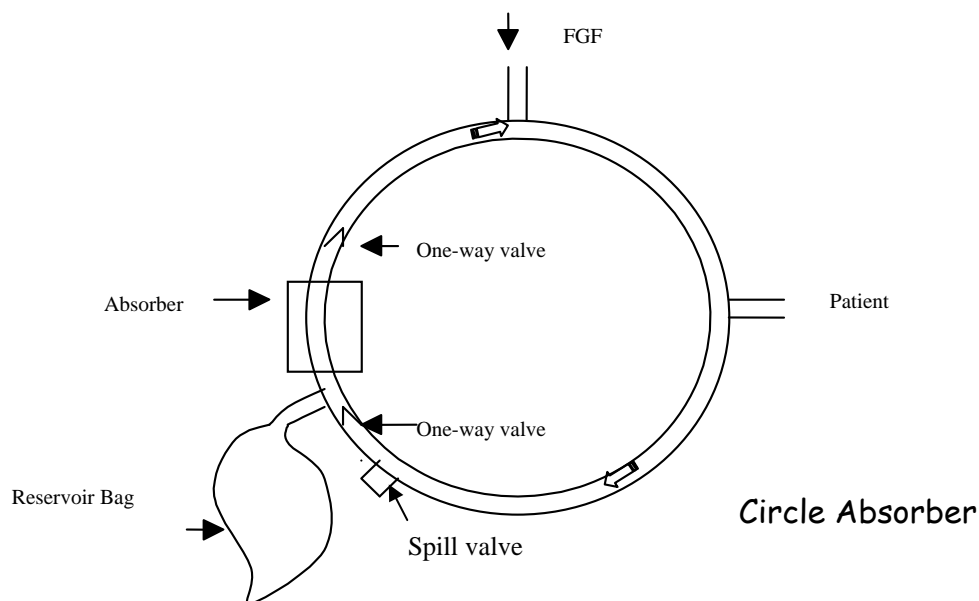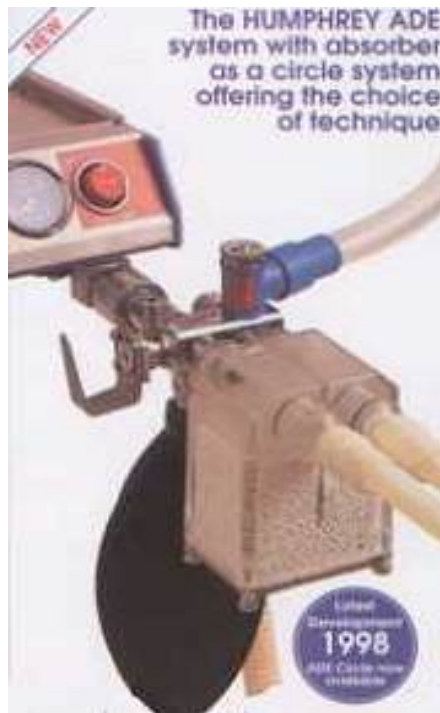
Humphrey ADE
The Humphrey ADE circuit has had something of a revival in recent years. This is an extremely versatile system that can act as a variety of non-rebreathing circuits : - Mapleson Series A *(Magill Attachment)* suitable for spontaneously breathing patients only, Mapleson Series D (a modified Bain circuit), or a Mapleson Series E (a *T-piece*), both suitable for patients requiring positive pressure ventilation. Recent incorporation of an optional CO_2 absorber means that this system can also operate as a rebreathing circuit ie a *circle absorber*.

The Mapleson classification of patient breathing systems is shown below.

Mapleson's classification of non-rebreathing circuits

Mapleson's classification divides non-rebreathing circuits into functionally similar groups, on the basis of the fresh gas flow required to prevent rebreathing and the ease with which intermittent positive pressure ventilation may be performed.

FG = Fresh gas   P = Patient
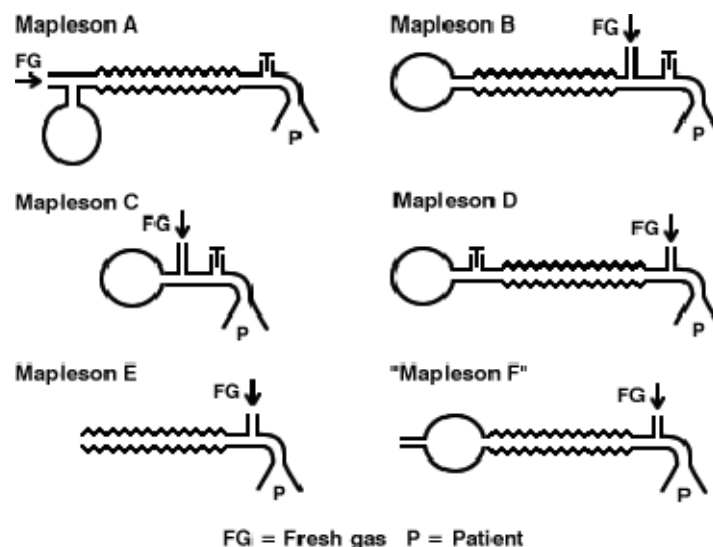
Mapleson's classification of non-rebreathing circuits

- Mapleson's A - the Magill and Lack circuits
- Mapleson's B and C - Rebreathing of exhaled gases occurs even when very high fresh gas flow rates are used, since inspiration is taken from the same space into which the previous breath was expired. These are unsatisfactory for anaesthesia, but may be used in emergency for resuscitation.
- Mapleson's D - the modified Bain circuit.
- -Mapleson's E Ayre's T piece
- Mapleson's 'F' - not originally classified by Mapleson, but is used to refer to Jackson-Rees' modification of Ayre's T-piece.

The Humphrey ADE circuit provides the ability to switch between the Mapleson's A, D and E arrangements.

ANAESTHETIC DELIVERY SYSTEMS

All the breathing systems described above can be used to deliver inhalation anaesthetic agents by incorporation of a vaporizer. Vaporizers are used to evaporate volatile anaesthetic agents so that the anaesthetic can be delivered with the inspired gas to the gas exchange surface of the lungs.

VAPORIZERS

The addition of a vaporizer to the system brings in new considerations.

A vaporizer, in the simplest terms, is a chamber containing an anaesthetic liquid /gas interface. The anaesthetic vapour in the space above the liquid is washed out in a controlled fashion by channeling some of the gas that the patient is to breathe through the vaporizer.

The saturated vapour pressure of the commonly used volatile anaesthetic agents is much higher than the partial pressure required for anaesthesia. The vaporizer brings about dilution of saturated vapour in a controlled fashion so that anaesthesia can be performed safely.

Saturated vapour

Liquid

Stylized drawing of a vaporiser
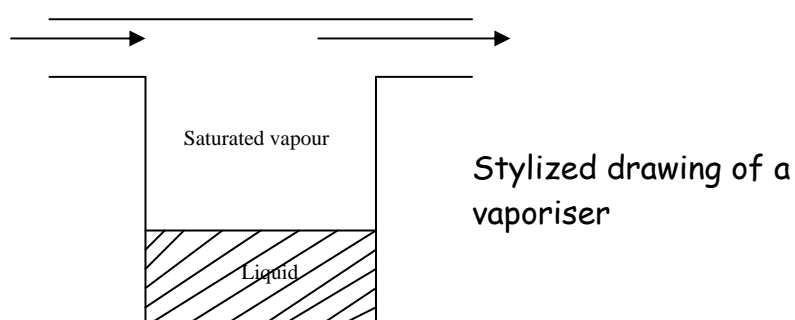
A **vaporizer must sit in a gas flow.** Breathing systems can have two gas flows – *the fresh gas flow* designated by the anaesthetist via a flow meter and the flow created by the *patient's breathing*. Broadly speaking, there are two types of vaporizers, *draw-over* and *plenum*

Draw-Over Vaporizers.

A draw-over vaporizer is designed to be *inserted into the patients breathing system*, and therefore must offer very *little resistance* to gas flow. They are used in **circle absorber systems**, in the patient determined gas flow (*in-circle vaporizers*). They are usually situated in the inspiratory limb of the circle. Examples of draw-over vaporizers are the Stephen's and the Goldman vaporizers. The Stephen's vaporizer is in common use in Australia as part of the Stephen's Circle Absorber. An adaptation of the Goldman vaporizer is in use on the Komesaroff Circle Absorber. (Both of these machines were designed by Australians)

The Stephen's and Goldman vaporizers are glass jars through which some of the patients inspired gas is drawn. A dial on top of the glass jar allows the proportion of the inspired gas entering the bottle to be altered, thus altering the concentration of anaesthetic vapour entering the inspiratory limb of the circle.

These are *relatively inefficient* vaporizers and the *output is variable*.

- The output varies with the *ambient temperature* (*low temperature, low output* and *vice versa*). *This is really important.*
- When the vaporizer is *turned on initially*, the chamber will contain saturated vapour and the *output will be high*. Subsequently, the vapour in the chamber will be less than saturated. If the *flow through the vaporizer is high*, the chamber will contain vapour that is much less than saturated and the *output will be low*. *Ie output varies with flow.*

These vaporizers are *not calibrated in percentages*, as the *output is variable and largely dependent on the patient's respiratory minute volume and the temperature*.

There are situations in which draw-over vaporizers become far more efficient (ie higher output) and the vaporizer setting *must be turned down*.

- In *hot weather*, the saturated vapour pressure increases. The output of the vaporizer will increase. This is really important.
- If the *patient is very large in relation to the volume of the breathing circuit*. The temperature of the gas in the circuit will rise towards body temperature with time and more anaesthetic will be available as vapour. This is really important.
- If the patient requires *positive pressure ventilation*, the Respiratory Minute Volume will increase. This results in increased flow through the vaporizer and an increased inspired concentration of anaesthetic vapour. This is really important.
- When the vaporiser is first turned on, the gas above the liquid anaesthetic is saturated with anaesthetic vapour. There will be an initial surge of concentrated vapour.
- If the vaporiser is shaken, the output will be high.

The Goldman-type vaporizer, theoretically, can achieve a maximum delivered concentration of 3% of isoflurane under normal conditions (spontaneous ventilation, low O_2 flow rate & ambient temperature of 20°C). Although these vaporizers are relatively inefficient, it must be remembered that an inspired concentration of 3% isoflurane can be lethal after a time, and that high temperatures or positive pressure ventilation will result in very high delivered concentrations of anaesthetic vapour.

The Stephen's vaporizer has a sleeve which, when pulled down close to or beneath the surface of the liquid, greatly increases the efficiency of vaporisation by causing the gas passing through the vaporiser to flow close to the surface of the anaesthetic liquid. The output of anaesthetic agent from the Stephen's vaporiser can be very high.

It must be remembered that ***draw-over vaporisers are designed to 'top-up' the patient's expired gas with anaesthetic vapour to bring the concentration back to an anaesthetic level before it is rebreathed by the patient.*** For example, a patient may inspire 1% isoflurane and expire 0.7%. (Remember that some of the inspired vapour crosses the alveolar membrane and enters the blood to keep the patient asleep.) The expired gas will then continue around the circle and pass through the vaporiser again. The vaporiser needs only to 'top up' this gas to 1%. In this situation, the output of the vaporiser need be only 0.3%.

Plenum Vaporizers.
The term "Plenum" refers to the system where *gas enters the vaporising chamber under pressure*, as opposed to the draw-over type, where gas is "sucked" through by the patient's inspiratory effort. These vaporizers are *situated in the fresh gas flow*, designated by the anaesthetist by turning on a flow meter. A proportion of the fresh gas is diverted into the vaporising chamber and takes anaesthetic vapour with it to the patient.
The plenum vaporizers in use today are mostly *temperature compensated*, *flow compensated*, are highly *efficient* and have *a highly predictable* output. The calibration of the Fluotec Mark 3 is almost independent of temperature in the range of 18 - 36° and flow from 250 ml/min to 10 l/min. Wicks within the vaporisation chamber ensure that the chamber contains saturated vapour. The Fluotec Mark 3 and subsequent "tec" type vaporizers are calibrated in percentages, and are accurate to within 10% of the dial setting.
Plenum vaporizers can be placed in the fresh gas flow of ***non-rebreathing and rebreathing systems***. The resistance to airflow through plenum vaporizers is too high for them to be inserted in the patient's breathing system (**eg *they cannot go <u>in the circle</u> of a circle absorber***).

Which vaporiser where?
***Non-rebreathing* systems**.
The non-rebreathing systems all have the *vaporizers in the fresh gas flows*. These days, the "tec" type vaporizers are most common. Because all inspired gas is fresh gas, (no rebreathing) the delivered *concentration of anaesthetic vapour is highly predictable*. If a precision vaporizer is set on 1%, the concentration delivered to the patient will be between 0.9 and 1.1%. (The vaporiser is accurate to about 10%).

Water's to-and-fro.

The vaporizer will be placed *in the fresh gas flow* in a Water's to-and-fro system. With a high flow and a precision vaporizer, the concentration of anaesthetic vapour will be highly predictable, as in the non-rebreathing systems.

Circle Absorber.

There is a *choice of vaporiser positions* in the circle absorber. A draw-over vaporizer can be placed in the patient driven gas flow eg a Stephen's or a Goldman type *in-circle*, or a plenum vaporizer can be placed in the fresh gas flow eg a 'tec'-type vaporiser *out-of -circle*. There are important ramifications of vaporizer position.

Out-of-circle vaporizer. If a precision vaporizer (eg 'tec' type) is placed in the fresh gas flow of a circle absorber patient breathing system and a high fresh gas flow is chosen (eg 1 × alveolar ventilation rate), the concentration of anaesthetic vapour delivered to the patient is highly predictable. With a high fresh gas flow, each breath taken will be mostly fresh gas ie there will be **little rebreathing**. Therefore, if the vaporizer is set on, say, 1%, then 1% will be delivered to the circle and the patient will breathe 1%. However, as the fresh gas flow is decreased, the gas is **rebreathed** by the patient. Anaesthetic vapour is taken up by the patient at each breath. The expired gas mixes with a small volume/min of fresh gas so that the circle contains gas with a lower percentage of anaesthetic vapour than that of the fresh gas. Thus the inspired % of anaesthetic vapour will be less than the % delivered to the circle from the fresh gas supply.

Eg Consider a 20kg dog breathing via a circle absorber with an Isotec (a precision isoflurane vaporizer), with the vaporizer set on 1%. The dogs Respiratory Minute Volume is about 2.4 l/min, and its metabolic oxygen requirements (oxygen uptake) will be about 100 ml/minute.

Consider the difference between selection of a fresh gas flow of 2.4 l/minute and 100 ml/minute (just enough to meet its oxygen requirements).

First, consider a high fresh gas flow rate, 2.4 l/min = RMV. There are two ways to look at this. Firstly, in terms of the absolute amount (mls) of isoflurane vapour delivered to the breathing circuit. Secondly, consider the different amounts of rebreathing of circuit gases by the patient.

High Flow: -

- 2.4 l/min of 1% isoflurane results in **24 ml/minute of isoflurane** vapour being delivered to the circle. In the first few minutes of the anaesthetic, the dog's isoflurane uptake will be very high, eg 12 – 15 ml of vapour/min. Because the flow is so high, the spill valve must be open and a lot of oxygen and isoflurane will be lost to the atmosphere. So, although there is lots of waste, enough isoflurane enters the circle to keep the patient asleep.
- If a high fresh gas flow rate is chosen for a circle absorber with the vaporiser out-of-circle, the system **_almost_** acts as a **non-rebreathing system** ie each breath is fresh gas. Therefore, the inspired concentration = the concentration leaving the vaporiser. Whichever way you look at it, the patient very likely will stay asleep.

Now consider a **low fresh gas flow**, eg 100 ml/min = O_2 uptake.

- 100 ml/minute of 1% isoflurane will deliver to the circle approximately **_1 ml/min of isoflurane_** vapour. At this low flow, the spill valve will be closed (FGF = O_2 uptake) so no isoflurane can escape, but the isoflurane uptake by the dog that is necessary to keep the dog asleep early in the anaesthetic is far in excess of 1 ml/min of vapour. The dog will wake up unless you can provide more isoflurane. So, turn the vaporizer up. The vaporizer can be set as high as 5%, but this will result in only 5ml of vapour delivered to the circle – this may still be below the requirements to keep the dog asleep in the early, high uptake phase of the anaesthetic. After only a few minutes of anaesthesia, *the dog's anaesthetic requirements drop dramatically*, and the vaporizer output will be adequate. (Even at this low flow rate, the *vaporizer will have to be turned down.)*

- Now, look at it the other way. At this low fresh gas flow rate, there will be a **high degree of rebreathing** of the gases in the circle, and the patient will continue to remove isoflurane from the gas in the breathing circuit, as it repeatedly inspires the gas. The concentration of anaesthetic vapour in the circuit gas will fall below that required to keep the patient asleep.

*What these two situations illustrate is that, although the vaporizer is precise and predictable, 100 ml/min and 2.4 l/min of 1% isoflurane will result in vastly different concentrations of anaesthetic vapour **delivered to the dog.** As the fresh gas flow rate falls, the concentration delivered to the dog begins to fall below the concentration that leaves the vaporizer.*

These two situations described are extreme, and in between these two flow rates are many satisfactory settings.

Points: -
  · ***With a circle absorber with the vaporizer out of circle, the concentration of anaesthetic vapour delivered to the patient will fall as the fresh gas flow rate is decreased and <u>rebreathing of circuit gases is increased</u>.***
  · ***What really counts is the <u>concentration of vapour in the inspiratory limb</u> of the breathing circuit.***

In-circle vaporizer.
Now consider a draw over vaporizer in the circle of a circle absorber. These systems are designed to be run with a very low flow so that there is a **high degree of rebreathing.** These vaporisers are inefficient at normal ambient temperatures, and the vaporizer output of anaesthetic vapour is low while the animal is breathing spontaneously. The vaporizer is in the flow generated by the dog and the output will be dependent on the dogs Respiratory Minute Volume. **Therefore, the *concentration of anaesthetic vapour cannot be predicted* by the setting on the vaporizer**

Again, consider the 20kg dog with an oxygen uptake of 100 ml/min and Respiratory Minute Volume of 2.4 l/min. *Remember, a circle absorber with a vaporizer in the breathing circuit is meant to be run on a low flow.*
Consider the difference between selection of a fresh gas flow of 100 ml /minute (just enough to meet its oxygen requirements) and 2.4 l/minute (RMV).

- With a flow of 100 ml/min, the spill valve can be closed. 100 ml/min of oxygen enters the circle, CO_2 is absorbed and anaesthetic vapour is picked up as the dog draws gas through the vaporizer. This system relies on a **high degree of rebreathing**. Eg if the dog inspires 1% vapour, it will exhale, say, 0.7%. This exhaled gas will then pass through the vaporiser again, and it is hoped that it will pick up 0.3% to bring this gas back to 1%. (However, over a period of time, the anaeathetic vapour uptake by the dog will decrease, and the temperature of the gases will rise, so the concentration in the circle will rise. So turn the vaporizer down, or the depth of anaesthesia will increase.)
- With a flow of 2.4 l/min, the spill valve will be open. Only oxygen enters the circle, and anaesthetic vapour and oxygen leave the circle via the spill valve. The high flow of oxygen results in a net loss of anaesthetic vapour in the circle, and so the dog will wake up. So, turn the vaporizer up. With a high fresh gas flow rate, there is **very little rebreathing**. The low output of the vaporiser may not be enough to keep the patient asleep.

What these two situations illustrate is that, in this system, economy of anaesthetic vapour is a consideration. As the flow of oxygen into the circle increases, more anaesthetic vapour is lost out the spill valve and the concentration decreases. ***You never know what concentration of anaesthetic vapour you are delivering with an in-circle vaporiser (unless you have an inhalation agent meter).***

Point. With a circle absorber with the vaporizer in circle, the concentration of anaesthetic vapour delivered to the patient will fall as the fresh gas flow rate is increased. This system relies on **a high degree of rebreathing, therefore a low fresh gas flow.***.*

Remember – what really counts is what gases are delivered to the patient!

With a circle absorber, the patient inspires what is in the inspiratory limb of the circle. The concentration of vapour in the inspiratory limb is what counts.

You must understand the effects of high and low fresh gas flows and of vaporizer position in terms of how changes will affect the gases delivered to the patient.

In or out-of-circle vaporizers?

> **VOC = vaporizer out of circle (in the designated fresh gas supply)**
> **VIC = vaporizer in the circle (in the patient generated gas flow.)**

Cost to buy
- VOC > VIC

Cost to run
- VOC >> VIC

Predictability of delivered concentration
- VOC, high flow – highly predictable wrt delivered concentration of anaesthetic vapour, T° and flow compensated, FGF determined by anaesthetist. VOC with low flow $\Rightarrow$ loss of predictability wrt delivered concentration of anaesthetic vapour.
- VIC - can *never* predict anaesthetic vapour concentration in the circle. Output affected by ambient T° and RMV, flow through vaporiser determined by patient.

Intermittent Positive Pressure Ventilation.
- VOC, medium - high flow   Delivered concentration not changed by IPPV
- VIC, low flow   IPPV increases flow through the vaporizer and increases delivered concentration

Humidification of inspired gas
- VOC, high flow   Inspired air is dry
- VIC, low flow   Inspired gas will be humidified by the patient after a time

Temperature of inspired gas
- VOC   Inspired gas will be at cold (compressed gas becomes very cold as it leaves the cylinder)
- VIC   Inspired gas will be warmed after a time

INHALATION ANAESTHESIA

DEFINITIONS
There are two common ways of describing quantities of inhalation anaesthetic agents: - partial pressures (mm Hg) and concentrations (volume %).

Concentration.   This is a simple concept that is useful when we speak of delivery of anaesthetic circuit gases. The delivered gas may be, say, 3 % by volume of the gas phase. However, this concept is confusing when we consider the passage of molecules of anaesthetic vapour dissolving in the blood. If a gas is present in the alveoli at 3% by volume, some molecules will cross the alveolar membrane and dissolve in the blood. The amount in the blood will be determined by the solubility of the anaesthetic in blood. However, it is common to refer to concentration of inhaled anaesthetic agent in volume % when in the gaseous phase.
- Precision vaporisers are calibrated in %.
- Most agent monitors give a % value for anaesthetic vapour in breathing circuit gases.

Partial pressures.   A mixture of gases in a closed container exerts pressure on the walls of the container. The pressure exerted by an individual gas within that mixture is its partial pressure ie part of the whole pressure. Usually, when we speak of a partial pressure we refer to conditions of standard atmospheric pressure and temperature ie 760 mm Hg and 20ºC. If a gas equilibrates with and dissolves in a liquid under standard conditions and the pressure of that gas in the gaseous phase is say 300 mm Hg then the pressure of the gas in the liquid phase will be the same, 300 mm Hg. However, the number of molecules per unit volume of each phase will differ, depending on the propensity of the molecules to occupy each of the available phases ie the solubility of the molecules in different phases. Understanding this concept is critical to an anaesthetist as biological systems are made up of many different phases. Eg inhaled anaesthetics must pass from the gas phase in the alveoli, through the alveolar membrane to blood and eventually and importantly dissolve in fatty membranes in the CNS to act on brain tissue.   Provided the anaesthetic is in equilibrium amongst these different phases, the partial pressures will be the same throughout.
- If we measure arterial blood gases, the results will be expressed as partial pressures eg PaO_2 may be 110 mm Hg. This will be similar to the partial pressure of oxygen in the alveoli. Thus, the concentration of oxygen in the alveoli is 110/760 ≈ 14%. (Return to pages 9 & 10 to refresh your memory about the 14%)

In the following text, the terms 'concentration' and 'partial pressure' are used. It is important that you grasp these concepts before you read on.

ABSORPTION
Volatile or gaseous anaesthetic agents are delivered in a variety of ways to be inspired by the patient. For anaesthesia to be achieved there must be partial pressure gradients for the anaesthetic agent between the inspired gas and the alveolar gas and between the alveolar gas and the blood. Diffusion across the alveolar wall is proportional to the pressure gradient from the alveolar gas to the blood. Several factors determine the partial pressure in the alveolus.
The anaesthetic gas or vapour partial pressure in the alveolus depends on the *inspired concentration* and the patient's *Alveolar Ventilation (AV).* Some of the anaesthetic vapour in the alveolus will cross the alveolar membrane into the blood. The rate of uptake from the alveolus depends on the *gradient* from alveolus to blood, as well as the *solubility* of the agent in blood and tissues and the *rate of blood flow* past the alveolus.

Factors affecting the concentration/partial pressure of anaesthetic agent in the alveoli and then in the blood.

a) **Concentration of anaesthetic agent in inspired gas**.   The higher the inspired concentration, the faster the alveolar concentration rises.

b) **Alveolar ventilation.** Ventilation of the respiratory tree results in changing the composition of the alveolar gas towards the inspired gas. A high AV (↑tidal volume and/or ↑ respiratory rate) rapidly brings the alveolar gas closer in composition to the inspired gas. The alveoli are 'flushed out' more efficiently.

c) **Solubility Partition Coefficients.**   The greater the solubility of a gas or vapour in blood or tissues, the greater will be its uptake by the blood and the tissues. Trying to achieve an anaesthetic level of an agent that is highly soluble in blood and tissues can be likened to filling a bucket with holes in the bottom - a very fast stream of water may eventually fill the bucket. Several agents no longer in popular use (eg methoxyflurane, ether and chloroform) were eventually by-passed, partly because of high solubility and therefore slow inductions in spite of very high delivered concentrations of anaesthetic agent. Solubility is defined as the ratio of the concentrations of the anaesthetic agent (gas or vapour) in two phases when the two phases are in equilibrium with respect to partial pressures eg the concentrations of the anaesthetic agent in solution (in the blood) and in the gaseous form (in the alveolus) .

alveolus 80 vols%

Blood / gas partition coefficient = 40/80 = 0.5

_____

Blood   40 vols %
_____

Remember, for a *fast induction* and *fast wake-up*, choose an agent with *low blood solubility*. Such an agent will equilibrate rapidly with the blood and tissues and will achieve an effective partial pressure in the brain quickly.
In a gas that has low blood solubility, it doesn't take many molecules of anaesthetic vapour to create the partial pressure necessary to anaesthetise the patient.
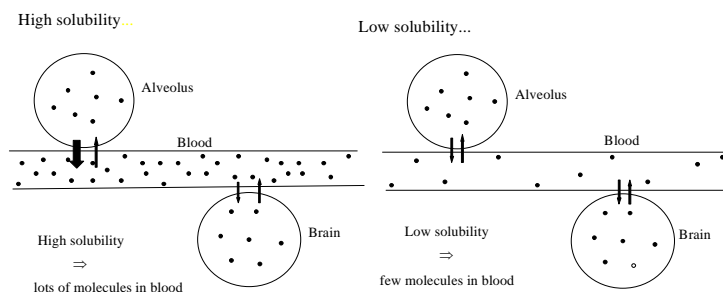
d)   Pulmonary blood flow. To achieve an effective brain concentration of anaesthetic agent, a high blood concentration is necessary. If the cardiac output is very high, the anaesthetic agent is taken away from the lungs very rapidly and achieving effective blood levels takes longer than if the cardiac output is lower. This becomes apparent when a very anxious patient is being anaesthetised. The patient has a very high cardiac output and takes a high dose of anaesthetic to achieve unconsciousness.

DISTRIBUTION

To achieve anaesthesia, a drug must act in the brain. Once dissolved in the blood, the anaesthetic agent must reach the brain at an effective partial pressure. However, the blood will deliver the agent to all tissues in the body, depending on *the blood supply* to the tissues and the uptake by the tissues. The uptake depends on the *solubility of the agent in the substances that make up the tissue.*
Blood is directed to different tissues depending on the metabolic requirements of the tissue.
Consider the various tissues in the body as belonging to one of the following groups: -
Vessel-rich group   *brain, heart, kidneys and endocrine glands*

Muscle group   *muscle and skin*

Fat group   *fat*

Vessel-poor group   *bone, tendons, ligaments and cartilage*

Table: -   Percentage body mass and percentage cardiac output received by each of vessel-rich, muscle, fat and vessel poor tissues.

	Vessel –rich	Muscle	Fat	Vessel-poor
% Body Mass	9	50	19	22
% CO	75	18	5.5	1.5

The *vessel-rich group* will be the first to reach equilibrium. This occurs in 5 – 20 minutes.
The *muscle group* reaches equilibrium in 1.5 – 4 hours.
The *fat group* reaches equilibrium very slowly, > 4 hours.
Anaesthetics that have high fat solubility, will enter tissues with high lipid content rapidly eg brain.

ELIMINATION

It was originally believed that almost all of an inhaled anaesthetic agent was simply breathed out when the anaesthetic was turned off. However, it is now known that all agents are metabolised to some extent.
Consider the elimination of inhaled anaesthetics to be by
- Exhalation of unchanged anaesthetic
- Via skin, milk, sweat, urine and mucous membranes
- Biotransformation (metabolism).

A small amount of elimination of the inhalation anaesthetics takes place via skin, secretions and excretions. The real issue is how much is breathed out and how much is degraded in the body ie exhalation vs biotransformation. *The ideal agent would be totally and rapidly exhaled unchanged*. This would avoid reliance on metabolism for recovery and production of toxic metabolites and 'hang-overs'. The amount of anaesthetic agent metabolised has been established for the following agents: -

Methoxyflurane	50%
Halothane	10 – 25%
Enflurane	2.5%
N_2O	0%
Isoflurane	0.17%
Desflurane	0.02%
Sevoflurane	~3%

RUBBER SOLUBILITY

Many anaesthetic machines have some rubber components eg the reservoir bag. Rubber absorbs anaesthetic agent during the procedure and releases it once the vaporizer is turned off. This can result in prolongation of anaesthesia if the patient is left breathing on the circuit to ensure adequate oxygenation during recovery. This is less of a problem now as most anaesthetic tubing is plastic and less anaesthetic agent is absorbed by plastic. However, it is advisable to be aware of relative rubber solubilities and to turn off the vaporizer or disconnect the patient earlier if the agent in use has very high rubber solubility. Here are some rubber/gas partition coefficients: -

Methoxyflurane	630
Halothane	120
N_2O	1.2
Isoflurane	62

MINIMUM ALVEOLAR CONCENTRATION (MAC)

The MAC of an inhalation anaesthetic is a measure of **how potent it is**.

The MAC of an anaesthetic is the concentration (%) in the alveoli that renders 50% of patients immobile when subjected to a painful stimulus.

There are two problems with this concept. One is that we need an agent monitor to measure the alveolar concentration and the other is that if we are about to subject a patient to surgery, a 50% chance that the patient will be insensitive to pain is not sufficient. An alveolar concentration of 1.3 × MAC renders 96% of patients insensitive to a painful stimulus.

Remember

- **a high MAC means low potency** and **a low MAC means high potency.**

MAC VALUES (%)

	Human	Dog	Cat	Horse
Methoxyflurane	0.16	0.23	0.27	0.22
Halothane	0.77	0.87	0.82	0.88
Isoflurane	1.27	1.28	1.68	1.31
Sevoflurane	1.8	2.25	2.58	2.31
Desflurane	7	7.2	9.8	7.6
Nitrous Oxide	101	200	250	190

These values show that eg methoxyflurane is > 800 times as potent as nitrous oxide in dogs.

It is important to remember that MAC is a <u>population statistic</u>. Any report of a MAC value must be accompanied by clear reference to the population that it refers to. For instance, the MAC for isoflurane for fit young adults is higher than for a group of geriatric patients. MAC values cannot be applied to individual patients.

The MAC for an anaesthetic agent for a defined population sample is established using the drug in isolation. Eg the MAC for halothane in dogs is 0.87%. This means that <u>in the absence of any other drugs,</u> 0.87% in the alveolus will render 50% of dogs insensitive to a surgical stimulus. We <u>almost NEVER</u> use an inhalation agent on its own. We often deliver less than 1 × MAC to an anaesthetized patient because of the combined effects of all drugs administered.

SATURATED VAPOUR PRESSURE
The saturated vapour pressure of a liquid tells you **how well it vaporises**. Values are expressed in mm Hg and a *high value means that you can achieve a high percentage of vapour*. Below are some saturated vapour pressure values at 20°C: -

Methoxyflurane	22.5 mm Hg
Halothane	241
Enflurane	175
N_2O	39,000
Isoflurane	250
Desflurane	700
Sevoflurane	160

Note that isoflurane and halothane vaporise to almost the same extent.
The very high value for nitrous oxide is because it is a gas at room temperature and atmospheric pressure *ie* it has totally vaporised. Nitrous oxide is marketed as a liquid under high pressures in steel cylinders.

From this data, you can calculate the concentration attainable for these anaesthetics.

> Eg   Isoflurane Saturated Vapour Pressure at 20°C = 250 mmHg
> Atmospheric pressure at 20°C = 760mmHg
> Of 100% gas, isoflurane will be 250 ÷760 × 100% ≈ 33%

HOW DO I CONVERT LIQUID TO VAPOUR??

A volatile anaesthetic is purchased as a liquid and delivered to the patient as a vapour. It is useful to know how much anaesthetic vapour will be produced from one ml of liquid. You need this information if you wish to calculate what an inhalation anaesthetic costs.

	Halothane	Isoflurane	Sevoflurane	Desflurane
ml vapour from one ml liquid @ 20°C, 760 mm Hg	**227**	**194.7**	**182.7**	**209.7**

HOW TO USE SOLUBILITIES, MAC'S & SATURATED VAPOUR PRESSURES.

Methoxyflurane enjoyed some popularity but is rarely used now for many reasons. It is possible to work out from the data presented in this chapter some of the reasons for isoflurane being preferred over methoxyflurane by most practitioners

	Blood/gas solubility	Oil/gas solubility	MAC %	Saturated Vapour Pressure (mmHg)
Methoxyflurane	13	635	0.23	22.5
Isoflurane	1.4	91	1.28	251

Blood/gas solubility.Methoxyflurane is much more soluble in blood than isoflurane. It will take longer for an effective blood concentration to be reached. This means slow inductions and wake-ups.

Oil/gas solubility.   There is a high correlation between fat solubility and potency. This is supported by this data. Methoxyflurane has a very high oil/gas solubility coefficient and very high potency (low MAC) compared with isoflurane. This, on its own, is not a problem.

Saturated vapour pressure and **MAC**.   Methoxyflurane may be more potent than isoflurane, but it has a very low SVP. The SVP of methoxyflurane is about 13 $\times$ MAC (3/0.23). The achievable concentration is 22.5 $\div$ 760 $\times$ 100% = 3% and the MAC is 0.23%.

The SVP of isoflurane is about 26 $\times$ MAC (33/1.28). Isoflurane is still very potent (MAC < 1.3%), and it will be easy to deliver a high enough concentration to achieve effective alveolar/blood concentrations. The achievable concentration of isoflurane at 20°C and 760mm Hg is 33% and the MAC is 1.28%.

Summary: -

- o With a low achievable concentration wrt MAC and huge loss of methoxyflurane to the blood and tissues, the rate at which an alveolar concentration that would cause anaesthesia could be achieved was very slow. Isoflurane has a much higher achievable concentration and is much less blood and tissue soluble – hence faster inductions than with methoxyflurane.
- o The differences between the SVP's and the MAC's shows why these two drugs require different vaporizers.
- o One reason methoxyflurane lost favour was because it took a very long time to get the patient asleep, and to wake them up. Other reasons include the fact that it is nephrotoxic and that about 50% of absorbed methoxyflurane is metabolised (compared with 0.17% isoflurane).
- o Methoxyflurane is still used as an analgesic in human patients. Ambulance officers carry small vials and apparatus for patient controlled delivery. Induction is so slow that the patient is most unlikely to become anaesthetized but methoxyflurane is a very good analgesic at sub-anaesthetic doses.

HOW TO COST ANAESTHESIA USING

VOLATILE AGENTS.

Consider the choice between isoflurane and sevoflurane delivered in oxygen via a circle absorber with the vaporiser out of circle.

Let's say the O_2 flow rate is set at 2 l/min, and the required duration of anaesthesia is 60 minutes.

The MAC for sevoflurane is higher than that of isoflurane, so for equivalent anaesthesia, the sevoflurane vaporizer will be set at a higher percentage.

MAC values (dog)		1.3×MAC
Isoflurane	1.3%	1.7%
Sevoflurane	2.25%	2.9%

O_2 delivered = 2 l/min×60min = 120 l = 120,000 mls

Isoflurane (vaporizer set on 1.7%)
1.7% of 120,000 = 2040 mls of vapour
1 ml liquid = 196 ml of vapour at 20 ºC
2040 ml vapour = 2040/196 ml liquid = 10.4 ml
250 ml isoflurane costs $54 (Feb 2015)
∴This anaesthetic uses $2.25 of isoflurane

Sevoflurane (vaporizer set on 2.9%)
2.9% of 120,000 = 3480mls of vapour
1ml liquid = 182.7mls of vapour at 20°C
3480mls vapour = 3480/182.7ml liquid = 19mls
250ml sevoflurane costs $280 (Feb 2015)
∴This anaesthetic uses $21.28 of sevoflurane

NITROUS OXIDE   (N$_2$O   MW = 44)

Nitrous oxide is a weak anaesthetic. In man, the MAC is > 100%. (dog > 200%) Therefore, it cannot be used as a sole agent but is used at sub-anaesthetic concentrations in combination with other agents to provide *analgesia*. It is usually used in combination with oxygen (70% → 50% N$_2$O & 30% → 50% O$_2$), as carrier gases for the delivery of a volatile anaesthetic agent.

Nitrous oxide is a gas under atmospheric pressure at room temperature. (Boiling point is minus 89°C at atmospheric pressure, 760 mm Hg.) It is stored in blue cylinders at 30 – 50 atmospheres of pressure. At these pressures, nitrous oxide is a colourless liquid with saturated vapour in the available space above the surface of the liquid. *The pressure gauge on the top of the cylinder will not start to fall until all the liquid has vaporised, and the cylinder is virtually empty, so the gauge is of little use in letting you know the contents of the bottle.*

Absorption, fate and elimination. N$_2$O *rapidly crosses the alveolar membranes* and equilibrates with the blood (low blood gas solubility) where it is carried in *simple solution*. *It rapidly diffuses into gas filled cavities* in the body. (Discussed below). It is rapidly and completely *eliminated through the lungs* at the end of the anaesthetic.

Side effects   Provided oxygenation is ensured by delivering a *minimum of 30% oxygen* when nitrous oxide is used, there are *few deleterious effects* on CNS, cardiovascular, respiratory and the gastrointestinal (GIT) systems. Prolonged exposure can result in *bone marrow depression*. Patients with cardiac or respiratory disease may require a minimum of 50% inspired oxygen to ensure adequate oxygenation of the tissues.

The high concentrations of nitrous oxide used and the fact that it has low blood and tissue solubility can result in problems during anaesthesia.

·   ***Nitrous oxide diffuses into gas filled body cavities.***

Gas-filled cavities in the body exist in physiological states eg GIT (especially in herbivores) as well as pathological states eg pneumothorax, gastric dilation and torsion.

Administration of N$_2$O to patients with gas-filled cavities can cause problems. Prior to connecting a patient to an anaesthetic machine, the blood, tissues and gas filled spaces are at equilibrium with air (79% N$_2$). Once the inspired gas changes to contain 0% N$_2$ and 70% N$_2$O, a larger volume of N$_2$O diffuses in than N$_2$ diffuses out. This is because N$_2$O is about 33 times as soluble as N$_2$. This leads to rapid expansion of the gas filled spaces.

In the case of GIT gas, the expansion can further compromise perfusion of the gut wall.   In the case of pneumothorax, the free gas in the pleural space will be greatly increased and respiration will be further compromised.

The differential solubilities of N$_2$ and N$_2$O can be taken advantage of when closing the chest wall at the end of a thoracotomy. At closure, there is always some gas trapped in the pleural space. Because of rapid diffusion from the tissues, some of this gas will be N$_2$O. Once the inspired N$_2$O is returned to 0%, diffusion gradients will result in N$_2$O rapidly diffusing from pleural space to blood to alveolus hence reducing the residual pneumothorax.

·   ***Diffusion hypoxia***

When N$_2$O is turned off at the end of anaesthesia, N$_2$O will rapidly diffuse into the lungs from the blood and body tissues. If the patient is disconnected from the anaesthetic machine and allowed to breathe room air, the volume of N$_2$O entering the lungs may be sufficient to result in O$_2$ concentrations low enough to cause the patient to become hypoxic ie nitrous oxide will displace oxygen. This can be avoided by leaving the patient connected to the anaesthetic machine and breathing a high concentration of O$_2$ for 5 – 10 minutes after the N$_2$O has been turned off, to ensure that alveolar O$_2$ concentrations remain adequate for oxygenation.

·   ***Second Gas Effect***

Consider a patient breathing room air, before being connected to a patient breathing circuit (eg a circle absorber) delivering isoflurane in nitrous oxide and oxygen.

Remember that the *alveolar* gases are not the same as the inspired gases (those delivered to the patient) as inspired gases are humidified and mixed with dead space gases. Water

vapour will be about 6 %, oxygen about 14 %, CO_2 about 5.6 % leaving about 74.4 % nitrogen. The gases dissolved in the blood will not be in equilibrium with the alveolar gases, but they will be close.

When the delivered gases change to, say, 29 % oxygen, 70 % N_2O and 1 % isoflurane but 0 % N_2, the alveolar gases will change.

There will be large partial pressure gradients across the alveolar wall – N_2 from blood to alveolus and N_2O from alveolus to blood.

These gases all have *unique solubilities* in blood and tissues. *Low solubility means fast equilibration between gas and liquid phase* ie between alveolus and blood.

The gases present in *highest concentrations* will most effect the volumes present
ie N_2 and N_2O

Both of these gases have low solubility in both blood and tissues, but N_2O is about 33 times as soluble as N_2. (N_2O - 0.47ml/ml blood, N_2 - 0.014ml/ml blood)

Therefore, the volume of N_2O leaving the alveoli and entering the blood will be much greater than the volume of N_2 returning to the alveoli from the blood.
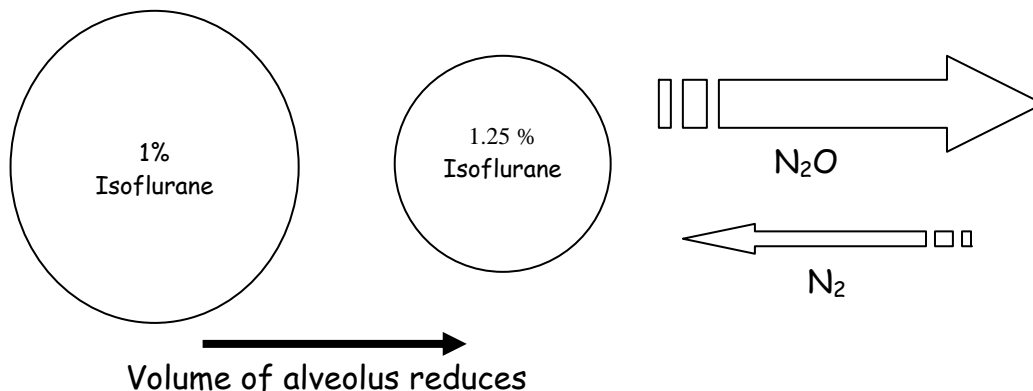
In the short term, the volume of gas in the alveolus will diminish. If the alveolar concentration of isoflurane is 1%, then this means one part in 100 parts. When the volume of the alveolus diminishes because of N_2O rapidly diffusing out of the alveolus, then isoflurane will be more concentrated eg one part in 80 ie 1.25%

This increase in the alveolar concentration of isoflurane increases the concentration gradient between the alveolus and the blood. Hence, induction of anaesthesia will be more rapid.

This can be used to assist in achieving anaesthetic levels of vapour in the patient. In other words, the quick disappearance of N_2O from the lungs into the blood creates a greater concentration differential of anaesthetic vapour increasing the speed of its uptake into the blood and eventually the brain.

Conversely, turning the N_2O off at the end of a procedure will assist in reducing the body content of anaesthetic vapour.

HALOTHANE

Halothane is a halogenated hydrocarbon, with a molecular weight of 197.4.   At room temperature it is a colourless liquid with a sweet, non-irritant odour. Halothane is stabilised by the addition of 0.01% thymol.   *As halothane evaporates, thymol builds up in the vaporiser. Regular servicing and cleaning of vaporisers is recommended.*

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        Br    F
         |    |
H  –  C  –  C  -  F
         |    |
        Cl    F
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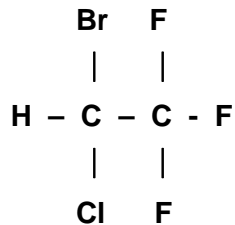
Halothane is a *potent* inhalation anaesthetic (MAC value for dogs = 0.87 %). It is easily vaporised, with a *saturated vapour pressure at 20°C of 243 mmHg*. This translates to an achievable concentration of $(243 \div 760) \times 100\% = 32\%$. This is much higher than the MAC and the concentration must be reduced from saturated vapour to allow safe anaesthesia. (An alveolar concentration of 3 % for 3 minutes can be lethal!)

Halothane has a *low blood/gas solubility coefficient*. Therefore it reaches anaesthetic levels in the brain quickly. Oil/gas solubility is 220, consistent with halothane being a highly potent agent. (Remember that there is a very strong positive correlation between potency and oil/gas solubility coefficient. High potency (low MAC) ⇔ high oil/gas solubility coefficient.)

The rubber/gas solubility is 120. This means that the uptake of anaesthetic into the rubber tubes of the anaesthetic equipment will cause some delay in achieving alveolar concentration. Conversely, when the vaporizer is turned off, halothane coming out of the rubber has the effect of maintaining the inspired concentration. These effects are relatively minor.

Halothane causes *dose related cardiovascular depression*.   The main effect is *hypotension* due to myocardial depression which results in *reduced cardiac output, stroke volume* and *myocardial contractility*. *Vasodilatation* further contributes to hypotension. *Bradycardia* can occur, even during the presence of hypotension. Halothane sensitises the myocardium to the effects of catecholamines, and *dysrhythmias* may occur.

Dose related respiratory depression, primarily *reduced tidal volume*, results in *diminished alveolar ventilation*.

Halothane induced *hepatitis* has been recorded in human patients. Ten to twenty five percent of absorbed halothane is metabolised in the liver, and this may be significant in the development of hepatitis. The medicolegal aspects of post-halothane hepatitis have resulted in a swing to newer agents in human patients. In dogs, horses and sheep, anaesthetised for long periods with halothane, minimal pathological changes have been observed in the liver and kidney. Both halothane and isoflurane have been widely used in veterinary anaesthesia. The cost of isoflurane continues to fall, and it is now the most widely used inhalation anaesthetic drug in veterinary practice.

Halothane can induce malignant hyperthermia, particularly in certain breeds of swine. Eg Landrace, Large White, Poland-China and Pietrain. This has been used extensively in research into malignant hyperthermia.

It is tempting to consider that halothane is a safe anaesthetic because its use has been so widespread, but one should not underestimate its potency. If a human patient expires maximally, then inspires maximally 4 % halothane, they will lose consciousness in one breath. Halothane must be administered *only via a vaporizer designed for halothane*. As a

concentration of 32 % is achievable, administration of halothane with crude apparatus can be lethal.

ISOFLURANE

Isoflurane is a halogenated ethyl methyl ether, an isomer of enflurane. Molecular weight is 184.5. At room temperature, it is colourless liquid with a pleasant fruity smell. It does not require a preservative and does not react with soda lime.

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      F   Cl        F
      |   |         |
F – C – C – O – C – H
      |   |         |
      F   H         F
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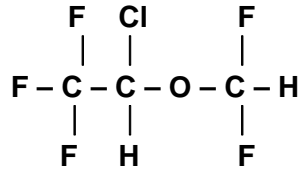
Isoflurane is a *potent* agent, though it has slightly lower potency than halothane (Isoflurane MAC 1.3%, Halothane 0.87%). Saturated vapour pressure (250 mm Hg) is almost identical to that of halothane. Achievable concentration is 33%.

Isoflurane has *low blood/gas solubility* (1.4 cf halothane at 2.4) and therefore reaches anaesthetic levels in the brain quicker than halothane. Oil/gas solubility is 91.

Isoflurane causes *dose-related vasodilatation and decrease in blood pressure*. There is little effect on cardiac contractility. Overall, cardiovascular stability is better with isoflurane that with halothane. Isoflurane is a more *powerful respiratory depressant* than halothane. This effect is evidenced by an *increase in pCO$_2$*. Isoflurane does not provide analgesia.

There is very little metabolism of isoflurane (approximately 0.2%). This fact, along with low blood solubility of isoflurane, results in a *fast and complete wakeup*. Isoflurane is widely used in veterinary anaesthesia.

SEVOFLURANE

Sevoflurane is a fluorinated methyl-isopropyl ether. Its blood gas partition coefficient is 0.6, lower than isoflurane (1.4) Thus, induction and recovery are faster than with the other available ethers. Cardiovascular stability is better than with isoflurane. It does not sensitise the myocardium to the effects of adrenaline. It is non-irritant to the respiratory tract, but causes respiratory depression. There is some metabolism of sevoflurane by the liver (3%) releasing fluoride ions which are associated with nephrotoxicity. As the degree to which sevoflurane is metabolized is very low, this is unlikely to result in a clinical problem. There is some breakdown in the presence of soda lime to produce a nephrotoxic alkene known as Compound A. The significance of this is not yet well understood. This is not considered to be a problem in humans with a fresh gas flow rate as low as 1 l/min. However, the range of concentration of Compound A measured in breathing systems of human patients includes the threshold for nephrotoxicity in rats. As yet, there is no relevant published data for dogs, but preliminary investigations conducted in a low flow breathing system indicate that investigation is warranted. It must be remembered that, in high flow breathing systems, Compound A build-up is unlikely as there is little rebreathing of exhaled gases.

MAC values: -

Cat	Dog	Horse	Rabbit	Rat
2.58	2.36, 2.10	2.31	3.70	2.40, 2.50

DESFLURANE

Desflurane is identical in structure to isoflurane, except for a fluorine atom replacing the chlorine atom on isoflurane. Blood gas partition coefficient is 0.42, resulting in *very* fast

inductions and wakeups. It causes dose dependent vasodilatation and a drop in blood pressure, but cardiac output is maintained without tachycardia. Overall, cardiovascular stability achieved is very good. Respiratory depression is more severe with desflurane than with the other commonly used agents.. Desflurane is stable in the presence of soda lime. It is metabolized by the liver to the extent of 0.02% - this makes the likelihood of hepatotoxicity very low.

The MAC is about 7.2% in dogs, but the achievable concentration is very high. (SVP at 20°C = 700mm Hg, ≡ achievable concentration of about 90%) Boiling point is very low (23.5ºC). As with all the currently used volatile anaesthetic agents, special calibrated vaporizers are needed to enable delivery of predictable concentrations to the patient.

ENFLURANE

Enflurane is a halogenated ethyl-methyl ether, an isomer of isoflurane. It is a potent anaesthetic (MAC = 1.68%), with low blood/gas solubility (1.9).   Saturated vapour pressure at 20°C is 171.8 mm Hg. Enflurane causes dose related cardiovascular and respiratory depression. Enflurane differs from the other volatile anaesthetics in that it causes marked, dose related CNS stimulation. Tonic clonic convulsions have been observed frequently. Seizures up to one week after enflurane anaesthesia have been reported.

Occurrence of CNS stimulation and cost of enflurane have seen it lose popularity in favour of newer agents.

METHOXYFLURANE

Methoxyflurane is a halogenated methyl-ethyl ether. It has a fruity smell, but is non-irritant to the airways. It is a very potent anaesthetic (MAC = 0.16%), but has very low saturated vapour pressure (25mm Hg ≡ maximum achievable concentration of 3%) and is very soluble in blood. Although the required alveolar concentration for anaesthesia is low, it is difficult to achieve, because of its low saturated vapour pressure and high blood solubility.

Methoxyflurane is a very powerful analgesic, with analgesia persisting for 24 hours after the patient awakes. The drug is highly metabolised (up to 50%). Metabolism results in release of fluoride ions, causing renal damage.

Methoxyflurane is still in use as an analgesic drug at sub-anaesthetic concentrations.

	Halothane	Isoflurane	Sevoflurane	Desflurane
CV stability	+	++	+++	++
Resp stability	–	--	----	--
Analgesia	No	No	?	?
Fast induction	+	++	+	+++
Metabolism	25%	0.17%	3%	0.02%
Soda lime stability	Yes	Yes	No?	Yes

INTERMITTENT POSITIVE PRESSURE VENTILATION (IPPV)

Adequate spontaneous ventilation depends on *muscular effort* to expand the thoracic cavity, sufficient *functional lung tissue* and *negative pressure in the interpleural space* to keep the lungs expanded. There are times when it essential or beneficial for the anaesthetist to ventilate the patient's lungs by intermittently applying positive pressure to the patient breathing circuit. This is called intermittent positive pressure ventilation (IPPV).

Some examples of clear indications for (IPPV) in veterinary patients under general anaesthesia are:–

* *Depressed respiratory function* due to *anaesthetic* or *analgesic* drugs administered.
* Administration of *muscle paralysing drugs.*
* *Collapse of the lungs* due to pneumothorax, haemothorax, penetrating chest wounds etc
* *Thoracotomy*

Clearly, if the patient cannot spontaneously and adequately ventilate its lungs, the anaesthetist must apply IPPV.

There are important *differences between spontaneous ventilation and intermittent positive pressure ventilation.* The *effect of IPPV on cardiac* function is detrimental.
During the *inspiratory phase of the respiratory cycle in spontaneous ventilation*, expansion of the chest wall and deflection of the diaphragm cause **negative pressure** in the thorax. As there is a slight negative pressure in the pleural space, the lungs follow the chest wall and expand. Thus, *air flows passively along the pressure gradient* into the lungs. During this phase, v*enous return is enhanced* by the increase in negative pressure in the thoracic cavity, and therefore the *stroke volume is increased* for a short time during the inspiratory phase of spontaneous ventilation.

During the *inspiratory phase of IPPV, air is forced into the lungs under* **positive pressure**. This positive intra-thoracic pressure *decreases the venous return*, and therefore *stroke volume and cardiac output*. This reduction of cardiac output can have serious implications, especially if the patient already suffers cardiac compromise or if blood pressure is reduced due to the effect of anaesthetic drugs. Also, positive pressure in the thorax has a detrimental effect on pulmonary perfusion.

The effect on venous return and therefore cardiac output and pulmonary perfusion can be kept to a minimum if *ventilation pressures are kept as low as possible*. This can be achieved by *paralysing the skeletal muscles, thereby lowering muscle tone and facilitating expansion of the thorax*. Ventilation pressures of between *12 and 20 cm H_2O* are usually satisfactory. If a decrease in measured blood pressure or in pulse amplitude can be detected during the inspiratory phase in IPPV, the pressure should be reduced. If this is the case, it may be appropriate to *increase the rate* of ventilation rather than the pressure to achieve adequate ventilation.

Capnography (measurement of the CO_2 levels in the respiratory gases) is a valuable adjunct to IPPV. End tidal CO_2 is related to pulmonary arterial CO_2. Therefore if end tidal CO_2 levels can be measured, IPPV rate and inspiratory pressure can be adjusted to maintain normocapnia (about 40 mm Hg).

An **endotracheal tube** (ETT) with the *cuff inflated to properly seal the trachea* is **essential** for safe IPPV.

That the ETT <u>seals the trachea must be tested</u> before a muscle paralysing drug is administered or before the thorax is opened. The anaesthetist must ensure that inflation pressures can be achieved.

If thoracic surgery *is undertaken, or if* muscle paralyzing drugs are to be administered, an **anaesthetist must be dedicated to ventilation of the lungs until the patient is demonstrated to be capable of adequate spontaneous ventilation**.

Before embarking on anaesthesia of <u>any patient</u>, the anaesthetist must be able to secure an airway and provide IPPV should it be necessary.

INJECTABLE DRUGS

PREMEDICATION

PHILOSOPHY

Premedicant drugs can serve many purposes. Drugs and dose rates carefully chosen for a particular patient and particular procedure can minimise risk to both the patient and the operator. **The main purposes include**

- Aid to *restraint*
- Reduce patient *stress/anxiety*
- Provide *analgesia*
- Provide *muscle relaxation (NOT paralysis)*
- *Potentiate* the actions of induction and maintenance agents, thereby *reducing the dose required and the side effects suffered*
- *"Smooth" induction, maintenance and recovery*
- *Obtund* some potentially detrimental *reflexes*

ANTICHOLINERGICS

eg   atropine, glycopyrrolate

The main reasons for using these drugs prior to or during general anaesthesia are

- to *reduce the glandular secretions* of the respiratory tract and gastrointestinal tract, including those of the oral and nasal cavities. (This was a big problem with *ether* which is no longer used)
- to *inhibit vagal tone* and therefore counteract bradycardia resulting from vagal stimulation. Some drugs have a vagotonic effect (eg acepromazine) or sympatholytic effect (eg halothane, isoflurane), and some procedures have inherent risk of vagal stimulation eg procedures involving handling structures of head, neck, thoracic or abdominal viscera.

Glycopyrrolate is a quaternary ammonium compound and does not cross the blood brain barrier. Therefore, it *does not produce CNS effects and cannot cross the placental barrier*. Its effects on the heart are increase in sinus activity, AV node conductivity and atrial contractility. Overdose causes tachycardia and other peripheral effects, such as excessively dry mouth.

Atropine is a muscarinic antagonist. As for glycopyrrolate, atropine causes an increase in sinus activity, AV node conductivity and atrial contractility. Atropine has marked effects on the gastro intestinal tract causing reduced secretory activity and motility. It does cross the blood brain barrier and can affect the CNS. Therefore, overdose results in CNS effects ranging from seizures to mild sedation. Vomition may be reduced. Atropine produces long lasting mydriasis and cycloplegia. Lacrymation and salivation are suppressed.

Route   Both drugs can be administered by SC, IM or IV injection.

		Dose Rate	
Atropine	Cats and Dogs	0.02 – 0.04mg/kg	Duration 60 – 90 min
	Horses	not routinely used ($\downarrow$ gut peristalsis, colic?)	
	Ruminants	not recommended (viscous secretions)	
	Pigs	0.04 – 0.08 mg/kg	
Glycopyrrolate	Cats and Dogs	5 – 10 ug/kg	Duration 2 – 4 hours
	Horses	2.5 – 5 ug/kg ($\downarrow$ gut peristalsis, colic?)	
	Ruminants	not recommended	
	Pigs	3 ug/kg	

SEDATIVES AND TRANQUILIZERS

Sedatives are drugs that relieve tension and anxiety, thereby producing and promoting sleep. These drugs more readily produce sleep in sufficient doses than do tranquillisers.
Tranquillisers = ataractics = neuroleptics. These are drugs that relieve tension and anxiety without undue sedation. These drugs are better anxiolytics than the sedatives.

SEDATIVES
α_2-adrenergic agonists   eg Xylazine, detomidine, medetomidine and romifidine
Mode of action.   There are three distinct α_2 adrenoreceptor sub-types – A, B and C.
α_2-A receptors are responsible for sedation, supraspinal analgesia, and centrally mediated bradycardia and hypotension. These are presynaptic inhibitory receptors found primarily in the cortex and locus caeruleus and in platelets.
α_2-B receptors increased vascular resistance causing reflex bradycardia. These are probably postsynaptic receptors (Maddison et al, 2002). Found in the dorsal root ganglia and vascular endothelium.
α_2-C receptors mediate the hypothermic response. Found in the dorsal root ganglion.

CNS depression results from stimulation of presynaptic α_2-adrenoreceptors in the CNS (α_2-A). These receptors are inhibitory. Stimulation of these receptors results in a reduction of noradrenalin release centrally and peripherally, and therefore decrease in CNS sympathetic outflow and a decrease in circulating catecholamines. Many of the *side effects* of the *α_2-adrenoreceptor agonists* are due to *poor receptor selectivity. There is a degree of* stimulation of post synaptic *α_2-adrenoreceptors* (α_2-B & C) and stimulation of *α_1-adrenoreceptors eg in blood vessel walls.*

New drugs have been designed for *α_2 > α_1 selectivity* in an attempt to decrease side effects.

Drug	$\alpha_2 : \alpha_1$ selectivity
Xylazine	*160 : 1*
Detomidine	*260 : 1*
Romifidine	*340:1*
Medetomidine	*1620 : 1*

Duration of action.	Dogs	Horses
Xylazine	*30 – 40 min*	*30 – 60 min*
Detomidine		*60 min*
Romifidine	*60 – 120 min*	*40 – 80 min*
Medetomidine	*60 – 120 min*	

Alpha$_2$-adrenergic agonists can be administered into the *epidural* and *subarachnoid* spaces to produce regional or segmental analgesia.

Effects of α-adrenoreceptor stimulation
CNS	Sedation, analgesia, hypotension, bradycardia
	Nausea/vomiting (cats and dogs)
CVS	Peripheral vasoconstriction initial hypertension, *profound reflex bradycardia*.
	Sinoatrial block, atrioventricular block, 1st & 2nd degree block, AV dissociation (3rd degree block), sinus arrhythmia.
	Central bradycardia and vasomotor depression, hypotension
	Direct depression of the myocardium and vascular smooth muscle
Gut	Relaxation, decreased motility
Salivation	Increased

Gastric secretion   Reduced
Uterus   Stimulation
Eyes   Mydriasis, decreased intraoccular pressure
Hormones   Reduced release of insulin ($\rightarrow$ hyperglycaemia), renin and ADH
Platelets   Aggregation
(Livingstone, 1969 as cited in Hall and Clarke,1991, Muir et al 1995, Thurmon et al, 1996)

It must be remembered that the **ruminants are extremely sensitive to the α_2-agonists** and therefore the recommended dose rates are much smaller than those for cats, dogs and horses (about 1/10).

PREMEDICATION WITH XYLAZINE IN ANY SPECIES DRASTICALLY REDUCES THE AMOUNT OF ANAESTHETIC AGENT REQUIRED. Failure to account for this reduced need can result in death of the patient. For example, expect to *cut the estimated dose of induction* agent eg thiopentone *by half* following xylazine premedication. Reduced cardiac output results in longer time to effect of IV drugs – 'arm to brain' time is increased eg from 20 – 30 sec to 90 sec). Hence, titration must be slow to account for this.

Xylazine can cause relaxation of the retractor muscle in male horses but there have been no reports of permanent penile paralysis as with acepromazine (Plumb 2008).

The α_2-adrenergic agonists are extremely *useful in large animals*, where rendering the animal tractable is very important for the safety of both patient and veterinarian. However in cats and dogs, the high incidence of nausea and vomiting, disturbance of cardiac rhythm, effects on blood pressure and the availability of suitable alternatives make these drugs less appealing.

Alpha$_2$ adrenergic agonist administration in all species induces a *loss of body temperature control*. It is imperative that the surroundings are appropriate when patients (especially large animals in the field) are left to recover from xylazine sedation. (Shade in summer, warmth in winter)

Table 1.   Dosages of several α_2-adrenergic agonists in several domestic species. Lower doses IV, higher doses IM. (Tranquilli and Maze, 1993)

Species	Xylazine (μg/kg)	Detomidine (μg/kg)	Medetomidine (μg/kg)	Romifidine (μg/kg)
Cat	200 – 2000	NA	40 - 80	NA
Dog	200 – 2000	5 – 20	5 – 40	NA
Horse	200 – 1000	10 – 40	10 – 30	30 – 80
Cattle	20 – 200	30 – 60	20 – 50	NA
Pig	2000 - 4000	NA	30 - 80	NA

α_2-adrenergic antagonists   Yohimbine (xylazine), atipamizole (medetomidine), tolazoline and idazoxan are of use in the reversal of the effects of the α_2-adrenergic agonists, but care must be taken as it is not certain that all the undesirable side effects are reversed equally.   Antagonists should be given before the sedative effect of the α_2-adrenergic agonists has worn off, or CNS excitement can occur (tremors, convulsions).

Convulsions resulting in death of the animal have been reported in adult cattle. Mostly, cases in which administration of α_2-adrenergic antagonists has resulted in convulsions have involved the anaesthetic drug ketamine, which may have been responsible for the convulsions once the muscle relaxing effect of the α_2-adrenergic agonist was removed.

Benzodiazepines (eg diazepam, midazolam, zolazepam)
Indications for the use of the benzodiazepines as premedicants
   - *Sedation/anxiolysis* – doubtful efficacy in dogs when used as sole agent
   - *Muscle relaxation* – of use with muscle spasm associated with painful fractures, spinal lesions
   - *Reduction of seizure activity*

The mode of action is through stimulation of *specific benzodiazepine receptors* (BZ), which then potentiate the inhibitory action of the transmitter, γ-aminobutyric acid (GABA). GABA acts on the chloride channel, increasing the flow of chloride ions into the cell, thereby causing hyperpolarisation, making it refractory to other stimuli. There are different types of BZ receptors, which explains differences between different BZ receptor agonists.

Diazepam (Valium®, Pamlin®)   Diazepam is the most widely used of the benzodiazepines. It is *insoluble* in water. Therefore, solutions contain solvents such as propylene glycol, ethanol and sodium benzoate in benzoic acid. That IV injections can be painful is thought to be due to the solvents not the drug itself. A newer preparation has recently become available that is based on an emulsion. Care must be taken in mixing drugs of differing water solubilities.

Diazepam, on its own, *does not seem to have sedative effects in dogs*. Dogs given diazepam as a sole premedicant often seem more anxious rather than less. It appears that the muscle relaxation (via internuncial neurones in the spinal cord) renders the animal less able to control its limbs and this leads to distress in the absence of anxiolysis. Diazepam has very *few side effects at clinical doses and very low toxicity*.

The major uses are

- The control of *convulsions*. *Status epilepticus* can be relieved in dogs by a slow IV injection of (0.5 - 1mg/kg) which may be repeated if necessary. It can also be used in dogs prone to seizures as a premedicant, or to lessen the risk of convulsions eg in dogs undergoing radiography with injection of contrast medium into the subarachnoid space.
- In *combination with the dissociative anaesthetic*, ketamine.

Diazepam	Dog & cat	0.1 – 0.5 mg/kg IV, IM
	Horse	0.02 – 0.04 mg/kg IV (ataxia can be a problem)
	Sheep	0.3 mg/kg

Midazolam (Hypnovel®)

Midazolam is water soluble in the injectable form, but is lipid soluble at body pH. Consequently, it has a much faster onset of action than diazepam. Midazolam is primarily liver metabolised, is highly protein bound and has a much shorter duration of action than diazepam.

Midazolam	Dog & cat	0.1 – 0.5 mg/kg IV, IM, SC
	Horse	0.02 – 0.04 mg/kg IV (ataxia can be a problem)
	Sheep	0.3 mg/kg

TRANQUILLIZERS
Phenothiazine derivatives

These drugs are *dopamine antagonists*. They give rise to *calming and mood-altering effects* and they have powerful *anti-emetic* action. They also have peripheral effects due to α_1-adrenoceptor antagonism. This explains most of the side effects:-

- Marked *hypotension*   due primarily to peripheral vasodilatation
- *Antiarrhythmic* effects on the heart   blocking cardiac α-adrenergic receptors
- *Antihistamine*
- *Loss of body T°*   dilated cutaneous vessels and central effect

Acepromazine   Acepromazine is probably the most *widely used* tranquilliser in veterinary practice. Good tranquillisation can be achieved in *most species*. It is important to note that there is a ceiling effect on sedation and increasing the dose will not increase sedation but will worsen the side effects. Profound hypotension may occur if acepromazine is given at large doses.

Contraindications: - Acepromazine administration can result in: -

- Profound *hypotension* in animals with hypovolaemic shock
- *Priapism or flaccid protrusion of the penis* with subsequent injury can occur in horses. **Don't use in breeding stallions**.

- *Syncopal attacks in Boxer dogs* (Acepromazine is vagotonic and Boxers have high resting vagal tone)

Dogs & cats   0.01 – 0.05 mg/kg IV
                        0.03 – 0.1 mg/kg SC
Horse   0.02 – 0.05 mg/kg IV/IM

Butyrophenones
These drugs have fallen from favour in people as they cause hallucinations, restlessness, agitation and even aggression.   These side effects are not always obvious to the observer but are reported by the patient after recovery. It is not possible for us to know whether our animal patients suffer similarly.
The mode of action and profile of activity of the butryophenones are similar to those of the phenothiazine derivatives. They are *dopamine antagonists*. The *cardiovascular effects are minimal*, but there may be slight hypotension resulting from α-adrenergic block.
Droperidol   Droperidol is the butyrophenone most often encountered in small animal veterinary practice, and that is usually in combination with an opioid eg fentanyl.
           Dog & cat   0.1 – 0.2 mg/kg IV
                                0.2 – 0.5 mg/kg SC
Azaperone   Azaperone is marketed as Stresnil® for use in *pigs*. IM injection results in good dose related sedation.
           Pig   1 – 2 mg/kg (low doses for large pigs) IM

OPIOID AGONISTS (NARCOTIC ANALGESICS)
Definitions: -
1) *Agonist* - the drug binds to the receptor, producing maximum stimulation at that receptor. All effects are dose dependent. *(eg morphine, fentanyl)*
2) *Antagonist* - the drug binds to the receptor producing no stimulation, but effectively blocking the receptor to other opioids. *(eg naloxone)*
3) *Partial agonist* - the drug binds to the receptor but produces only weak stimulation thus achieving less than the maximum effect. This is called a 'ceiling effect'. *(eg buprenorphine)*
4) *Agonist/antagonist* – have agonist or partial agonist activity at one or more types of opioid receptors, have the ability to antagonise the effects of an agonist at one or more types of opioid receptors. *(eg nalorphine, butorphanol)*

Opioids are used as analgesics as well as premedicants
- To provide *analgesia* in the peri-operative period.
- To *reduce the required dose of sedatives/tranquillisers, induction and maintenance agents.*
Opioids can be used as premedicants in patients suffering pain (eg GA for painful fracture fixation), or as pre-emptive analgesics (patient pain likely to be increased or caused by surgery), or simply to reduce the requirements for other drugs in the perioperative period.
Side effects of the opioids: -
- *Respiratory depression*. Decreased respiratory rate and tidal volume, with a shift to the right and reduction of slope of the carbon dioxide dissociation curve are probably due to depression of medullary respiratory centre neurons. ie sensitivity to CO_2 is reduced. This can be lethal, especially in patients breathing room air. This is a feature of all pure μ-agonists, but is less so in mixed agonists/antagonists (eg nalbuphine) and partial agonists (eg buprenorphine). Respiratory depression is much less in patients that are in pain at the time of administration than in pain free patients.   **The use of opioid analgesics at clinical dose rates is rarely associated with serious respiratory depression.   However, there have been deaths of patients left on continuous rate infusions unsupervised over night. Supervision of patients is necessary.**

- Some opioids tend to cause *arterial and venous dilatation* (direct α-*adrenergic blocking* activity or *histamine release*). Morphine and pethidine have both effects but fentanyl has neither. *In particular, pethidine should not be used in patients with mast cell tumours because of the possibility of release of histamine from tumour cells.* **Pethidine should never be administered IV.** Fentanyl is a useful adjunct in patients with cardiovascular compromise, as long as positive pressure ventilation can be provided should respiratory depression cause problems. In the main, opioids are associated with good *cardiovascular stability* at clinical doses.
- All pure μ-opioid agonists *delay gastric emptying* and *decrease intestinal motility*. Clinically, this can result in constipation.
- Stimulation of the chemoreceptor trigger zone can result in *nausea and vomiting*. (common with morphine if the patient is pain free at administration)
- *Urine retention* can occur.
- *Euphoria or dysphoria* occur on occasions.

Opioid Receptor Antagonists

Naloxone is a pure opioid antagonist and can be used to relieve opioid over-dose symptoms when necessary. Its duration of action is very short, and more than one administration may be necessary if the duration of action of the agent to be reversed is greater than that of naloxone.

      Dog & cat   0.04 mg/kg IV, IM, SC

It is possible to carefully titrate naloxone against unwanted side effects to reduce side effects and maintain analgesia.

      Narcan® is 0.4 mg/ml naloxone.

      Take 0.1 - 0.25 ml Narcan® and dilute to 10 ml with saline. (cats & small dogs, 0.1 ml Narcan, large dogs 0.25 ml Narcan.)

Titrate 1 ml/min until undesirable side-effects have subsided. It is possible to relieve unwanted side-effects without obliterating the analgesic effects by careful titration.

Naltrexone is a longer acting pure antagonist than naloxone.
Nalorphine and *diprenorphine* are partial agonists that are used to reverse unwanted side effects.

Sedative (or Neuroleptic) - Narcotic Analgesic Combinations

When used as premedicants, opioids are usually combined with tranquillisers or sedatives. A low dose of both agents results in *synergism* of sedation and analgesia but not of side effects.

There are many combinations of drugs in use and some are marketed ready-mixed. The sedative effect of these combinations depends on the dose given, anything from mild sedation to a state in which a dog can be intubated without an induction agent. High doses may result in respiratory depression with the patient requiring positive pressure ventilation.

Etorphine - phenothiazine tranquilliser mixtures are marketed in some countries as Immobilon®, large and small animal formulations. Large animal Immobilon® (2.45mg etorphine and 10mg acepromazine per ml) is used extensively in the capture of wild animals. It can be used at a range of dose rates causing recumbency to standing sedation. Etorphine is not recommended for cats, large or small. Small animal Immobilon® is 0.074mg etorphine and 18mg methotrimeprazine per ml. Naloxone is the recommended reversal agent. The dose rate of naloxone in humans is 0.2 – 0.4mg/kg IV (this may be repeated as necessary). *Etorphine is a very powerful respiratory depressant* and **deaths of veterinarians** have occurred due to accidental injections or mucosal contact of Immobilon®. If etorphine is to be handled, it is essential that persons competent in administering drugs IV to people and ample supplies of naloxone accompany the veterinarian. (Immobilon® is not available in Australia except on application for special Health Dpt approval.)

At the University of Queensland small animals are commonly premedicated with a combination of methadone and acepromazine administered subcutaneously or intravenously. Morphine is frequently used in place of methadone, but it causes a high incidence of nausea and vomiting in pain free animals.

For dose rate suggestions, see Appendices 9 (horses), 11 & 12 (cats and dogs) plus the Chapter on species differences.

INTRAVENOUS ANAESTHETIC AGENTS

Characteristics of an ideal injectable anaesthetic: -
1   Physicochemical and pharmacokinetic
- *Water soluble*
- *Long shelf life*
- *Stable when exposed to light*
- *Small volume required for induction of anaesthesia*
2   Pharmacodynamics
- *Minimal individual variation*
- *High therapeutic ratio*
- *Onset within one vein to brain circulation time*
- *Short duration of action*
- *Inactivated to non-toxic metabolites*
- *Smooth emergence*
- *Absence of anaphylaxis*
- *Absence of histamine release*
3   Side effects
- *Absence of local toxicity*
- *No effect on vital organ function, except anaesthetically desirable CNS effects*
                                                                (Thurmon, Tranquilli and Benson, 1996)

Such an anaesthetic agent is not available currently. Therefore, we must choose from the available agents, selecting an agent that will best satisfy the needs of the individual patient.

BARBITURATES *(in veterinary use since the 1930's)*
The barbiturates in use as induction agents are classed as "ultra-short" acting barbiturates.
CNS effects. The barbiturates appear to act on all levels of the CNS, but are clinically useful because the cerebral cortex and reticular activation system are more sensitive to the depressant effects than the vital medullary centres.
The barbiturates have no significant analgesic action.
All barbiturates have anti-convulsant properties.
Cardiovascular system.   Normal hypnotic doses have little effect on the cardiovascular system. A slight fall in blood pressure and pulse rate may occur. Large doses cause direct depression of the vasomotor centre. Arrhythmias on induction are not uncommon.
Respiratory system. All barbiturates are respiratory depressants. (Cause a reduction in the sensitivity of the respiratory centre to CO_2), as well as muscle relaxation.
Thiopentone (eg Pentothal[®])   Thiopentone (a thiobarbiturate) has stood the test of time in that it has been in use since 1935, and is still used today.
Thiopentone is sold as a sterile powder, to be dissolved in sterile water for injection. It should be refrigerated after being made up. The solution is stable for several days, after which time the drug should be discarded. The 2.5% solution is at pH 10.5 – 11, being buffered by 6% sodium carbonate. This very high pH results in a highly irritant solution, suitable only for intravenous injection (where it is diluted rapidly in the blood). The more concentrated the solution, the more irritant it is. Should the solution accidentally be injected extravascularly, the tissue should be infused with 0.9% saline, or dilute lignocaine HCl. Untreated extravascular injection can result in tissue necrosis and slough.

About 70% of plasma thiopentone is bound to albumin, and only the remaining 30% in simple solution is active. Therefore, in patients with hypoalbuminaemia, lower doses may be required (more drug is free).

The onset of action is 20 – 30 seconds, one vein to brain circulation time. Duration of action is 10 – 15 minutes. Forty percent of dogs suffer arrhythmias on induction. Rapid injection can result in significant vasodilatation and hypotension.

Recovery from thiopentone anaesthesia is partly by redistribution from the vessel rich tissues to the muscles, and partly by metabolism in the liver. Neonates are deficient in the enzymes necessary to metabolise the thiobarbiturates. Therefore, *animals less than 12 weeks old* and animals presented for *caesarean section* are usually not given thiopentone. For the same reason, *sight hounds* (greyhounds, salukis, deerhounds etc) should not be given thiopentone.

Pentobarbitone sodium. (Nembutal®) Pentobarbitone is rarely used as an anaesthetic in veterinary practice. It is classed as a short acting barbiturate, but has a slower onset and longer recovery time than the ultra-short acting barbiturates (0.5 – 3 minutes and 1 – 2 hours respectively). Its veterinary use is limited to treatment of *status epilepticus and laboratory animal anaesthesia. Pentibarbitone has recently become available again in a form suitable for use as an anesthetic after having been unprocurable for years except as a highly concentrated solution formulated for use in euthanasing animals.*

PROPOFOL Propofol, 2,6-diisopropylphenol, is unrelated to other classes of intravenous agents. It is almost *insoluble in water* and is, therefore, presented in a soya bean oil emulsion that is very similar in composition to the intravenous feeding solution, Intralipid®. This formulation is opaque, milky. This medium will support *bacterial growth* if contaminated. Therefore, *it is imperative that sterile methods are used to handle the drug, which should not be stored once opened.* An aqueous formulation is now available, but this formulation is associated with anaphylactoid reactions.

Induction of anaesthesia with propofol is *comparable to that with thiopentone*. Recovery is due partly to *redistribution* from the brain to other tissues and partly to *metabolism*. Metabolism is faster than it would be by the liver alone, indicating that other tissues contribute to its breakdown. One site of extra-hepatic metabolism is the lung parenchyma. *Duration* of anaesthesia in cats and dogs is *20 – 30 minutes* (awake, minimal ataxia). The incidence of post-anaesthetic side effects (pawing, sneezing and vomiting) is about 15% in unpremedicated patients. The dose required is *reduced by the prior administration of a tranquilliser or a sedative.* Propofol is *non-cumulative*, and is therefore suitable for administration by *infusion*. Propofol is suitable for use in *sight hounds*.

Propofol reduces myocardial contractility. Hypotension occurs, primarily due to arterial and venous dilatation. Propofol reduces intracranial and cerebral perfusion pressures. Respiratory depression occurs and the severity is related to the dose and the rate of administration.

The very rapid and complete recovery observed after propofol anaesthesia make this drug suitable for patients with obstructive ventilation eg brachycephalic patients.

Mode of Action Central nervous system depression is produced by enhancing the effects of the inhibitory neurotransmitter, GABA, and thereby decreasing brain activity.

Side Effects

- Transient apnoea, respiratory depression.
- Decreased blood pressure due to ↓ systemic vascular resistance (> thiopentone)
- In cats, anorexia, diarrhoea and malaise may occur due to oxidative injury to red blood cells. Cats are deficient in the enzymes necessary to conjugate phenols. However, propofol is used with success in cats.
- Pain on injection.

Propofol anaesthesia is practiced in most species but cost can be prohibitive in large animals.

STEROID ANAESTHETICS

History: - Alphaxalone (alfaxalone) and alphadalone are two steroid anaesthetics which were once marketed in combination. Alphaxalone is the more potent of the two, but the addition of alphadalone was believed to improve the solubility of alphaxalone. These drugs were marketed as Alfaxan® and Saffan® (9mg of alphaxalone and 3mg of alphadalone dissolved in 20% Cremophor EL, a polyoxyethylated castor oil).

25% of cats administered Saffan® or Alfaxan® developed *oedema of the feet, muzzle and ears*. This usually disappeared within 2 hours. Some dogs, however, had an allergic response to first exposure of the drug, characterised by a prolonged fall in blood pressure, urticaria and erythema. This could be reduced by prior administration of an antihistamine. Death due to anaphylactiod reactions have been recorded in cats and dogs.

New formulation: - Because of these problems, Jurox Pty Ltd (Australia) has developed a **new formulation of alphaxalone in cyclodextrin**. This was released for use in cats and dogs in 2000 and has rapidly become popular with small animal veterinarians. There is *no histamine release* associated with this formulation.

Alphaxalone as an anaesthetic: -

* High therapeutic index
* Non-cumulative
* Rapid, complete recovery
* Good cardiovascular stability
* Little respiratory depression at therapeutic doses
* Good muscle relaxation (some transient muscle twitching at induction in some patients)
* Non-irritant

Induction with IV alfaxalone is rapid and recovery is usually uneventful. Prolonged anaesthesia with alfaxalone can result in some neuroexcitation. However, this is rarely a clinical problem in premedicated small animals.

The effects of alfaxalone are dose dependent. It can be administered at a low dose as a sedative or at a higher dose to induce and/or maintain anaesthesia.

DISSOCIATIVE ANAESTHETICS

Phencyclidine, ketamine and tiletamine are dissociative anaesthetics.

Dissociative anaesthesia = an anaesthetic state induced by drugs that interrupt ascending transmission from the unconscious to the conscious parts of the brain, rather than generalised depression of all brain centres. A *cateleptoid state* is produced, in which the eyes remain open, and some *protective reflexes are maintained*. (eg palpebral, pharyngeal, laryngeal). A *hypertonic* state exists (ie muscle rigidity), making ketamine unsatisfactory as a sole agent for surgery. These anaesthetics are in a minority group of anaesthetic agents that provide *analgesia*. Analgesia is intense but of short duration.

There is a *rise in blood pressure* and cardiac output is maintained or increased due to release of catecholamines into the circulation. (However, the dissociative anaesthetic agents cause myocardial depression if applied to isolated cardiac muscle.) Ketamine causes minimal respiratory depression at therapeutic doses. *Intraocular pressure is increased*. These agents are *contraindicated* in patients with *raised intracranial pressure, penetrating eye wounds, or those undergoing eye surgery*.

The dissociative anaesthetics undergo extensive hepatic metabolism, and there is significant excretion of unchanged drug via the kidneys. As with the barbiturates, redistribution plays an important part in recovery of consciousness

When used on its own in dogs, ketamine causes extreme muscle tone, twitching and occasional *convulsions*. When dissociative anaesthetics are used in combination with *benzodiazipines* or α_2-adrenoreceptor agonists (both good muscle relaxants), suitable muscle relaxation is achieved. Ketamine is used with xylazine, diazepam, midazolam and medetomidine.

Tiletamine is marketed in combination with zolazepam, a benzodiazepine, as *Zoletil®* *(Telazol® in USA)*.

Ketamine in combination with diazepam (or midazolam, xylazine etc) and Zoletil® have become popular in veterinary anaesthesia.

SKELETAL MUSCLE PARALYSING DRUGS

It is important to distinguish between muscle relaxation and muscle paralysis.
- Drugs like the benzodiazepines (eg diazepam) and the α_2-adrenergic agonist drugs (eg xylazine) cause **muscle relaxation**. This results in a decrease in skeletal (voluntary) muscle tone and may cause an animal to stagger, or have difficulty rising. There may be some reduction in tone of the muscles of ventilation resulting in hypoventilation.
- **Muscle paralyzing drugs** block the neuromuscular junction and prevent voluntary muscle movement. An effectively paralysed animal cannot move its limbs or, importantly, use the muscles of ventilation at all.

Neuromuscular blocking drugs. These drugs act at the neuromuscular junction, (NMJ), stopping nerve impulses from causing contraction of the muscle. There are two modes of action of NMJ blocking agents.
a) *Competitive blocking drugs* compete with acetyl choline (ACH) for the end-plate receptors (nicotinic receptors), but once attached to them, do not cause depolarisation. The term "non-depolarising" is sometimes used. The activity of these drugs is enhanced by anaesthetic drugs. Rocuronium, pancuronium, vecuronium and atracurium act in this manner. Their action can be opposed by increasing the local concentration of ACH by administration of anticholinesterases (eg neostigmine). **Neostigmine must always be preceded by atropine or profound bradycardia and even cardiac arrest can result as this drug stimulates muscarinic and well as nicotinic ACH receptors. .**
b) *Depolarising blocking drugs* are agonists of ACH, but the depolarisation caused by these drugs persists longer. Depolarisation by ACH lasts only a few milliseconds, as the ACH is hydrolysed rapidly to the almost inactive choline and acetic acid. Like ACH, suxamethonium attaches to the receptor and causes depolarisation, but the molecule remains on the receptor longer, causing a protracted block.

Indications for Muscle Paralysis

- To *relax skeletal muscles for surgery*
- To *facilitate control of ventilation* during thoracic surgery
- To assist with *reduction of dislocated joints*
- To *limit the amount of general anaesthetic agent* required to keep the patient suitably relaxed and still for surgery.
- To *facilitate endotracheal intubation*.

It must be remembered that neuromuscular blocking agents must be used only in patients that are already anaesthetised.

Competitive Neuromuscular Blocking Agents
The competitive muscle relaxants *bind to plasma proteins* to a large extent. The unbound portion is active, so if the plasma albumin levels are low, smaller doses will be required. Local anaesthetics as well as the aminoglycoside antibiotics (neomycin, gentamycin, streptomycin) diminish the release of ACH from nerve terminals and therefore enhance the action of the competitive neuromuscular blocking agents.

Rocuronium
Duration of action in dogs – approximately 20 min in dogs and 13 min in cats.   Very fast onset of action (complete blockade in dogs 2–3 min, cats 30– 0 sec).   Eliminated primarily via the liver. Minimal cardiovascular effects and histamine release.

    Dog   0.5 mg/kg IV
    Cat   0.6 mg/kg IV

Pancuronium
Duration of action in dogs is approximately 40 minutes. More than half is eliminated via renal excretion. Can trigger malignant hyperthermia in pigs.
CVS moderate rise in pulse and C.O. Not seen after atropine – so must be due to vagolytic effect.   No histamine release.

    Dog & cat   0.02 – 0.06 mg/kg IV

Vecuronium
Duration of action in dogs – approximately 20 minutes. More than half is eliminated in the bile, and some via renal excretion.   Very specific in its action. Hence, little cardiovascular system effect, and low incidence of histamine release.

    Dog & cat   0.06 mg/kg IV

Atracurium
Duration of action in dogs – approximately 40 minutes. Elimination is via Hoffmann degradation, *spontaneous breakdown in plasma* at body temperature and pH. Elimination independent of renal or hepatic pathways is possible.

Depolarizing Neuromuscular Blocking Agents

Suxamethonium (chloride and bromide) (succinylcholine)
CNS effects – none. Large, highly polar molecule (quaternary ammonium compound) – therefore cannot cross the highly lipid blood brain barrier.
CVS – maybe vagal stimulation, bradycardia. (Precede with a vagolytic drug eg atropine)
Causes $\uparrow$ serum K^+ in burns patients
Muscular system. Rapid, profound block preceded by fasciculation due to depolarisation of the motor end plate. This has been reported to cause extreme pain in people.
Histamine release can occur.
Abrupt and short-lived increase in intraocular pressure. *Do not use in penetrating eye wounds.*
In most species, the block caused by suxamethonium is very short lived (several minutes), but dogs are extremely sensitive. A dose of 0.3mg/kg lasts for approximately 20 minutes in dogs, compared with 3 – 5mg in cats lasting 5 – 6 minutes. In anaesthetized cats, a dose of 0.2 mg/kg is suitable to facilitate intubation. The anaesthetist must be confident that they can immediately achieve intubation of the trachea.

Use in mammals only, not in birds, reptiles or amphibians.

Only one dose of a depolarizing muscle paralyzing drug should be administered, or prolonged block results.

The anaesthetist must prepare to administer intermittent positive pressure ventilation BEFORE administering a muscle paralyzing drug. It is vital to have an endotracheal tube in place with the cuff adequately inflated and the breathing system set up and checked. Inflate the lungs by squeezing the reservoir bag several times to demonstrate that inflation is possible.

When you are sure you can ventilate, then administer the muscle paralyzing drug.

MONITORING THE ANAESTHETISED PATIENT
Helen Keates and Brenda Dixon

"A good scare is worth more to man than good advice."

A   INTRODUCTION

1.   REASONS FOR MONITORING

When monitoring the anaesthetised patient, the anaesthetist has several aims. Firstly, the *patient's safety* must be ensured. Secondly, patients must be in a *state suitable for the planned procedure* to be carried out (eg radiology, surgery).

The following is a breakdown of these aims: -

- The patient must be *asleep*.
- Adequate *antinociception* must be provided. A patient can have detrimental responses to noxious stimuli in spite of being unconscious and thereby unable to feel pain.
- The patient must be *still* in spite of stimulation caused by the procedure, and *muscle relaxation* must be adequate for the required manipulations.
- The patient's *physiological state* should be such that *complete reversal of anaesthesia* is possible without residual, detrimental effects. Thus, the patient's tissues must be *adequately perfused with oxygenated blood*, CO_2 *levels must be maintained, body temperature* must be maintained *etc*.

Historically, objective measurements, such as the patient's pulse and respiratory rates have been recorded, as well as the subjective observations, such as the character of the respiration and pulse, colour of mucous membranes etc. Over the last few decades, there has been a swing towards *electronic monitoring* devices that give *objective measurements*. The enthusiasm for such devices has been driven partly by curiosity, partly by the need for objective measurements for real scientific progress, and partly by the medicolegal climate, in that accurate, objective medical records are essential. There is no doubt that the quality of patient care has improved, but it must be remembered that the *new monitoring devices act as adjuncts to, not replacements for diligent, hands-on monitoring. A veterinarian with very basic equipment can be a competent anaesthetist.*

2.   Philosophy

1. It must be remembered that whatever you measure, *one measurement gives a "window" into a dynamic situation*. Only *repeated measurements* give insight to the dynamic picture.
2. There is a limit to the accuracy of any monitor, and because of this, the *trends are often more meaningful than the actual numerical values obtained*.
3. It is *easy to do a "bad job" of monitoring with the very best of equipment*. For example, if a pulse oximeter ceases to give read-outs of oxygen saturation and pulse rate, the anaesthetist may spend several minutes replacing the probe without checking that the patient has a pulse. A serious situation may go undetected for longer than if there had been no pulse oximeter attached to the patient. If a monitor fails to give an appropriate reading, the anaesthetist's *first priority must be to do a basic, but thorough examination of the patient and the breathing system* before attending to the monitor. Once the wellbeing of patient has been established, the monitor can be attended to.

Check the patient before checking the monitor.

Monitor the patient, not the monitors.

A monitor should not replace a skilled anaesthetist.

B   ASSESSMENT OF ANAESTHETIC DEPTH

The most accurate way of measuring depth of anaesthesia is to measure brain activity. This is achieved by attaching a series of electrodes to the patient's skull so that an electroencephalogram can be monitored. Interpretation of the resulting EEG makes this a useful tool in human patients.   Whilst this technology is becoming increasingly available for use with human patients, most veterinary anaesthetists do not have access to this sophisticated equipment.

There is *no one variable* that the veterinary anaesthetist can assess that will establish depth of anaesthesia.   Overall assessment of *many parameters* will allow the experienced anaesthetist to *gauge depth of anaesthesia* as well as the *physiological status* of the patient. The *cardiovascular and respiratory systems* are of prime importance in assessing *depth of anaesthesia* as well as *physiological stability*. These two systems together are responsible for perfusion of the tissues with oxygenated blood, and for removal of CO_2 and other metabolites. Other variables can be measured to give further information.

1.   BASIC MONITORING – or what you can do with a stethoscope, a watch and a thermometer.

a)   Cardiovascular system

The **pre-anaesthetic examination** of the cardiovascular system is necessary if the anaesthetist is to assess changes that take place during anaesthesia.   Many agents administered result in dose-related vasodilatation and/or cardiac depression, and the pre-anaesthesia examination will provide a *baseline* for comparison with subsequent measurements.

Auscultate heart sounds   A patient with a heart murmur requires further investigation. Patients will occasionally develop a cardiac murmur *after* administration of premedicants or anaesthetics. This is often associated with vasodilatation and a compensatory increase in cardiac output, increased flow resulting in turbulence.

The information to be derived from **palpation of superficial arteries** goes well beyond counting the rate. Information can be obtained about: -

- rate
- rhythm
- amplitude of the pulse wave and
- tone of the vessel wall

The *rate and rhythm* can be assessed objectively. The pulse rate of a conscious animal examined by a veterinarian is rarely the animal's normal resting rate. It is usually increased in response to many different stimuli eg unfamiliar surroundings and people, pain etc. A sinus arrhythmia is normal (identified by increased heart rate on inspiration and decreased rate during the expiratory pause. The respiratory centre is stimulated by general neural traffic)

Usually, the amplitude of the pulse wave and the *tone of the vessel* can be assessed only subjectively, but useful information is obtained if repeated assessments are made.   The *amplitude* reflects the *stroke volume* and *peripheral resistance*.   The *tone* of the vessel reflects *blood pressure*.   The *small arteries* are useful because they are *accessible* to the anaesthetist during surgery, and because they *become difficult to feel as blood pressure drops*. As stated previously, it is changes that are meaningful. For example, if the tongue pulse can be palpated at the start of anaesthesia but not after an hour, then it is likely that the blood pressure has dropped.

Colour of the mucous membranes can be subjectively assessed, but the animal will be severely hypoxic by the time the mucous membranes are observed to be blue.
- o Cyanosis occurs when the blood contains 5g/100ml of deoxygenated haemoglobin. If the patient has eg 12 g/100 ml haemoglobin, the mucous membranes may not appear blue until only 7 mg/100 ml Hb is oxygenated. This means, less than 60 % of the haemoglobin is oxygenated by the time you can detect the change in colour.
- o If the patient is breathing a high percentage of oxygen (eg 99%), the mucous membranes will remain pink for many minutes after respiratory arrest.

Mucous membrane refill time should be < 1 – 2 seconds. Whilst noting changes in refill time is worth doing, it should be noted that refill time can remain within normal range well after the animal is dead. Refill can be due to backflow from venules as well as inflow from arterioles.

b)   Respiratory system
What the anaesthetist would like to be sure of is that respiratory function is adequate ie the blood is *oxygenated and CO_2 is removed*. Simple observation does not provide this information.
The rate, rhythm, depth and character of ventilatory movements should be noted in spontaneously breathing patients.
The *rate* of ventilatory movements can be counted and recorded intermittently. Small animals usually have higher rates than large animals.
The *rhythm* of ventilatory movements under surgical anaesthesia should be regular. Breath holding can indicate light anaesthesia. Aberrant respiratory rhythms can indicate respiratory centre dysfunction.
Observing thoracic and reservoir bag movements allows subjective assessment of the *depth* of ventilation.
The *character* of the ventilatory movements is of particular value in assessing the depth of anaesthesia. In light anaesthesia, ventilatory movements are often irregular, and "jerky". In surgical anaesthesia, ventilatory movements are usually smooth and regular. As anaesthesia deepens, the rhythm usually remains regular, although the tidal volume reduces. If only the rate is noted, the anaesthetist may miss the gradual reduction of tidal volume until ventilation fails.

c)   Ocular signs
i)   The **position of the eye** and **size of the pupil** vary with depth of anaesthesia. In cats and dogs, during the very early stages of induction of anaesthesia, the eyeball will be active and appears to be watching. As light surgical anaesthesia is approached, the eyeball will roll down.   As depth of anaesthesia increases, the eyeball will roll back up. (Eye position varies somewhat with the individual, but there are major differences between species). The pupils will then gradually dilate as very deep anaesthesia is approached. These *stages are not reliably followed in all cases* and are influenced by degree of stimulation.
ii)   **Palpebral reflex**   If the *palpebrae are lightly touched* in a lightly anaesthetised patient (most species), they will close. This response is obtunded as anaesthesia deepens. This reflex is usually maintained in horses in surgical anaesthesia. The palpebral reflex must not be confused with the corneal reflex.
iii)   **Corneal reflex**   If the *cornea is touched*, the palpebrae will close. This is quite different from the palpebral reflex described above. The corneal reflex is maintained throughout very deep planes of anaesthesia, and can even be elicited after cardiac arrest in some circumstances. It is not useful in assessing anaesthetic depth.
d)   Temperature   a) *Core temperature*. Heavy sedation and anaesthesia render the patient less able to maintain body temperature. Thus, the animal's core *temperature will tend to follow ambient temperature*. In most situations, loss of body heat is the concern, but in field cases in Australia, animals can overheat if not properly cared for whilst recovering. Large animals have low surface area to body mass ratio, and therefore are less

likely than small animals to suffer loss of body temperature. In hot weather, it is important to choose a site for anaesthesia that is in the shade for horses and cattle likely to have a prolonged period of sedation or anaesthesia and recovery.

Small patients lose heat from breathing circuits (cold dry medical gases), open surgical wounds, contact with cold metal tables etc. To avoid hypothermia, place patients on thermostatically controlled heating mats, warm IV fluids, wrap the patients where possible and keep the duration of the anaesthetic to a minimum. Recent research indicates *that active warming of the extremities* contributes to maintaining body temperature.

If the core temperature is allowed to decrease, the incidence of *cardiac dysrhythmias* will increase. Cardiac arrest can result. Hypothermia *decreases anaesthetic requirements* and *prolongs recovery time*. If hypothermia is severe, the patient may not recover from anaesthesia unless actively warmed.

b)   Peripheral temperature is not a reliable indicator of core temperature. Peripheral vascular shutdown will contribute to maintenance of core temperature whilst allowing the periphery to cool down.   Peripheral and core temperatures can differ by up to 6°C in an individual patient.

e)   Muscle relaxation   In general, *muscle relaxation increases with depth of anaesthesia*. To a limited extent, the degree of muscle relaxation can be used as an indication of anaesthetic depth.

- However, if specific muscle paralyzing drugs (neuromuscular blocking agents) are administered, the degree of muscle relaxation is of no use in assessing depth of anaesthesia.
- The dissociative anaesthetics, eg ketamine, tiletamine, phencyclidine, cause increased muscle tone. They are often administered in combination with sedative agents that provide muscle relaxation eg benzodiazepines (diazepam, zolazepam) or α-agonists (eg xylazine, medetomidine).

2.   MONITORING DEVICES

a)   ECG recordings give valuable information about the *electrical activity of the heart*. Electrical changes may herald serious problems. It must be remembered *that electrical activity of the heart cannot be equated to cardiac function*. Cardiac output can be much reduced with reasonable electrical activity.

Monitoring the ECG under general anaesthesia allows the anaesthetist to constantly evaluate the electrical activity of the heart. Monitoring the ECG under general anaesthesia allows the anaesthetist to constantly evaluate the electrical activity of the heart. It will not however give any indication to mechanical activity. The electrodes ideally should be placed on both forelimbs and the left hind limb, but may be placed anywhere on the body if any of these positions are unavailable (i.e. surgery site). In general lead *II* is the preferred lead as it is the most familiar but any lead can be used. An ECG is the only accurate way of specifically diagnosing a dysrhythmia, which is essential to the implementation of appropriate treatment.

b)   Heart rate monitors are available for veterinary patients. Most work by detecting electrical activity of the heart. Therefore they *do not denote cardiac function*, but rather electrical activity.

c)   Oesophageal stethoscopes consist of a long thin tube, with a closed "balloon" at one end for insertion into the oesophagus to the level of the heart. The other end is connected either to a stethoscope, replacing the diaphragm attachment, or to a microphone and speaker. This can be set up cheaply. *Heart rate and rhythm* can be assessed with accuracy. As the sounds heard represent turbulence associated with valve closure, the *information gained is about function, not simply electrical activity*. Subjective assessment can be made of stroke volume ie if the heart sounds are quieter, it may mean that the stroke volume is smaller.

Oesophageal stethoscopes are valuable as *respiratory rate monitors* as the breath sounds can be heard. Partial obstruction resulting in increased turbulence can be detected as increased respiratory noise, as can leakage due to improperly cuffed endotracheal tubes.

d)   Central venous pressure (CVP) monitoring.   CVP can be measured by cannulating a major vein (usually the jugular) and connecting the cannula to a simple manometer filled with sterile isotonic solution or to a pressure transducer. Measurements taken from these simple manometers are usually reported in centimeters of water. This can be converted to mm Hg by dividing by 1.36 (1 cm water ≡ 1.36 mm Hg). This is simple to set up, and gives a fair estimate of mean blood pressure, especially if related to baseline readings. Measurements taken by electronic means (catheter connected to a transducer etc) are easier to set up than those taken with a water manometer. Many multipurpose monitors have this capacity. It is imperative to position the manometer so that the base of the column of fluid (or the transducer) is at the level of the right atrium.

e)   Blood pressure is everything!
Hypotension under anaesthesia in small animals is defined as a mean arterial blood pressure lower than 60 mmHg. Untreated profound hypotension can lead to organ damage and failure, and ultimately respiratory and cardiac arrest. Arterial blood pressure is determined by cardiac output and systemic vascular resistance. Systolic blood pressure is primarily determined by stroke volume and arterial compliance, diastolic blood pressure by systemic vascular resistance and heart rate. Mean blood pressure is the average arterial pressure over time: one half of the pulse pressure wave form. If the pulse-pressure wave form is a perfect triangle the mean would be diastolic pressure plus one third the difference between systolic and diastolic pressures. If the pulse-pressure waveform is tall and spiked then the mean pressure will be much closer to the diastolic pressure. The mean arterial pressure is the most important pressure of all because it is the driving pressure for tissue perfusion. Mean blood pressure less than 60 mmHg results in compromised tissue perfusion, particularly to the splanchnic organs, and mean pressures lower than 40 mmHg will result in inadequate perfusion of the vessel rich organs such as brain and heart. Blood pressure measurement must always be considered along with your clinical assessment of the patient. A patient with a mean blood pressure of 60 mm Hg with peripheral vasodilation i.e. bright pink mucous membranes and a fast capillary refill time is in a much better haemodynamic state than a patient with a mean blood pressure of 60 mmHg with peripheral vasoconstriction i.e. pale mucous membranes and slow capillary refill time.
Hypotension is a common occurrence under general anaesthesia. Drugs can cause hypotension in a number of ways: they can reduce cardiac output by a exhibiting a negative inotropic effect e.g. inhalation agents, thiobarbiturates, propofol and α_2 adrenergic agonists; they can have a negative chronotropic effect (lowering heart rate) e.g. opioids, inhalation agents and α_2 adrenergic agonists; or they can produce vasodilation resulting in a relative hypovolaemia e.g. acepromazine, inhalation agents, propofol, thiobarbiturates and Alfaxan®. Remember there is no perfect drug!
It is imperative to be able to identify hypotension to be able to treat it successfully. It is much better to instigate early appropriate treatment in the course of hypotension rather than wait until there has been a significant deterioration in tissue perfusion over a protracted period of time resulting in organ ischaemia. **If you don't monitor blood pressure, you won't know if hypotension exists!**

Some treatment options for the hypotensive patient (mean < 60 mmHg, systolic < 100 mmHg):
1. Decrease inhalation agent to a minimum, e.g. turn down vaporizer, administration of an opioid may help to facilitate this.
2. If the patient is hypotensive and bradycardic then it is appropriate to treat the bradycardia with an anticholinergic agent first e.g. atropine or glycopyrrolate. High parasympathetic tone under general anaesthesia will not only decrease heart rate but also decrease inotropy. Administering an anti-cholinergic should not only increase

heart rate but will often increase blood pressure also (cardiac output = heart rate X stroke volume). Be careful however in an animal with heart disease as tachycardia will increase myocardial oxygen consumption and if this demand is not met then myocardial ischemia will result. If the blood pressure does not increase sufficiently after an anti-cholinergic agent has been administered then further specific treatment for the hypotension is warranted.

3. Ensure adequate fluid loading. Small boluses of crystalloids (5ml/kg increments up to a total dose of 20ml/kg in dogs, half these doses in cats) or colloids (2 – 5ml/kg increments, up to total dose of 20ml/kg in dogs and 10ml/kg in cats) can usually be tolerated even in geriatric patients. The exceptions to this rule are animals that are at risk of developing pulmonary oedema. No fluid loading will be tolerated in these patients.

4. Positive inotropes can and should be instigated if these initial treatment options have not successfully increased blood pressure, dopamine is usually appropriate in most situations, ephedrine can also be used.

5. An α $_1$ adrenergic agonist e.g. phenylephrine, will result in peripheral vasoconstriction with little or no cardiac effects, making it a good choice for those patients where an increase in myocardial contractility is best avoided e.g. cats with hypertrophic cardiomyopathy (HCM).

Arterial blood pressure monitors are described as direct and therefore invasive or indirect and non-invasive.

Direct measurement is the most accurate of all blood pressure monitoring and provides a continuous reading. It does, however, necessitate the catheterisation of a peripheral artery, it is relatively expensive as it requires a single use sterile transducer administration set and requires some skill and special equipment. Most of the newer multi-function monitoring devices have direct blood pressure measurement available. It is the gold standard of blood pressure monitoring and is recommended for high risk cases and those patients undergoing extensive surgery as it will give accurate readings even in very low perfusion states, unlike the indirect blood pressure monitors.

Indirect measurement involves a *sphygmomanometer cuff and a sensor*. The width of the cuff should be about 40% of the circumference of the limb. It should be applied snugly, not tightly around the limb. If the cuff is too tight, measurements will be erroneously low. Conversely, if the cuff is too loose, the measurements will be too high. *The cuff is inflated around an extremity to occlude the artery*. (See diagram) As the pressure is slowly released from the cuff, the *occlusion is overcome and blood begins to flow* through the artery again. At the point when the blood first begins to flow, there *is turbulence*, and therefore *noise* (called *Korotkoff* sounds). A stethoscope on an artery distal to the sphygmomanometer cuff will detect these sounds. (This method does not work well in animals.) When the sound of turbulent flow is detected, the cuff pressure equals the systolic blood pressure. Once the blood flow is no longer affected by cuff pressure, the diastolic pressure has been reached, and the noise or turbulence disappears. Various devices are available to pick up this noise/turbulence/change in flow. (Stethoscope, microphones with amplifier)

There are two devices in common use in veterinary anaesthesia that use flow occlusion by placement of a cuff. These are called NIBP (*non invasive blood pressure)*.

Devices that rely on sphygmomanometry can be inconsistent in small animals. If sphygmomanometry is used in cats and dogs, it is important that the readings are considered in relation to baseline values, not as absolute readings. Some success has been achieved in horses with the cuff placed on the horse's tail.

1)   Oscillometric devices eg Dinamap$^®$ (*device* for *indirect non-invasive automatic mean arterial pressure*) by Criticon.

This is a device that ***measures changes in pressure wave oscillations***. Oscillometry involves the placement of an appropriate sized cuff over a peripheral artery as well, but the monitoring device automatically inflates and deflates the cuff at discrete pre-set time

intervals and analyses the fluctuations of pressure in the cuff that occur with each pulse wave in the limb. It provides a display of systolic, diastolic and mean blood pressures. This device detects changes (or lack of change) in pulse wave amplitude.

The points at which the changes in pressure oscillations peak and trough give the systolic and diastolic pressures. If the cuff is inflated to occlude the artery (pressure > systolic) and then released to allow some blood through (systolic pressure) the wave form changes from no wave (occlusion) to a detectable wave. This is an infinite change. Conversely, diastolic pressure can be detected by loss of change of wave form. Mean pressure is calculated.

An oscillometric blood pressure monitor has advantages over the Doppler ultrasound monitor (described below) of being automatic (less 'hands on') as well as providing diastolic and mean pressures but it is less likely to provide accurate readings in very low perfusion states and generally cannot be used successfully in very small patients.

The PETMAP® is a small hand-held oscillometric device used successfully in cats and dogs.

2)   Doppler shift _**flow detectors**_ placed distal to the cuff can be used with reasonable accuracy to measure blood pressure. As the pressure in the cuff is released, the pressure at which *flow* is first detected is equal to the systolic pressure.

Doppler ultrasound involves the placement of a probe containing a piezoelectric crystal over a peripheral artery. The crystal emits sound waves at a certain frequency and when they hit the moving red blood cells they are reflected back to the probe at an altered frequency (the 'Doppler shift'), which is then converted to an audible signal. They can be used as a pulse detector, but if a closed cuff system attached to a sphygmomanometer is placed between the probe and the heart then a systolic blood pressure can be obtained. The cuff is inflated to a pressure which occludes the artery and the audible signal is lost, slowly the pressure is released until the first faint audible signal is heard. This will be the systolic blood pressure. Accurate readings require correct probe placement, appropriate sized cuff etc. Research has shown that even when the system has been set up correctly the 'real' systolic blood pressure may differ by up to 20 mmHg and particularly in cats; it may read closer to the mean than systolic blood pressure. It should always be presumed to be reading systolic blood pressure, however, as this is a safer presumption for the patient. Newer more sophisticated devices can also measure diastolic blood pressure as well. Doppler devices can be used in all sized animals.
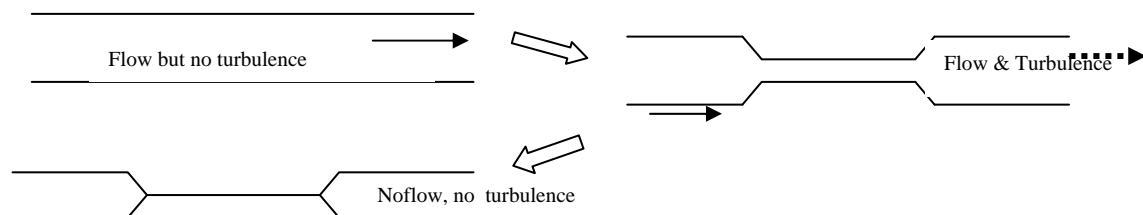
All of the non-invasive techniques are less accurate than direct blood pressure measurement. They are however extremely quick and easy to apply, easy to use and after the initial purchase of the device very low cost per use. The Doppler can be easily attached to an awake patient and so can be placed prior to induction of anaesthesia; it is easily transportable and so can remain in place allowing for frequent blood pressure assessment for the duration of the entire procedure.

f)   **Pulse oximeters** measure the *percentage saturation of haemoglobin with oxygen.* A probe is placed on unpigmented mucosa or skin (commonly the tongue, lip, vaginal or preputial mucosa or interdigital webbing).

Pulse oximetry works on the principle that oxyhaemoglobin and deoxyhaemoglobin absorb light at different wavelengths. The pulse oximeter uses two light–emitting diodes that pulse red and infrared light through a tissue bed. The amount of light absorbed at each

wavelength is measured by photo detectors and the data is expressed as a percentage of oxygenated haemoglobin to total haemoglobin. They are able to distinguish pulsatile blood flow and so the technology has been named 'pulse' oximetry. Typically they display the amount of oxygenated haemoglobin expressed as a percentage of total haemoglobin (SpO_2), as well as a pulse rate (this should equate to heart rate if there are no dysrhythmias present). Percentage of saturation gives an indication of the adequacy of both oxygenation and to some degree circulation.

The oxygen-haemoglobin dissociation curve (below) is a graphic representation of the relationship between the SpO_2 and the partial pressure of oxygen in arterial blood (PaO_2). Note that below 85 % SpO_2 the curve becomes steep and almost linear and a small decrease in SpO_2 results in large decrease in PaO_2. Thus, in this range, even minimal decreases in SpO_2 place the patient in danger of developing hypoxemia. The curve can be shifted to the right or left by changes in pH, temperature and 2, 3- DPG levels in red blood cells, resulting in either increasing or decreasing the amount of PaO_2 for the same SpO_2. (I.e. Altering the affinity of oxygen for haemoglobin and making it more or less tightly bound to the molecule determining how easily it is picked up at the alveolus and how easily it is let go in the tissues).

Arterial pulses must be of an adequate strength to be detected by a pulse oximeter. It will struggle to detect a pulse in low perfusion states, such as profound hypotension but also in the presence of high peripheral resistance e.g. after an α_2 adrenergic agonist. Generally a reading of above 95% SpO_2 equates to a PaO_2 of 80mmHg or above and so represents adequate oxygenation.
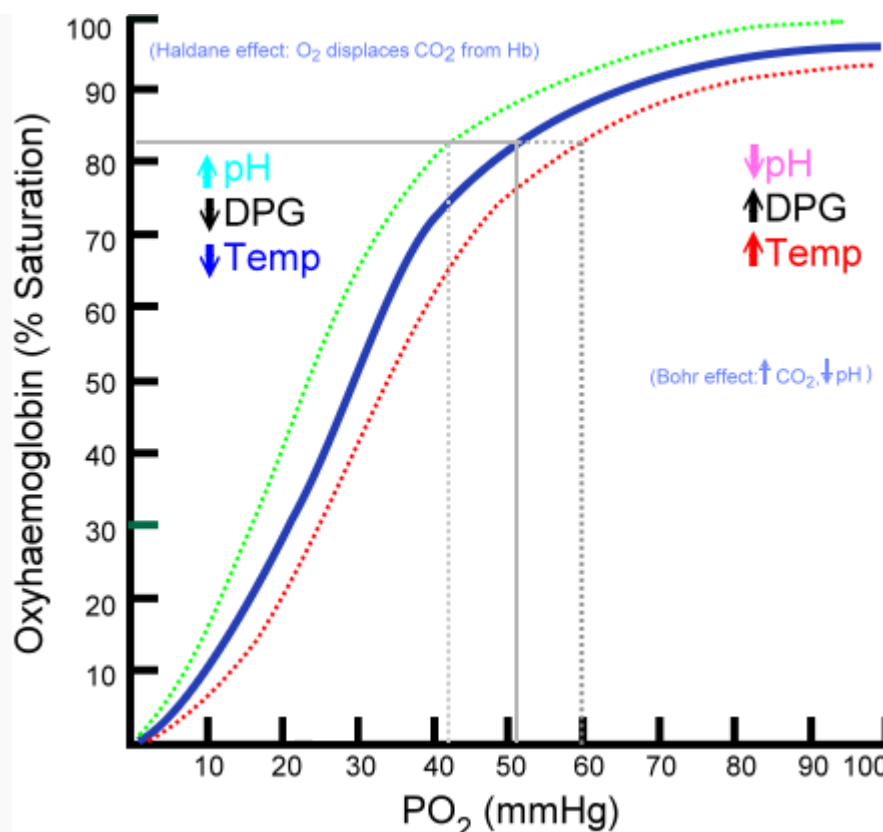
Oxyhaemoglobin dissociation curve (reproduced from Ganfyd)

NOTE: When a patient is inspiring more than 30% oxygen (cf 21% room air) e.g. during oxygen therapy or under anaesthesia, the SpO_2 reading can be high even in the presence of profound hypoventilation and so a pulse oximeter cannot be used to determine the adequacy of ventilation. Hypoventilation , regardless of adequacy of

oxygenation, will result in accumiulation of CO_2. Oxygenation and ventilation are two very distinct processes that occur in the lungs and one must be able to assess the adequacy of each individually. Pulse oximetry can be used as a non-invasive monitor of oxygenation; capnography a non-invasive monitor to assess the adequacy of ventilation.

Also, when a patient is on high levels of inspired oxygen, it may maintain an adequate SpO_2 despite having poor gas exchange capability. This can only be determined by performing a blood gas analysis to measure the PaO_2, and then using the alveolar gas equation to determine the adequacy of gas exchange and see if the patient is *relatively* hypoxaemic. When the inspired oxygen is close to 100%, the PaO_2 should be over 500 mmHg. A good rule of thumb to apply is that the PaO_2 should be about 5 times the predicted inspired oxygen concentration, i.e. if a patient is breathing 21% oxygen in room air then the PaO_2 should be about 100 mmHg (i.e. 21 X 5 = 105), if the patient is breathing 30% inspired oxygen with nasal insufflation then the PaO_2 should be about 150mmHg (i.e. 30 X 5 = 150). If the PaO_2 is less than that predicted with the 5 times rule then you can deduce that there is a reduction in the oxygenating ability of that patient. If the PaO_2 is only 2 to 3 times the inspired oxygen concentration rather than 5 times, then that animal has a moderate reduction in its oxygenating ability, if it is only twice or less than the inspired oxygen concentration then the patient has very poor oxygenating ability. A patient that has a normal SpO_2 reading whilst on close to 100% oxygen on the anaesthetic circuit but does have a severe reduction in its oxygenating ability will suddenly deteriorate when the inspired oxygen concentration is decreased to 21% on room air. To approximately measure gas exchange and in particular oxygenating ability, it is always worthwhile to disconnect the patient from the anaesthetic machine and measure pulse oximetry for 5 minutes (to allow for equilibration) with the patient still intubated to see whether it is able to maintain an SpO_2 above 95%. If not, supplemental oxygen should be delivered in recovery. Pulse oximeters are very useful, but have limitations. Eg oxygen saturation will be normal in anaemic patients, whereas tissue oxygenation may be inadequate due to poor oxygen carrying capacity of the blood.

In a patient breathing a high concentration of oxygen, a sudden drop in oxygen saturation may indicate **circulatory failure** eg cardiac arrest.
In studies of human anaesthetic incidents, the pulse oximeter has been shown to be superior to other commonly used monitors in early detection of patient problems.

g)   Respiratory (ventilatory) rate monitors and apnoea alarms.   Most respiratory rate monitors have a thermistor placed in the breathing circuit so that warm expired air can be detected. Thus, any air movement is recognized as a breath. This system gives *no indication adequacy of ventilation* and therefore will not detect advancing respiratory failure due to declining tidal size of ventilatory movements.

h)   Tidal volume monitors. Tidal volume can be measured with a respirometer eg Wright's respirometer. This instrument is fitted into the breathing circuit and will measure individual *tidal volumes and respiratory minute volume*. This allows an estimate of the alveolar ventilation.

i)   Blood gas analysis is the ***most efficient way of measuring respiratory efficiency***, and also provides information as to the *acid-base balance*. Although the equipment necessary is expensive an increasing number of practices have the capacity to perform blood gas analysis. Collection of an arterial blood sample into a heparinised syringe is usually via a preplaced arterial catheter which is kept patent by repeated flushing with heparinised saline (2 IU/ml). Essentially, arterial blood pH and partial pressures of CO_2 and O_2 are measured (eg normal values for a canine patient could be, eg pH 7.40, $PaCO_2$ 35 mm Hg, PaO_2 102 mm Hg). Variables that denote the acid-base status of the patient (HCO_3^-, Total CO_2, Base Excess in mm/L and oxygen saturation %) can be derived from the measured values for pH and $PaCO_2$.

j) **Capnography** is the measurement of the *CO_2 content of the respiratory gases*. Infrared analysis is carried out on gas sampled from the breathing circuit (usually at the junction of the ET tube and the breathing circuit), giving a continuous reading of the CO_2 levels throughout the respiratory cycle.

- ***It is important to establish that the partial pressure of CO_2 in the inspired gas is zero.*** If the inspired gas has > 0 mm Hg CO_2, there is a problem with the patient breathing circuit. With a non-rebreathing system, inspired CO_2 > 0 mm Hg is most likely due to a fresh gas flow rate that is too low. With a rebreathing system eg circle absorber, the absorbent may be exhausted or one of the one-way valves may not be closing etc.
- Of particular interest is the ***end tidal CO_2*** as this is related to the partial pressure of CO_2 in *arterial blood*. End tidal gas should contain about 40 mm Hg. High CO_2 usually means hypoventilation, but may also be observed in patients with pulmonary disease or pulmonary embolism etc.

Capnographs are *very useful* in practice. Accidental intubation of the oesophagus can be diagnosed with capnography (absence of wave form, very low pCO_2).
Capnographs are most useful if a waveform of pCO_2 is displayed rather than a digital display of end-tidal pCO_2. Changes in the slope of the pCO_2 vs time graph at inspiration and expiration indicate changes in rate of inflow/outflow of gas. For example, if the curve flattens in the expiratory phase, this would be consistent with a partial expiratory obstruction.

While arterial CO_2 ($PaCO_2$) is a good indication of the ventilatory status of a patient, end – tidal CO_2 ($ETCO_2$) levels are a good indicator of $PaCO_2$ levels, usually being only 2 – 4 mm Hg lower than the actual $PaCO_2$ in most situations. Capnography therefore represents a valuable non-invasive monitor to assess the adequacy of ventilation in the anaesthetised patient. Most common capnographs are side stream gas monitors continuously drawing gas from the breathing system, usually from an adapter that connects between the endotracheal tube and the anaesthetic breathing system. In mainstream gas sampling, the sensor is inserted between the endotracheal tube and the anaesthetic breathing system. Mainstream sampling capnographs are more accurate with small patients. Capnographs allow continuous measurement of the CO_2 content of the sampled gases. Arterial blood gas analysis will give a more accurate measurement of blood carbon dioxide and oxygen levels but this is invasive in comparison, expensive to perform, requires appropriate equipment and arterial blood collection requires some skill. The normal range for $PaCO_2$ is between 37 ± 3 mm Hg in the dog and 31 ± 6 mm Hg in a cat with $ETCO_2$ values 0 – 5 mm Hg lower. Levels of $ETCO_2$ above these values indicate hypoventilation.

Inspired CO_2 should always be zero. Inspired CO_2 levels above zero indicate a anaesthetic breathing system issue - the patient is rebreathing its own expired gases. One possible cause is that the soda lime is exhausted.

Hypoventilation is very common in the anaesthetised patient due to the respiratory depressant effects of the drugs used. All aspects of ventilation are depressed but the most significant is the respiratory centre's sensitivity to CO_2. Increasing levels of $PaCO_2$ from normal results in physiological derangement in a dose related fashion. Respiratory acidosis and sympathetic nervous system stimulation result from increasing $PaCO_2$ above normal. Initially vasoconstriction will be seen due to the increased sympathetic tone but as the $PaCO_2$ continues to increase, vasodilatation will result. Increased $PaCO_2$ levels resulting in cerebral vasodilatation can be particularly devastating to the patient with intra-cranial disease &/or raised intra-cranial pressure. A $PaCO_2$ in excess of 60 mmHg in the dog and 50 mmHg in the cat may be associated with excessive respiratory acidosis and warrants instigation of positive-pressure ventilation.

The end-tidal CO_2, the inspired CO_2 and the respiratory rate are usually displayed on the capnograph. Most machines will sound an alarm after a period of apnoea e.g. 30 seconds. Many monitors display a continuous digital waveform.
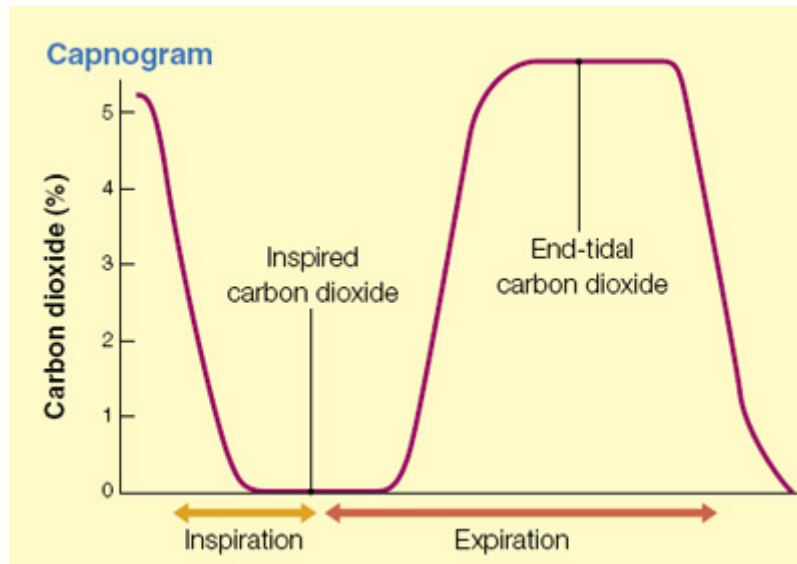
A typical capnograph waveform**.**

h)   Agent monitors

An agent monitor is a device that measures the anaesthetic vapour content of the breathing circuit gases. Gases are usually sampled at the connection between the breathing system and the patient's endotracheal tube. Many of the devices available measure oxygen and nitrous oxide as well as anaesthetic vapour.

Knowing the concentration of anaesthetic vapour is of particular value if the patient is connected to a circle absorber with the vaporiser in circle (VIC) or VOC with a low fresh gas flow rate. In these situations, the anaesthetic concentration cannot be predicted.

THE PARALYSED PATIENT

If the patient is given a *muscle paralyzing drug*, the anaesthetist must be aware that *some of the parameters usually assessed to determine anaesthetic depth are no longer valid*.

The paralyzed patient will not be capable of any voluntary muscle movement.

The *muscles of respiration will be paralysed* and the patient will be ventilated by IPPV, so there will be *no change in respiratory rate, depth or character* should the depth of anaesthesia be inadequate. Similarly, the patient will be *unable to move in response to painful stimulus*.

The anaesthetist must rely on signs that are associated with *the autonomic nervous system*.

- *Lacrimation, salivation and rhinorrhoea* may signify awareness in the paralyzed patient.
- *Cardiovascular response* to awareness can vary. Tachycardia and hypertension are most likely, but bradycardia and hypotension are not uncommon. Mucous membranes may blanche.
- *Pupils* may constrict or dilate in response to pain/distress.

ANALGESIA

Introduction

Definitions

Analgesia is absence of or relief from pain.

Anaesthesia is the absence of all sensory modalities.

Pain is "An unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage. Pain is always subjective" (Anon, The International Association for the Study of Pain)

Pain is a perception and therefore cannot be measured objectively. As stated by our former Dean, the late Professor Mike Rex, "Pain is what the patient says hurts".

Nociception is the reception, conduction and central nervous processing of nerve signals generated by the stimulation of *nociceptors*. In the conscious patient, perception of pain is dependent on this process.

A noxious stimulus is one that is actually or potentially damaging to the body tissues.

An anaesthetised patient can be affected by a noxious stimulus but will not perceive pain.

Assessment of Pain

Is there a place in pain management for subjective assessment? Pain is a subjective phenomenon, so subjective assessment may be as meaningful as objective. If an animal, by its position, demeanour or behaviour gives the appearance of being in pain, we can justify treating it for pain, even though the assessment is purely subjective.

Is there a place in pain management for anthropomorphism? It's all right to be anthropomorphic about pain. If it looks like it would hurt you, assume it hurts the animal. It is never possible, even with humans capable of describing pain, to measure how they feel. It is even less likely that a veterinarian will have a measure of how non-verbal patients feel. However, we do have some obliquely referable information. For instance, green-stick fractures are known to cause people severe pain. It would seem logical, therefore, to assume that a dog with a green-stick fracture would require treatment for pain.

Pain scoring systems. Pain assessment has been the subject of extensive investigation in human medicine for the comparison of different analgesic regimens, as well as for contribution to medico-legal proceedings. (The term "jurisgenic" pain has been coined to refer to pain the assessment of which could affect the outcome of legal proceedings!) The *McGill Comprehensive Pain Questionnaire* is a lengthy, detailed document to be filled in by patients with pain. This is of no use to us as veterinarians. *Visual analogue pain assessment scales* can be used by literate and non-literate patients to "score" their pain. Patients simply mark on a line where their pain lies, with 'no pain' represented by one end of the line and 'worst imaginable pain' by the other end. These systems are of some use to the veterinarian who can score for their patient, based on their observations, both subjective and objective. Similarly, *numerical scoring systems* can be used, after assignation of meaning to the numbers. McIlvaine *et al* (1988) devised a pain scoring system for use with human paediatric patients which has been adapted for use in veterinary practice by Thompson and Johnson (1991). Scores are given for heart rate, crying, movement and agitation to give possible total scores of 0 – 10, from pain free to severe pain. This represents a mix of objective (eg heart rate increase) and subjective (eg agitation). There are now many scoring systems that have been devised for use with veterinary patients, notably the Glasgow Composite Pain Scoring System (Reid *et al*, 2007) Pain research is often carried out using laboratory rats, for which testing systems and the interpretation of responses are clearly defined.

Nociception in the anaesthetised patient. If pain is a conscious perception, why give analgesics to the anaesthetised patient? In 1936, Hans Selye published an article in Nature entitled "A syndrome produced by diverse nocuous agents". He is credited with being the first person to realise that stress, in itself, is injurious. Although an anaesthetised patient is unable to *perceive* pain, there are *still metabolic, immunological, hormonal and haemodynamic responses to noxious stimuli* – the nociceptors still signal the brain that there is potential for injury, and a response is mounted. There is clinical and experimental

evidence *that recovery from surgery is better if adequate antinociception is provided in the pre-, intra- and post-operative phases*. If analgesics are given *before the start of surgery* (called *pre-emptive analgesia)*, then subsequent *pain control is far easier* than if the patient is allowed to wake up in pain.

METHODS OF PROVIDING PERI-OPERATIVE ANTINOCICEPTION

Opioids   *Naturally occurring opioids* from the opium poppy, *Papaver somniferum*, were used by man for analgesia in ancient Egypt. . The incised surface of the unripe seed exudes a milky fluid, opium, which contains at least 50 alkaloids, the major constituent being *morphine*. The use of opium dates from 3,500BC. The word *opium* is derived from the Greek *opion*, or poppy juice. The term *morphine* is derived from that of *Morpheus*, the Greek god of sleep.

Many of the alkaloids in opium have therapeutic uses eg codeine and thebaine. After thousands of years in use as an analgesic as well as for its soporific and euphoric effects, morphine is still the mainstay in treatment of severe pain. There have been changes in the way in which morphine is used, but not in the drug itself.

The opioids are also referred to as *narcotic analgesics*. The term *narcotic* is derived from the ancient Greek prefix, *narco*, meaning *to deaden or numb*. *Narcosis* has come to mean sleep.

All narcotic analgesics are subject to rigorous controls. As they are potential drugs of abuse, they are Schedule 8 or Controlled Drugs, requiring detailed records of amounts of drug held and all drugs to be locked in a safe. Health Department officials periodically check records, which must be properly maintained.

Other opioids : -

Pethidine HCl (Called meperidine (Demerol®) in North America)   Sufentanyl
Buprenorphine HCl   Carfentanyl
Butorphanol tartrate   Alfentanyl
Methadone   Alfentanil HCl
Codeine phosphate   Pentazocine
Fentanyl citrate   Hydromorphone
Oxymorphone (Available in North America - very popular in small animal practice.)

DRUG	POTENCY	DURATION	ACTIVITY
Morphine	*1*	*3 – 6 hr*	*$\mu > \delta$ &κ agonist*
Pethidine	*0.5*	*1 – 2 hr*	*μ agonist*
Methadone	*2*	*4 hr*	*$\mu > \delta$ &κ agonist and*
		some NMDA receptor activity	
Fentanyl	*100*	*20 min*	*μ agonist*
Buprenorphine	*3 – 5*	*3 – 12 hr*	*μ partial agonist*
Butorphanol	*2 – 5*	*¾ - 1.5 hr*	*μ antag & κ agonist*
Oxymorphone	*5*	*1 – 3 hr*	*μ & κ agonist*
Hydromorphone	*5*	*1 – 3 hr*	*μ agonist*
Naloxone	*-*	*45 – 60min*	*μ, κ & σ <u>antagonist</u>*

Terminology
1) *Agonist -*   The opioid drug binds to the receptor, producing maximum stimulation at that receptor. All effects are dose dependent. *(eg morphine, fentanyl)*
2) *Antagonist -*   The opioid drug binds to the receptor producing no stimulation, but effectively blocking the receptor to other opioids. *(eg naloxone)*

3) *Partial agonist* - – The opioid drug binds to the receptor but produces only weak stimulation thus achieving less than the maximum effect. They produce morphine-like effects at low concentrations are incapable of producing greater morphine-like effects at higher concentrations and therefore have a 'ceiling' effect. (Mather, 1986). *(eg buprenorphine)*

4) *Agonist/antagonist* – These drugs have agonist or partial agonist activity at one or more types of opioid receptors, have the ability to antagonise the effects of an agonist at one or more types of opioid receptors. *(eg nalorphine, butorphanol)*
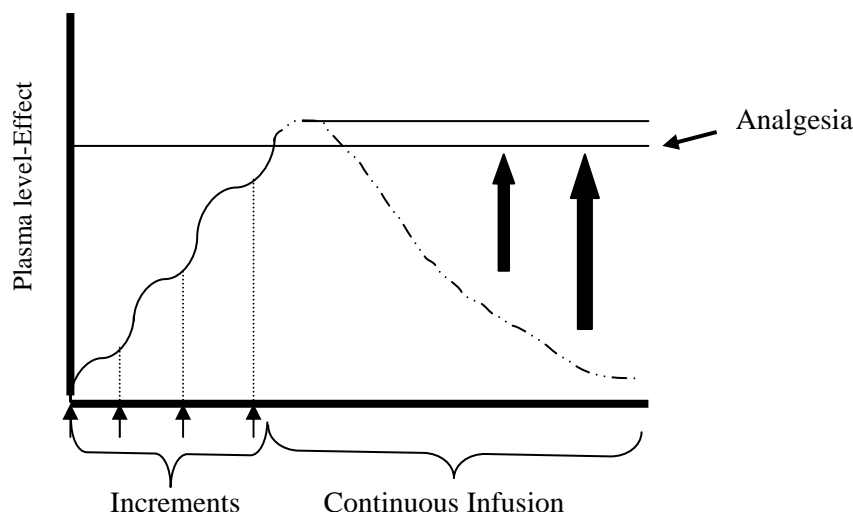
Routes of Administration
Systemic *Oral, S/C, I/M and I/V bolus or continuous rate infusion.*

Oral administration of opioids has increasing popularity in human pain relief because of the introduction of slow-release oral preparations of morphine and oxycodone. Problems of monitoring outpatients and scheduling restrictions make this form of dosing difficult in veterinary patients.

Administration by *injection* is most common. Frequency of injection can be established by referring to the table above, but mostly by monitoring the patients. Morphine can be *titrated* so that only that the amount required to achieve analgesic levels is given. This reduces the likelihood of respiratory depression and other side effects. Once analgesia appears to have been achieved, a *continuous infusion* can be used to maintain effective levels. As in all analgesic treatment in animals, defining success is a problem. Can we ever be sure that a) the animal was in pain in the first place, or b) that the animal is pain-free after treatment? Careful, constant monitoring must accompany morphine titration and infusion. Should respiration be depressed, naloxone can be titrated to reverse the respiratory depression (see Appendix 9). Respiratory depression can be assessed by objective measurement of end-tidal CO_2 (using a capnograph) or oxygen saturation (using Pulse Oximetry) if the patient is breathing atmospheric air or arterial blood gases.

A patient on an opioid infusion will benefit from insertion of a urinary catheter as bladder function will likely be affected.

Increments   Continuous Infusion

Epidural Opioids
There are large numbers of *opioid receptors in the dorsal horn* of the spinal cord. It has been shown that analgesia can be achieved by administration *of some opioid agonist drugs into the epidural space*. The advantages of this technique are that long-term *pain relief can be achieved without muscle paralysis or weakness, and without significant haemodynamic effects*. A single dose of 0.05 – 0.2 mg/kg of morphine administered into the epidural space will have an onset time of 30 – 60 minutes, and duration of action of 10 – 24 hours. It appears that there is considerable individual variation in requirements of morphine administered epidurally, so it may be preferable to insert a catheter and administer the

morphine by small increments, until the patient appears to be pain free. Epidural catheter placement is not without risk and requires some skill.

Local application   There has been considerable research into *peripheral opioid binding sites*. Most of this research has been on inflamed rat paw tissue. There are opioid receptors that appear in the periphery *in response to an inflammatory insult*. Opioid binding sites have been identified and characterised in normal human *lung tissue*, as well as normal and inflamed rat lung tissue (Cabot *et al*, 1994). Morphine binding sites appear on cultured human *T-lymphocytes* after stimulation with interleukin-1, an inflammatory cytokine (Roy *et al*, 1996). Similarly, normal *canine stifle joint* tissue has very little morphine binding ability, but 12 hours after an inflammatory insult, there is a high density of morphine binding sites (Keates et al 1999). The binding sites of these T-lymphocytes, lung tissue and inflamed joint tissue appear to be the same, having low affinity, high density and preferentially binding morphine (a plant alkaloid) over peptide opioid ligands. It is likely that in all these cases, the identified opioid binding sites are situated on immunocytes, hence their appearance in inflamed tissue. The airways have a high resting population of immunocytes due to constant barrage of antigens.
Clinically, administration of intraarticular morphine to human patients has been shown to provide analgesia of slow onset and long duration. Nebulised morphine causes dilation of the airways in human patients with pulmonary neoplastic disease. It would appear that peripheral administration of opioids has potential for clinical use in veterinary patients. Early trials in horses and dogs have produced encouraging results.

Intracerebroventricular morphine In some human cancer patients, with pain emanating from the head and neck, morphine is administered directly into the cerebral fluid surrounding the brain. This is of particular use in patients who have become tolerant to morphine. The production of metabolites of morphine in the liver (morphine-3-glucuronide, a μ-opioid antagonist) is instrumental in development of tolerance and direct injection of morphine into the CSF avoids liver metabolism. A well is created through the skull into the CSF, and the skin closed over. Very small doses of morphine administered once daily have had astounding results. The side effects are minimal as very low doses are used. There are no reports of ICV morphine in veterinary patients.

Transdermal Opioid Patches   Fentanyl is available in transdermal patches, labelled according to the rate of release and absorption of fentanyl. Allow 12 - 24 h for effect in dogs and 6 - 12 h in cats. Patches last about 3 days from onset of action. They should be applied to clean dry skin, covered with a bandage and the bandage labelled with time and date of application and size of patch. Absorption of drug increases with temperature, eg inflamed skin, pyrexic patients, patients on heating mats. Great care must be taken with the securing and disposal of patches as oral mucosal absorption is very high if the patch is mouthed by puppies (or children). Patches can be cut or covered to ensure the dose applied is appropriate.

Species	Weight (kg)	Fentanyl release rate/patch	Fentanyl contents/patch
Cats		25 – 50 ug/h	
Dogs	3 – 10 kg	25 ug/h	2.5 mg
"	10 – 20 kg	50 ug/h	5 mg
"	20 – 30 kg	75 ug/h	7.5 mg
"	> 30 kg	100 ug/h	10 mg

A variety of other opioid transdermal patches are available for use in human patients eg buprenorphine, morphine. Buprenorphine patches are used at the UQ Veterinary School.

Non-Steroidal Anti-Inflammatory Drugs   The non-steroidal anti-inflammatory drugs *act at the site of tissue injury* to *decrease the production of inflammatory mediators*, notably *prostanoids*, which facilitate the production and transmission of sensory impulses that *give*

rise to the perception of pain. Apart from reducing pain, these drugs also *suppress* the development of the other characteristics of inflammation, *swelling and redness.* It is now established that the NSAID's also act centrally.

Historically, the NSAID's have been used in patients suffering *chronic* pain. There is increasing use of NSAID's in surgical cases. A combination of an *opioid with an NSAID* is often administered. It is advisable to administer the NSAID post-operatively as there has been some suggestion that hypotension associated with many anaesthetic events may predispose the patient to the renal and gastro-intestinal side effects associated with NSAID administration. .

Side effects encountered after administration of NSAID's *include gastric and intestinal ulceration* with *secondary anaemia and hypoproteinaemia. Platelet function* may be impaired. There is a risk of *nephropathy* in patents with conditions in which renal perfusion may be reduced eg hypovolaemia, dehydration or congestive heart failure. Chronic or repeated use of NSAID's can result in *chronic interstitial nephritis and renal papillary necrosis. Blood dyscrasias* have been reported. Remember, it may be that the **gastrointestinal and renal side effects are more likely if the patient is hypotensive** due to cardiovascular side effects of *anaesthetic agents*.

α_2-adrenergic agonists. The α_2-adrenergic agonists (eg xylazine, detomidine, medetomidine and romifidine) are generally considered to be sedative-hypnotics and are most commonly given to produce sedation. However, they are also potent analgesics. Xylazine is considered to be a more powerful analgesic in equine colic cases than opioids or non-steroidal anti-inflammatory drugs.

The α_2-adrenergic agonists can be administered *systemically* or *epidurally* for their analgesic activity. However, if they are administered systemically, sedation, as well as the documented side effects, notably cardiovascular depression, will accompany analgesia. There is some interaction between the α_2-adrenoreceptors and opioid receptors.

Local anaesthetics Techniques for the administration of local anaesthetics will be covered later in the chapter on Local Anaesthesia. Many surgical procedures are carried out in conscious animals (particularly large animals) with a combination of sedation/tranquillisation and local anaesthesia.

1.   In *small animals anaesthetised for surgery*, the application *of local anaesthetic blocks* will *reduce the anaesthetic requirements and the need for post-operative analgesic drugs*.
eg
- Epidural anaesthesia in a dog anaesthetised for hind limb surgery
- Maxillary and mandibuloalveolar nerve blocks for dental extractions

2.   Patients having undergone thoracotomy will often show dramatic improvement following administration of *local anaesthetic via chest drain into the pleural space (beware – rapid absorption, systemic toxicity)*.

3.   *Injection of local anaesthetic into a fracture site* can be an effective way to relieve pain, but care must be taken with sterility.

4.   *IV lignocaine* has been shown to reduce severity of post-operative pain in humans and animals without causing problematic side effects. Lignocaine can be delivered as a single injection, or as a continuous infusion. A loading dose of 2 mg/kg followed by an infusion of 40 µg/kg/min is a useful adjunct to other analgesic administration.

Immobilisation of fractures. Immobilising bandages can reduce the pain associated with fractures.

Nursing   Good nursing contributes to the well being of patients suffering from trauma and post-operative pain. Remember that patients in pain may be reluctant to urinate or defaecate due to pain when positioning – this can result in extreme discomfort and distress. It may be advisable to place a urinary catheter in patients immobilised after surgery to ensure bladder emptying. Keeping the patient quiet, warm, comfortable and well hydrated will contribute to the well-being of the patient.

Refer to Appendix 13 (p139) for further suggestions and dosing regimens.

Refer to Appendix 15 (p144) for details of epidural anaesthesia in cats and dogs.

SPECIAL CONSIDERATIONS WITH RESPECT TO SPECIES

ANAESTHESIA OF THE CAT.
- Cats are anaesthesia candidates worthy of respect. An apparently docile and affectionate cat can become *intractable* after one failed attempt at IV injection.
- Cats belong to that group of animals that are prone to *laryngospasm*.
- During very light anaesthesia, cats have very active vagal reflexes which can be triggered by stimulation of the nose, eyes and especially the pharyngeal tissues. The result can be laryngospasm and/or cardiac arrest.
- Mucosal oedema and obstruction can occur in response to traumatic attempts at intubation.
- Often, cats will ventroflex their heads during recovery from anaesthesia, causing *obstruction* of the upper airways.
- Being small in size, cats are prone to *hypothermia* during long periods of anaesthesia.

Restraint   Most cats can be given an intravenous injection with gentle and subtle restraint. Some cats require scruffing and firm restraint. Rarely, they will not tolerate handling at all and must be anaesthetised by other means. eg by IM injection through a wire cage or by placing the cat in its cage into a large *clear* plastic bag that is connected to a supply of oxygen and an inhalation anaesthetic agent. (Be sure that you can see the cat at all times – respiratory obstruction is likely as the cat loses consciousness.)

Intravenous injections   Commonly, the cephalic vein is used. (Also, the dorsal pedal vein, the recurrent tarsal vein and the jugular vein) With light restraint and a very sharp needle, most cats can be given an intravenous injection of anaesthetic agent successfully. The importance of using short, sharp needles cannot be over-emphasised. Twenty-six or 27 gauge, 0.5" (12 mm) needles are well tolerated by most cats. Should repeated injections be necessary, the choice of fine needles minimises the likelihood of haematoma formation.

Endotracheal intubation   Bearing in mind that cats are prone to laryngospasm, it is wise to give oxygen by mask to cats as soon as they have lost consciousness. Laryngospasm is less likely to occur in a cat that is well oxygenated, and should laryngospasm occur, the well oxygenated cat is likely to survive whilst the anaesthetist secures an airway.
Precautions must be taken to avoid spasm during intubation. If the arytenoids are touched, a reflex may be elicited that results in the closure of the glottis. The reflex can be blocked on the afferent or the efferent side, or it can be obtunded.
- The afferent side of the reflex can be blocked by *spraying the mucosa with local anaesthetic* (~ 0.5 ml 2%) and waiting for 20 – 30 seconds before intubating. This is the *most common* method. *Note that the arytenoids will usually react to spraying by 'fluttering' movements and closure of the glottis is possible.*
- The efferent side of the reflex can be blocked by using a *neuro-muscular blocking agent*. Suxamethonium can be used as it has a very short duration of action in the cat. (Not so in the dog, where it lasts for about 20 minutes) At a low dose rate (0.2mg/kg) it usually does not stop the cat's respiration. However, if this method is employed, *preoxygenation is essential, intubation must be successful* and the cat *must be ventilated* if it suffers a period of apnoea.
  Do not attempt this until you are skilled at intubating cats.
- The cat can be taken to *deep planes of anaesthesia* so that the reflex is obtunded. Whilst the *risks of respiratory and cardiovascular depression must be recognised, it is usual to take cats to a somewhat deeper plane of anaesthesia as compared with dogs in which laryngospasm is unlikely.*

Should *laryngospasm* occur, an *airway must be secured rapidly*, or the cat will die.

- A larynx in spasm may not result in complete closure of the glottis.*. **If there is any respiratory noise, there is some airflow. The louder the noise, the smaller the opening. However, complete obstruction is quiet.** If there is some airflow, give oxygen by mask for a short time before attempting intubation with a small diameter tube. If there is a small opening, it is likely to be ventral.
- If laryngospasm causes total occlusion, a *tracheostomy must be performed*. An emergency tracheostomy can be achieved using an *18G needle or catheter* through the skin into the trachea. A needle hub will attach to a 3.5mm endotracheal tube connector, so oxygen can be delivered from the breathing circuit. Although this is a very small airway, it is sufficient to allow emergency oxygenation until the spasm passes. **Care must be taken to avoid laceration of the trachea if a needle is used. A catheter is much safer.**

Endotracheal intubation can be performed with the cat in *dorsal, ventral or lateral recumbency*. My preference is dorsal recumbency using the method described below. With the tongue gently pulled forwards, the tip of the blade of the laryngoscope is placed *in front of the epiglottis* (not touching it) and the blade is used to lift up the mandible. This method gives a very clear view of the larynx and usually permits the passage of an uncuffed tube of 5 – 6 mm diameter. **Always preoxygenate the cat.** The following description assumes the anaesthetist is right handed: -

- Have the cat held in dorsal recumbency with its head hanging over the edge of the table.
- Hold your left hand with the back of the hand towards you, thumb down.
- Pull the cat's tongue forward and hold it between the second and third finger of the left hand. The tongue will hold the cat's lower jaw up. (Remember, the cat is upside-down.)
- Use the fourth finger of the left hand to push the hard palate down. ie push the top jaw down. Your finger will be positioned just behind the canine tooth.
- Using the right hand, place the laryngoscope (with handle upwards) so that the tip of the blade is just *in front of* the cat's epiglottis.
- Pass the handle of the laryngoscope to the left hand and hold it between the thumb and the second finger.
- With your hold as described, gently elevate the pharynx with the laryngoscope blade while extending the neck towards you.
- Your right hand is free to spray the larynx with lignocaine (0.5 ml 2% lignocaine onto the arytenoids).
- Wait 20 – 30 seconds for the lignocaine to act. Give the cat oxygen by mask during this time. Then replace the laryngoscope as described.
- Insert the endotracheal tube with the right hand with a slight gentle twisting motion.

Once mastered, this technique is simple and allows the insertion of uncuffed endotracheal tubes of 5 – 6 mm diameter in most cats.

In the breathing systems commonly used for cats, the greatest source of resistance to airflow is the endotracheal tube. Hence, it is important to place the largest tube that can be inserted easily (no force!). To ensure adequate internal diameter of the tube, large uncuffed tubes can be used. The external diameter of a 4 mm cuffed tube can be the same as that of an uncuffed 6 mm tube.

Premedication I prefer to premedicate cats by subcutaneous injection. Most cats will tolerate one intravenous injection, but some cats are not so cooperative when faced with a second. A subcutaneous injection of tranquilliser/sedative (with or without an opioid) should render the cat more tractable, and improve the chance of a successful intravenous injection of an induction agent. Allow about 20 minutes for the premedication to take effect. There are many drugs/combinations of drugs available for use in cats. ***Remember that ketamine stings!***

Eg

- acepromazine (0.02 – 0.05)/pethidine (1 – 2 mg/kg) S/C
- acepromazine (0.02 – 0.05 mg/kg)/methadone (0.2 – 0.5 mg/kg) S/C

- acepromazine(0.02 – 0.05 mg/kg)/ketamine (2 – 5 mg/kg) S/C
- ketamine(2 – 5 mg/kg)/diazepam (0.1 – 0.5 mg/kg) S/C
- ketamine (2 – 5 mg/kg)/ midazolam (0.1 – 0.5 mg/kg) S/C
- acepromazine (0.05 mg/kg)/methadone (0.5 mg/kg)/ketamine (2 – 5 mg/kg) In one syringe, S/C. For a nasty cat!
- alfaxalone 1 mg/kg IM

If a cat cannot be handled at all with safety, an injection may be given through the bars of cat cage by tipping the cage on its side so that the cat is sitting on the wire. A needle may be inserted into the cat's abdomen through the cage wire.

Alternatively, a mixture of ketamine and acepromazine can be squirted into the cat's open mouth as it "hisses".

Instead, it may be prudent to proceed, without premedication, straight to general anaesthesia by placing the cat in a cage, enclosing the cage in a *clear* plastic bag and delivering an inhalation agent (halothane, isoflurane, sevoflurane) in oxygen into the bag. It is imperative that good visibility is maintained, and that the cat is removed from the cage as soon as it has lost consciousness. Tip the cage to establish that the cat has lost its righting reflex. Make sure that the cat's airway is not obstructed by its position. Once the cat is removed from the cage, oxygen and anaesthetic agent can be delivered by mask (or by intravenous injection) until the depth necessary for intubation is achieved.

Induction   Most cats will tolerate an intravenous injection. Thiopentone, propofol, a combination of ketamine/diazepam, Zoletil$^®$ (tiletamine and zolazepam) and alfaxalone$^®$ are commonly used.

Remember that ketamine and Zoletil$^®$ sting if given by subcutaneous injection.

Maintenance   For lengthy procedures, inhalation agents (eg halothane, isoflurane, sevoflurane) or non-cumulative intravenous agents (eg propofol, alfaxalone) can be used.

Recovery from anaesthesia   Whilst recovering from anaesthesia, many cats ventroflex their heads and occlude their airways. *Close supervision of cats during recovery is essential.* It may be necessary to *extend the head and neck* and gently *pull the tongue* forwards to maintain a patent airway until the cat is more aware.

NSAID's   Many of the NSAID's commonly used in dogs CANNOT BE SAFELY USED IN CATS. For some NSAID's, dose rates for cats are published, but these take into account the much slower clearance rates in cats (due to absence of some metabolic pathways) and therefore increased dosing intervals. Ketoprofen, carprofen and meloxicam are licensed for limited use in cats (eg one dose only).

Paracetamol is toxic to cats.

ANAESTHESIA OF THE DOG

General considerations

As a species, dogs have few peculiarities that make that make them particularly risky candidates for anaesthesia. *Intravenous injection is tolerated well by most dogs*. Most breeds have *easily visualised glottal openings* and they *do not suffer from laryngospasm* when stimulated while under light planes of anaesthesia.

However, wide breed diversity results in some dogs having extreme inherent risk factors.

Size   Adult dogs vary from <1 kg to >100 kg. Metabolic rate is related to body mass to surface area ratio, the *dose per kilogram* is likely to be higher in a 1 kg animal than a 100 kg animal. However, the small doses required for a 1 kg animal must be delivered with much greater accuracy. A small volume error may be a large *percentage error*. Eg if the total volume to be delivered to a small animal is 0.05 ml, then an extra 0.05 ml s a 100 % error. If the same volume error is made in a 100 kg dog, it may be an error of 0.05ml in, say, 2ml, an error of 2.5 %. *Use insulin syringes for small volumes. They have no 'dead space' and are far more accurate with small volumes, especially if drugs are mixed in the syringe.*

Small animals are more at risk of becoming *hypothermic* than large animals. (Heat loss is related to the ratio of body mass to surface area)

Brachycephalic dogs   Relative nose length varies according to breed.   In dogs with long noses, (eg German Shepherds, Collies etc) the glottis is easily visualised and correct placement of endotracheal tube is easy.   In the *brachycephalic breeds*, (eg boxers, pugs, shitzhus, pekes etc and especially British Bulldogs) *visualisation is difficult* and the *tracheal diameter is small*.   Brachycephalic dogs are *predisposed to airway obstruction* on *induction* and *recovery*.   Some families of *boxer dogs* have particular *sensitivity to the phenothiazine derivatives* eg acepromazine.   Such dogs can suffer *syncopal episodes* as a result of *severe bradycardia*, a direct vagotonic effect of acepromazine.

Metabolic differences

The *sight hounds* (greyhounds, whippets, salukis, Irish Wolfhounds etc) are *lacking in an enzyme important in the breakdown of the thiobarbiturates*. Thiopentone is best avoided in these species. Propofol, alfaxalone or ketamine with a benzodiazepine may be useful in these dogs.

Owners will frequently report breed peculiarities with respect to anaesthesia, but supportive scientific literature is often difficult to find.

Premedication   Most dogs tolerate restraint and intravenous injection, which means that, in the main, there is a choice of route of administration of premedication. Care must be taken with administration of opioids. *Pethidine,* in particular, can lead to *histamine release*. If pethidine is administered SC, some dogs behave as if they have been stung by a bee. This is most likely due to a local histamine release. However, if pethidine is administered IV, a systemic histamine release may precipitate generalised vasodilation and a profound drop in blood pressure. These episodes may be well tolerated in a fit and healthy dog, but may be life threatening in a dog with cardiac disease. ***Do not administer pethidine IV.***

If time permits, it is probably *best to administer the premedication by subcutaneous injection* and *wait for 20 minutes* before proceeding with induction of anaesthesia.   The less stimulated the animal is during this period, the more effective will be the tranquilliser/sedative. If possible, the animal should be left in a *quiet cage in a supervised low traffic area.*

If the dog is aggressive and injection is difficult, the following graded responses can be tried.

- Hold the skull of the dog, not the skin
- Apply a well-fitting muzzle
- Control the dog with two chains – one out each side
- Restrain with a "dog-catcher" (a pole supporting a noose)
- Apply a "crush" technique

Drugs that may be of use in savage dogs

- Acepromazine (0.05 – 0.1mg/kg) + Morphine or methadone (0.5 -1mg/kg) IM or SC

- Xylazine 1 -2 mg/kg IM or SC (unpredictable results, CV side effects)
- Medetomidine (0.001 – 0.002 mg/kg) + morphine (0.2 – 0.6 mg/kg) IM or SC (both of these drugs are reversible)

Intravenous injections   The *cephalic vein* is appropriate for intravenous injection in most cases, but there are other easily accessible veins. The *recurrent tarsal vein* is large and easily located, but is highly mobile. It is situated very close to the hock joint, so securing a catheter in a small dog may be difficult. The veins on the *dorsal surface of the metatarsals* are large, easily accessible and ideal for catheter placement as the catheter is "splinted" by the metatarsals. An aggressive dog may be threatened by an approach to the head end necessary for cephalic vein injection, but may tolerate an injection away from its head end.

Induction   Once the dog has been sedated, intravenous injection is usually feasible in most dogs. Thiopentone, propofol, alfaxalone and ketamine/diazepam are commonly used. Don't forget that ketamine is convulsogenic in dogs, and should be used only after administration of a drug that decreases muscle tone, such as diazepam, midazolam or an α_2-adrenergic agonist.

Intubation   *Good visibility for intubation* can be obtained with the dog's head elevated, but this can cause *orthostatic hypotension, resulting in poor cerebral perfusion*. The dog is best *in lateral recumbency*, with the *head and neck extended*. With the tongue pulled forwards over the lower incisors, the epiglottis can be depressed, giving a clear view of the opening of the larynx. The tip of the endotracheal tube should be inserted and the tube advanced with gentle twisting if necessary. Always make sure that the *tip of the tube is cranial to the thoracic inlet* to avoid the possibility of intubation of one bronchus. This can be achieved by first holding the tube beside the dog to estimate where the tip of the tube will reach or by very gentle palpation after the tube has been inserted into the trachea.

Maintenance As for cat.

Recovery   Except for dogs especially predisposed to *obstruction* (eg brachycephalic or dogs with tumours in the oropharynx), dogs are not particularly likely to suffer airway obstruction. Care must be taken with maintenance of *body temperature* in small dogs, or dogs that have undergone protracted procedures.

As always, recovery is as safe as it is supervised.
Never leave any patient unattended until is awake enough to swallow, the tube has been removed and you have checked that there is unobstructed airflow.

Gastric reflux   It is not uncommon for *gastric contents to enter the oesophagus* during anaesthesia. This is often *undetected*, as they do not reach the mouth. If the stomach is empty before anaesthesia, the *pH of the gastric secretions is likely to be particularly low*. If this problem is detected, the oesophagus should be *irrigated with normal saline and a solution of sucralfate (Carafate®) left in the oesophagus*. Possible sequelae to this event if untreated are *oesophageal inflammation*, *scarring* and *stricture*.

EQUINE ANAESTHESIA – General considerations

Anaesthetic morbidity/mortality In a large multicentre study of 42,824 cases of horses undergoing anaesthesia, the overall *mortality rate was found to be 1 – 2%.* (Johnston *et al, 2002).* This figure refers to problems encountered in the period covering premedication, induction, maintenance, recovery and the post-operative period. The survey included all cases, including high-risk cases such as those with colic. Compared with other domestic animals that commonly undergo anaesthesia, there is a high incidence of *traumatic injuries* associated with induction and recovery. A horse must be taken from being able to stand relatively steadily to being recumbent in a very short time as a horse that is unstable is likely to injure itself and those handling it. This rapid induction of anaesthesia is not without risk. A horse has a *high centre of gravi*ty and a *large mass*, so *fractures of the limbs and spine* can occur on induction or recovery. Horses are "flight" rather than "fight" animals and tend to *panic and flee* if they become excited after premedication or during induction of anaesthesia. If a horse lightens whilst on the surgery table, it may begin to "gallop", injuring both itself and personnel.

These risks, combined with the cardiopulmonary changes that accompany anaesthesia and recumbency, account for the high morbidity and mortality rates in equine anaesthesia.

Size Horses vary in size from young foals at less than 40kg to heavy horses at more than 900kg.

Large size provides difficulty in both awake and asleep horses

- Injuries can be related to getting up or down, or to the animal's weight on the soft tissues.
- Moving / positioning horses for surgery may be difficult once they are recumbent

Mask induction can be the method of choice in very young foals small enough to be held still. (mask or nasal ET tube).

Resistance to breathing is not well tolerated by horses. Therefore, wide bore tubing is necessary in endotracheal tubes (23 – 40 mm) and anaesthetic machines (50 mm).

Breed Excitable breeds eg thoroughbred (cf draft or quarter horse) will require more sedation and therefore may be expected to have longer recoveries.

Weight Mechanical aids (pulleys & winches) or many handlers may be required to shift a horse once anaesthetised.

CARDIOPULMONARY DISTURBANCES (Taylor & Clarke, 1999)

Hypotension

All volatile anaesthetic agents cause vasodilatation and myocardial depression. The effects of halothane are more pronounced than those of the newer inhalation agents (eg isoflurane, sevoflurane). Low cardiac output and consequent poor tissue perfusion is one of the most serious detrimental effects of general anaesthesia in equine patients. Simple monitoring (auscultation, palpation etc) does not effectively measure blood pressure. Direct arterial blood pressure measurement is relatively simple in horses. The dorsal pedal and the facial arteries are easily accessed in horses so that direct blood pressure measurements can be made.

Many anaesthetised horses, especially those under inhalation agents are treated with positive inotropes to maintain mean arterial blood pressure above 70 mm Hg. Dobutamine can be given by IV infusion at rates between 1 and 5 µg/kg/min. Dobutamine is a β-adrenergic agonist and as such, increases the force of cardiac contractility and to a lesser extent, increases heart rate. The effect of IV dobutamine is very rapid. Ephedrine has both ά- and β-adrenergic effects. It increases both rate and contractility and also provides some pressor effect. A bolus dose of 0.05 – 0.1 mg/kg will last approximately 20 – 30 minutes.

Cardiac rhythm disturbances

Bradycardia is often encountered in anaesthetised horses – especially fit thoroughbreds. Bradycardia is associated with low cardiac output and hypotension, and can lead to ventricular fibrillation

Most bradycardias respond to treatment with anticholergic drugs. If the patient's heart rate is less than 25 beats/min, then anticholinergics are indicated.

Second degree AV block is not uncommon, particularly after alpha$_2$ adrenergic agonist administration.

Atrial fibrillation: - occurs occasionally. Give fluids to improve ventricular filling. Avoid anticholinergics ($\uparrow$rate, $\downarrow$ventricular filling time). Maybe give dobutamine.

Ventricular dysrhythmias: - May be due to toxaemia, sympathetic stimulation, hypoxaemia, hypercapnia, electrolyte disturbances. Treat with IV lignocaine 0.05 mg/kg. Repeat if necessary up to a total of 0.2 mg/kg.

Hypoxaemia

Horses tend to become hypoxaemic when anaesthetised even when there is a high inspired O_2 concentration. In the *standing horse*, the *difference in pO_2 between alveolar gas (A) and arterial blood* (a) is about *18mmHg ($pAO_2 > paO_2$)*. Once the horse is *recumbent*, the difference between *arteriolar tension and alveolar oxygen tension increases and may even double.* The increase in (A-a) pO_2 is *associated with an increase in pCO_2*, suggesting that this effect is simply hypoventilation, but *IPPV does not significantly reduce the difference* even if the pCO_2 is normalised. *Hypoxia can occur during recovery even if O_2 is supplemented*. One causative factor is that, because a horse's diaphragm is far from vertical, the weight of the viscera markedly compresses the lungs, contributing to shunting (perfusion of lung tissue that is not ventilated) and mismatching (inadequate ventilation of lung tissue resulting in partial oxygenation of the blood). Simply, a considerable proportion of lung tissue is either underperfused or underventilated so that the blood leaving the lungs to enter the systemic circulation has reduced oxygen content.
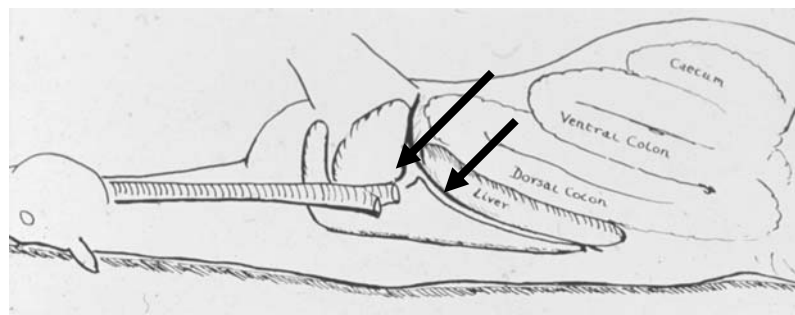
Summary of factors contributing to hypoxaemia in the anaesthetised horse: -

1. *Pulmonary shunts* (blood perfusing lung tissue that is not involved with gas exchange). *Ventilation of lower lung is restricted* due to size of animal and weight of viscera. The level of *pulmonary shunt* is approximately *5% in standing, awake horse* and approximately *14% under halothane anaesthesia*. During general anaesthesia, a *horse's functional residual capacity is so reduced* that *airway closure* may occur. If *gas is trapped in an alveolus*, O_2 *is progressively absorbed* and the blood that perfuses that alveolus contributes to *the increased intrapulmonary shunt*. This is more of an issue if the patient is inspiring 100% oxygen. X-rays of recumbent horse lungs show that lower lung volume is reduced. The changes are suggestive of *alveolar collapse*, *oedema* and *pulmonary congestion*.

2. The effect of *gravity* is to cause *uneven distribution of blood flow to the lungs*. This occurs in all species, but the effect is more noticeable in large animals as the lungs are large and therefore the gradient is large. Maldistribution of pulmonary blood flow relative to ventilation occurs (*ventilation/perfusion mismatching*). The uppermost lung tissue will be over ventilated relative to perfusion and the lower lung will be over perfused and under ventilated.

3. As with all species, there is progressive *atelectasis* with time during anaesthesia. (Due to loss of the sigh reflex & compression of lung tissue). This is more marked in the horse as compared with most other species.

4. Anaesthesia without IPPV results in *respiratory depression* leading to *increased arterial pCO_2*. *Alveolar hypoventilation due to loss of sensitivity to CO_2,* (effect of sedatives, analgesics & anaesthetics and *decreased tidal volume* (relaxation of muscles of ventilation). As lower lung function is impaired, and the right lung is larger, it is advisable to have the patient in *left lateral recumbency if possible*. However, position is usually dictated by the procedure.

5. *There is a decrease in cardiac output without reduced tissue 0_2 consumption.* Halothane causes *decreased cardiac output* by decreasing both *stroke volume and cardiac contractility*. Isoflurane is associated with better cardiovascular stability than halothane. Cardiac output is even *further reduced if intermittent positive pressure ventilation is applied*. This may be due to *decreased venous return* resulting from *intermittent increase in intrathoracic pressure*.

6. *IPPV* can cause *lower pO_2 of mixed venous blood*. This may be due to *poor perfusion* because of decreased cardiac output (effect of IPPV on venous return), and therefore reduced rate of blood flow. ie *sluggish flow, more O_2 extracted*.

The major problems are related to compression of lung tissue*.*
It is important to measure haemoglobin oxygen saturation (pulse oximeter) or arterial blood gas tensions.

Measures to avoid hypoxaemia and tissue hypoxia under anaesthesia: -

What is worth doing: -

1. ***Keep anaesthesia time as short as possible***
2. *Deliver a high concentration of oxygen throughout the procedure and well into the recovery phase.* Monitor blood gases if possible – hypoxia can lead to arrhythmias and sudden death.
3. *Monitor blood pressure.* Aim to maintain a mean arterial blood pressure greater than 70 mm Hg. Administer IV fluids and, if indicated, positive inotropes (drugs that increase myocardial contractility eg dobutamine, dopamine or ephedrine).
4. Use of *light general anaesthesia with muscle relaxation and IPPV* will spare the cardiovascular system and result in better O_2 delivery to the tissues.
5. If possible, choose lateral recumbency rather than dorsal (rarely have a choice). Right lung up where possible.

Overall, get them up fast!

Measures that are of little use: -

- *Tilting to take weight of viscera* off the diaphragm is *not successful* as the diaphragm is conical and it is not practical to tilt enough. Tilting may result in problems with *ischaemia in hind limb muscles.*
- *Positive end expiratory pressure (PEEP) reduces alveolar collapse but any advantage gained may be counteracted by depression of cardiac output.*
- *Repeated turning* of the horse during recovery is *not safe!*

Postoperative myopathy.
This is a serious problem in horses which can cause life threatening lameness following anaesthesia. Hypotension during anaesthesia is considered to be a major contributing factor. Prolonged anaesthesia increases the likelihood. It is most common in horses with high body weight.
The problem usually becomes apparent early in the recovery period, but can develop up to several hours after the horse regains consciousness. Mostly, the muscle groups which have been compressed are affected ie those muscles on the 'down' side. In cases of fore

limb lameness, it can be difficult to differentiate between radial nerve damage and myositis. However, myositis is associated with hard, swollen, extremely painful muscles, and myoglobinuria.

Initially, the horse will appear distressed with laboured respiration and profuse sweating. On occasions, the horse will be unable to rise after the anaesthetic and will panic. Mostly, the diagnosis can be confirmed by measuring creatine kinase levels which peak within a few hours of the muscle injury. Muscle breakdown will also release myoglobin which results in red urine and can cause renal failure by blocking renal tubules.

Recumbency results in compression of blood vessels in muscle masses and therefore decreased arterial supply and stagnation of blood due to closed veins. This coupled with hypotension common with anaesthesia using volatile anaesthetic agents and long anaesthetic time results in a 'cycle of damage'. The muscle fascia around a muscle group is virtually non-distensible. As muscle cells are damaged and swell, the pressure within the muscle group increases, causing further perfusion failure. It has been established that the perfusion pressure for a muscle group must be about 30 mm Hg above the intra-compartment pressure for perfusion to be adequate. Normal compartment pressure (10 mm Hg) is increased to 35 – 65 mm Hg during recumbency. Thus, for perfusion to be adequate, the mean arterial pressure needs to be 65 – 95 mm Hg. Halothane, in particular, is often associated with mean arterial pressures much lower than this. (Taylor & Clarke, 1999)

Obstructed venous drainage can be as effective in preventing muscle perfusion as low arterial pressures. Take care when positioning limbs not to restrict venous drainage.

Low blood oxygen tensions will contribute to muscle injury. However it should be remembered that at low pH (as in ischaemic tissues) oxygen is extracted at much lower arterial tensions than at normal pH. This is due to a shift in the oxygen dissociation curve.

Prevention
- ***Avoid prolonged anaesthesia***
- ***Avoid hypotension*** (***measure blood pressure and treat hypotension!)***
- Ensure adequate oxygenation
- Raise limbs to reduce weight on muscle masses
- Pull lower forelimb forward with respect to the upper limb.
- Provide soft surfaces/padding eg water bed to ensure even weight distribution

Treatment

Once the problem has occurred, further problems can arise because of the horse's inability to stand, and also because of the eventual replacement of necrotic tissue by fibrous tissue.

Initially, the horse should be given analgesics (opioids, NSAID's), tranquillisers/sedatives (acepromazine if hydration is OK), detomidine, romifidine only if horse is very distressed – these drugs will decrease perfusion!). Large volumes of IV fluids should be administered (eg 20 – 40 l crystalloids). (Taylor & Clarke, 1999)

Likely causes of postoperative lameness
- Myopathy
- Fractures
- Brachial plexus avulsion
- Nerve injury

ANAESTHESIA - Procedures

Frequently encountered problems in anaesthetised horses.
- Apnoea or hypoventilation (with hypoxaemia and hypercapnia)
- Hypotension
- Bradycardia
- Arrhythmias
- Waking during procedure

Pre-anaesthesia examination

History: - The owner of the horse should be questioned about *previous anaesthetics* the horse has experienced eg drugs used, duration and quality of recovery etc. The horse's *exercise tolerance* should be established as well as history of *respiratory disease* and or *rhabdomyolysis*. *Respiratory noise* at rest or with exercise should be noted. This may herald upper airway obstruction once the horse is anaesthetised, especially on recovery after the endotracheal tube is removed.

Examination: -

Cardiovascular system: - Palpate *peripheral arteries*, check *colour of mucous membranes*, capillary *refill time*, assess *skin turgor* and *auscultate the* thorax. Second degree AV block is very common in healthy horses. This usually disappears if the horse is trotted out. Irregular heart rhythm should be investigated by ECG assessment if the diagnosis is uncertain. Heart murmurs are common in very fit horses and should be evaluated along with exercise tolerance. Frequent ventricular premature contractions, paroxysmal tachycardias or persistent ventricular tachycardia represent considerable anaesthetic risk and the horse should not be subjected to anaesthesia for elective procedures. (Hubbell, 2001)

Respiratory system: - The entire lung field on each side of the thorax should be auscultated with the horse in a quiet environment. Further diagnostic workup may be necessary.

Nervous and musculoskeletal systems: - The horse should be observed walking to establish absence of lameness.

Premedication Premedication in horses requires more "precision" than in small animals. The premedication can be titrated so that appropriate levels of sedation and muscle relaxation are ensured before induction. *Ataxia is dangerous for both the horse and the anaesthetist*.

Commonly used drugs: -
- acepromazine
- α_2-adrenergic agonists (xylazine, detomidine, romifidine)
- opioids (pethidine, butorphanol, methadone)
- benzodiazepines (diazepam, midazolam)
- guaifenesin

Induction A suitable environment is necessary before general anaesthesia is attempted in the horse. For field anaesthesia*,* a large, flat expanse, devoid of fences or other obstacles and preferably *grassed* is ideal. Purpose-built equine theatres have *induction/recovery rooms.* These rooms are ideally *padded*, both floor and walls, and are fairly *small* so that the horse cannot gather speed whilst it is regaining its feet on recovery.

A catheter should be inserted into the jugular vein for all inductions. Some commonly used agents are highly irritant if injected perivascularly eg thiopentone, guiafenesin. Secure catheters with Super Glue or with sutures. *(Leave the catheter in place until the horse has recovered from anaesthesia and is standing/walking steadily.)* Induction in horses has to be rapid, as the horse must go from being able to *stand steadily to being unconscious on the floor very rapidly.* A horse that is unstable standing is *dangerous to itself and to personnel.* If the horse becomes light while recumbent, it will often *paddle or "gallop".*

Intubation Intubation in the horse is performed *"blind".* The horses head is extended, mouth opened by pressure on upper and lower jaw, tongue gently pulled forward, and the tube inserted. If the tube is curved, the end of the tube should first curve up to *disengage*

the soft palate from the epiglottis, then the tip should be turned down towards the opening of the larynx. Gently manipulate the tube and change the degree of head extension until it advances with minimal/no resistance*. Oesophageal intubation offers more resistance than tracheal.* Horses have a *poorly developed cough reflex*, and usually do not cough as the tube enters the trachea. Tube sizes vary from 12 – 14 mm for neonates to 20 - 40mm in adult horses.

It is *important to seal the trachea*, so tubes are either *cuffed* or shaped with a *"collar"* which seals the opening to the larynx (Cole tubes). Cole tubes have been associated with arytenoid mucosal damage.

Maintenance Practices that regularly anaesthetise horses are likely to have *a large animal circle* absorber for the delivery of isoflurane. A circle absorber breathing system suitable for horses has wide bore tubing – about 5 cm diameter. Water's to & fro systems can be used in horse practice. *Total intravenous* anaesthesia is practiced, especially for short procedures in the field*. Supplemental oxygen is desirable in all anaesthetised horses because of the ventilation/perfusion problems already outlined.* A demand valve can be used to ventilate the patient with oxygen in the absence of a patient breathing system.

Monitoring   The equine patient should be monitored continuously with recordings made every 5 minutes.

At surgical planes, the eye rolls medially, the palpebral reflex is obtunded and tear production ceases. (Apply ophthalmic lubricant to the corneas.) Lateral nystagmus may persist until surgical anaesthesia is achieved. The palpebral reflex persists throughout all planes of anaesthesia and is therefore of little use in monitoring depth of anaesthesia. The anal reflex is progressively obtunded as depth increases and it is lost in very deep anaesthesia.

The heart rate is usually between 25 and 50 beats per minute. Mean arterial pressure should be between 60 and 90 mm Hg. Hypotension should be treated by a) reducing delivered anaesthetic agent, b) delivering intravenous fluids and c) the administration of cardio- and vasoactive drugs. Dobutamine is probably the drug of choice once the anaesthetic agent has been reduced and fluid therapy has been instigated. Dobutamine can be given by IV infusion at rates between 1 and 5 µg/kg/min. Dobutamine is a β-adrenergic agonist and as such, increases the force of cardiac contractility and to a lesser extent, increases heart rate.   The effect of IV dobutamine is very rapid.   Ephedrine has both ά- and β-adrenergic effects. It increases both rate and contractility and also provides some pressor effect. A bolus dose of 0.05 – 0.1 mg/kg will last approximately 20 – 30 minutes.

Many horses, particularly those that are very fit, have very low respiratory rates under anaesthesia. A respiratory rate of 5 – 8 breaths/minute should be OK if the tidal volume is adequate. However, to be sure of the ventilatory status of the equine patient, blood gases should be measured. At very least, end-tidal CO_2 should be measured. Many horses will have very low ventilation rates or will become apnoeic when anaesthetised and will require IPPV.

Recovery   Horses are commonly administered a small dose of an $α_2$ – adrenergic agonist (eg xylazine 0.1 – 0.2 mg/kg) and/or acepromazine prior to recovery to ensure that the horse is calm and does not attempt to stand before it is able to do so with relative safety. It may be necessary to prevent the horse from rising by kneeling on its neck and/or pulling its head dorsally.   (A horse must lift its head and roll onto its sternum before it can rise.) If the horse has nystagmus, it is unlikely to be able to stand steadily. Sometimes, it is necessary to support the horse with both a halter rope and a tail rope as it stands.

Vagolytics   Vagolytic drugs such as atropine and glycopyrrolate are not routinely administered in equine anaesthesia. Administration of these drugs has been associated with postoperative ileus, tachycardia and increased myocardial oxygen demand. They are indicated if the heart rate is very low, particularly if blood pressure is not measured. (A low heart rate is OK if the blood pressure is adequate.) As a 'rule of thumb', it may be advisable to administer atropine (eg 0.01 - 0.02 mg/kg) or glycopyrrolate (0.005 – 0.01 mg/kg) if the heart rate falls below 25 beats per minute, depending on the rate of fall and the fitness of the horse.

Suggestions of drugs and dose rates are included in Appendices 9, 10, 11 & 12.

STANDING SEDATION

There are many indications for standing sedation in horses eg dentistry, wound management, bandage changes in minimally handled horses.
See Appendix 11, Page 130 for suggested drug combinations and dose rates.

PIGS
Restraint
Large pigs can be *both difficult and dangerous to handle*. A rope snare around the upper jaw works well, but is quite traumatic. It is often possible to avoid using a snare by giving adequate IM sedation. In large pigs, the use of *sedation* is important for the safety of the handler.

Intramuscular injections
Intramuscular injections are commonly given in the neck muscle. If it is safe to get in the pen with the pig, stand on one side of the pig with your thigh next to the neck. Quickly stab and inject into the opposite side of the neck. The pig will pull away from the needle and be braced by your thigh. Alternatively, use a long extension set between the syringe and the needle. This allows the pig some movement without dislodging the needle. Allow for the volume of the extension set when drawing up the drug. Otherwise, inject rapidly over the fence.

Intravenous injection
IV injections are usually given into the *veins on the external aspect of the pinna*. The veins can be raised by holding the base of the earflap, or by placing a rubber band around the ear base if an assistant is not available. If the veins are not obvious, they can be made so by gently slapping or rubbing the pinna. Catheter placement is advisable if continued access to a vein is required. Take care to clean and dry the skin before introducing the catheter, as otherwise adhesive tape/glue for holding the catheter in place may not stick. A roll of bandage on the inside of the pinna makes securing the catheter easier. A technique for catheterisation of the jugular vein is described in Hall and Clarke, 1991.

Fasting
Pigs should be fasted for 6 – 8 hours before administration of general anaesthesia. (Less time for very young pigs) Water should be withheld for 2 hours. Whilst vomiting at induction is rare in pigs, a full stomach will press on the diaphragm and reduce inspiratory excursions.

Premedication
Azaperone is a butyrophenone and therefore a dopamine antagonist. *It is marketed as Stresnil® with 40 mg/ml azaperone. Azaperone* is *inexpensive, relatively safe* and *effective* in pigs, and hence very popular. It is best administered by *deep IM* injection at 1 – 8 mg/kg (Hall *et al*, 2001) 1 - 2mg/kg is usually sufficient. It is advisable to stick to the lower end of the dose range in large boars (eg about 1 mg/kg), as there is a risk of *penile protrusion* and subsequent damage.
Side effects include vasodilatation and subsequent *fall in blood pressure*. Hypothermia is a risk in cold climates as cutaneous vessels are dilated.
Acepromazine is an *effective tranquilliser* if given by IM injection at a dose rate of 0.03 – 0.1mg/kg.
A combination of *azaperone (1 mg/kg) and ketamine (3 mg/kg)* provides moderate sedation in mature sows.
Xylazine can be used.
Atropine can be administered at 0.04mg/kg.

Induction
Induction is usually by IV injection into an ear vein. Thiopentone (5 – 10 mg/kg) can be used. Recent studies at UQ (H.Keates, Vet Record, Nov 15, 2003) have demonstrated the suitability of alfaxalone as an induction agent in pigs. Dose rates of 0.9 mg/kg in gilts (average weight 115 kg) and 0.7 mg.kg in mature sows (average weight 220 kg) following azaperone premedication resulted in satisfactory intubation conditions.

Intubation

Pigs are relatively *difficult to intubate*.

Visualisation of the larynx is difficult, and the *opening to larynx is relatively small*. The trachea dips ventrally and then turns sharply dorsally. Pigs can go into *laryngospasm* if the larynx is stimulated. Therefore, they need to be *fairly deeply anaesthetised or paralysed* for intubation or have the laryngeal opening sprayed with *lignocaine*. If the animal is to be paralysed, *oxygen* should be administered for a few minutes prior to administration of muscle relaxant. *Suxamethonium* (1 - 2mg/kg) preceded by *atropine*, will provide muscle relaxation for a couple of minutes. Until spontaneous respiration resumes, *positive pressure ventilation* must be provided. ***Do not paralyse any animal unless you are confident that you can intubate the trachea and you are able to give IPPV. (I don't paralyse pigs for intubation.)***

Endotracheal tube sizes required are *small* with respect to body weight. (25kg – 6mm, 50kg – 9mm, large boar – 14 – 16 mm) Tubes need to be fairly *long (try 50 cm in adult pigs)*.

A *stylette* down the centre of the endotracheal tube may help, but take care that the end of the tube covers the stylette. Rough attempts can easily result in *haemorrhage*, which further obscures the view and can block the narrow airways.

Straight blade human adult laryngoscopes are suitable for small pigs, but *large animal Soper or Rowson blades* (at least 30 cm) are best for adult pigs. A Drinkwater gag placed between the molars and the use of a long stylette to disengage the epiglottis from the soft palate before placing a laryngoscope blade improve the likelihood of success. The lower jaw is elevated with the laryngoscope blade. I prefer to intubate smaller pigs in sternal recumbency. If you have slender hands, manual intubation is possible in large pigs.

Delivery of oxygen and anaesthetic by mask.

Pig snouts could have been designed with anaesthetic masks in mind. The round end of the snout fits snugly in a *conical mask* without reaching the end, which could cause obstruction. A mask can be made from the top of a 2-3 litre soft drink bottle. Pigs have *long soft palates* and *rarely regurgitate or vomit* during anaesthesia. Thus, aspiration is unlikely. Salivation is not usually troublesome. Large pigs are best intubated as they have long soft palates and are prone to obstruction.

Porcine Malignant Hyperthermia

Some strains of Landrace, Large White, Poland China, and Pietrain pigs suffer from a biochemical myopathy of genetic origin. Administration of some drugs (especially halothane and suxamethonium) to susceptible pigs results in generalised muscle rigidity, a severe and sustained rise in body temperature, hyperkalaemia and acidosis. Notably, pigs suffering from Malignant Hyperthermia have very high ET CO_2 and $PaCO_2$ in spite of hyperventilation. This is due to very high CO_2 production. Such cases are rare and should the anaesthetist encounter one, cease drug administration, *cool the patient* and administer *bicarbonate*. Early signs are *spreading apart of the digits*, *blotchy redness* of the skin and *body temperature increase*. Administration of Dantrolene sodium (a skeletal muscle relaxant) is the most effective treatment if given early, but it is very expensive.

Analgesia

Adequate pain relief should follow all surgical procedures. *Narcotic analgesics* can be given in doses similar to those used in dogs. The following dose rates may be useful: - *morphine* at 0.2 – 0.4 mg/kg): *pethidine* 0.5 - 2 mg/kg: buprenorphine 0.01 – 0.02 mg/kg: butorphanol 0.1 – 0.5 mg/kg. IV, IM, SC

Suggested regimens for pigs: -
Regimen 1
Premedication Azaperone (Stresnil®)   40mg/ml 1 - 2 mg/kg (maximum dose of 400 mg)
Inject IM, wait 15 – 20 minutes
Induction   Thiopentone   1g/90kg or alfaxalone 0.7 - 1 mg/kg

Maintenance   Isoflurane in oxygen delivered by mask

In big pigs, give 1g thiopentone or 200mg alfaxalone and put straight on isoflurane. They will be too light to intubate, but will be fine on a mask. If you intend to intubate, give more induction agent slowly to effect.

Regimen 2   **very useful for smaller pigs(expensive!)**

Xylazine   0.5 – 1 mg/kg IM   ) Can be mixed in one syringe
Ketamine   5 - 15 mg/kg   IM   )

Unsteady in 3 – 4 min, down in 5 – 7 min, relaxed, asleep in 14 – 15 minutes. Some signs of excitement early in the process eg bulging eyes, stiffness, shaking. Lasts 30 – 45 minutes. If longer required, halothane or isoflurane via mask

Pot bellied pigs require low doses

Regimen 3   suitable for adult sows

Premedication   Azaperone   1 – 2 mg/kg IM (lower dose for big sows)
                         + Ketamine 3 mg/kg

Once pig is sedated, place and secure catheter in ear vein for induction.

Induction   Alfaxalone   0.7 – 1.0 mg/kg IV (lower dose for big sows) Can give more if necessary.

Maintenance   Intubate or mask and maintain on halothane or isoflurane

Eg full term adult Large White or Landrace sow (> 300 kg)

- Inject premed IM in same syringe.
  - Azaperone (Stresnil)   10 ml (400 mg)
  - Ketamine   10 ml (1000 mg)
- Once sow has relaxed, 'sneak up' and insert 20 G catheter in ear vein. Secure with Super Glue and tape.
- Induce with alfaxalone 20 ml (200 mg) bolus dose. More if required Give oxygen and isoflurane by mask as soon as possible.
- Induce with alfaxalone 20 ml (200 mg) bolus dose. More if required.
- Intubate
- Connect to breathing system.

CATTLE

Cattle are *not good candidates for general anaesthesia*. Therefore, many procedures are carried out under *sedation and local anaesthesia*.

Many of the problems associated with anaesthesia of cattle are a related to the fact that **adult cattle have a rumen and therefore, they voluntarily eructate and regurgitate regularly.** Once the animal is *unconscious and unable to elevate its head*, the *oesophageal opening is submerged*, and *eructation of gas is not possible*. This is a problem in light as well as deep anaesthesia. If the animal is light, active regurgitation may occur. In deep anaesthesia, the oesophageal sphincter and oesophageal muscle will be relaxed, making passive regurgitation more likely. Thus, there are *two potential problems related to the rumen.*

1. Rumenal liquid over the oesophageal sphincter means *regurgitation of oesophageal contents* is likely. Measures must be taken to reduce the *risk of aspiration* of regurgitated rumenal contents.
2. Loss of eructation means *accumulation of rumenal gas, or bloat*. As the rumen expands with gas, the *diaphragm is pushed forwards, reducing inspiratory volumes*. Also, *increased abdominal pressure* due to bloat *increases* the *risk of regurgitation.*

Contributing to these problems is that **cattle salivate copiously,** even when anaesthetised. Saliva can pool in the pharynx, drain out of the animal's mouth or pass down the trachea.

To minimise the risk of these problems :-
1. *Withhold water for 12 – 24 hours before anaesthesia.* (Remember that cattle have rumenal fluid to draw on to maintain hydration.)
2. *Withhold food for 24 – 48 hours before anaesthesia.*
3. ****Intubate the trachea with a cuffed endotracheal tube. Intubate with the cow in sternal recumbency and the head elevated.***
4. *Limit the length of anaesthesia to a maximum of 2 hours.*
5. *Position the animals head so that the occiput is higher than the general level of the body to allow fluid to drain away from the tracheal opening.*
6. *Be prepared to place a rumenal trocar.*

The **large size** of adult cattle has similar implications as in horses. Once anaesthetised, cattle are difficult to manoeuvre and position. Frequently, anaesthesia is induced where the surgery is to take place.   The use of soft positioning pads should be used to reduce pressure due to body mass on areas like the point of the shoulder. Ischaemic nerve and muscle damage can occur as in horses.

All large herbivores have voluminous gastrointestinal tracts to accommodate *fermentation chambers*. As the abdominal contents are so large, these animals rarely voluntarily lie in lateral recumbency for long. Once the animal is in lateral recumbency, the abdominal contents press on the diaphragm limiting **respiratory movements**, the conscious animal becomes hypoxic and sits up. Thus during anaesthesia, *intermittent positive pressure ventilation* may be necessary if arterial hypoxaemia is to be avoided.

Young calves are essentially monogastric, and therefore have fewer problems under general anaesthesia.

Premedication *Xylazine is very popular for sedation in cattle.* **Cattle are extremely sensitive to xylazine** compared with small animals and horses. *Injection of 0.05 – 0.15 mg/kg IV or 0.1 – 0.2 mg/kg IM produces* mild sedation to profound sedation with recumbency, depending on route and dose chosen. The effect of an *IV dose peaks at about 5 min*, IM at 10 – 20 min. Sedation lasts 30 – 35 minutes*, but the effects of the drug take many hours to wear off totally. The use of xylazine is* contraindicated in the last trimester of pregnancies *(reports of premature births).* Rumenal activity is depressed*. Whilst sedated, the animal* retains the ability to eructate, cough and swallow. Recoveries should be supervised *as animals will occasionally become laterally recumbent, and develop rumenal tympany.*

Other side effects include an *increased plasma glucose concentration* and *suppression of ADH production*, together resulting in *diuresis.* The patient will *lose the ability to control its core temperature*, which will tend to follow ambient temperature.

As with small animals, xylazine causes *bradycardia* in cattle.

There is *debate over whether xylazine produces analgesia* in cattle. Therefore, *surgery on conscious animals should be only after local anaesthesia blocks*.

Detomidine Cattle require *doses comparable to those in horses*, making detomidine an *expensive* alternative. Side effects are similar to those seen with xylazine, except that low doses appear to have *little effect on uterine activity*, and detomidine *causes hypertension*.

Medetomidine Medetomidine appears to cause deep sedation without recumbency, but it is prohibitively expensive.

Induction *Thiopentone* can be used to induce cattle for *short procedures* or for *intubation prior to maintenance with inhalation agents.* A dose rate of 1 gram per 100 kg will be adequate for an animal that has not been premedicated**. *If the induction is preceded by xylazine sedation, this should be reduced to 1 gram per 200 kg*.** To achieve rapid injection that is necessary to maximise the effect of these relatively low doses of thiopentone, the thiopentone can be made up as a 5 -10 % solution. At these concentrations, thiopentone is highly irritant, and any drug accidentally injected outside the vein should be diluted by a subcutaneous infusion of 0.5 – 1.0 litres of normal saline. Avoid extravascular injection by inserting and securing **a large bore catheter for injection eg 12G, 4 inch.**

A very brief period of apnoea may follow injection of thiopentone, but IPPV is rarely necessary.

Ketamine Ketamine, administered after sedation with xylazine, can give satisfactory anaesthesia in cattle. Eg

- Xylazine 0.2 – 0.35 mg/kg IM or 0.1 – 0.15 mg/kg IV Wait until the animal is recumbent, 10 – 20 minutes after IM injection, 5 – 10 minutes after IV.
- Ketamine 2.2mg/kg IV

Intubation

Intubation is carried out by *palpation* with the anaesthetised patient in **sternal** *recumbency*. The *head should be elevated*, the *mouth held open* and *the tongue gently pulled forwards*. Use one hand to *cover and protect the cuff* of the endotracheal tube and the other to push the tube into the mouth. With the hand that is protecting the cuff, *feel for the epiglottis* and *pull the tip forwards and down*. Just caudal to the epiglottis is the glottal opening. *Spread the arytenoid cartilages* with the fingers and *direct the tube caudal and* **ventral** *into the trachea*. Once the tube is in the trachea, it should be easy to advance. (If the tube is placed in the oesophagus, there will be more resistance to advancing it.) Once the tube is in the trachea, airflow will be detected in the tube as the patient exhales. Once the tube is in place, the *cuff must be inflated to seal the trachea* and protect against inhalation of saliva and/or regurgitated rumenal contents. ***Accidentally passing the ET tube into the oesophagus stimulates regurgitation.***

Maintenance Halothane and isoflurane are both satisfactory anaesthetic agents in cattle. If prolonged anaesthesia is anticipated or if severe bloat is detected, the rumen should be pierced with a trocar and cannula. The trocar should be removed and the cannula left in place until the animal is sitting and able to eructate again. 'Triple drip' (guiafenesin, ketamine + and alpha$_2$ adrenergic agonist) can be used but the patient must be intubated with a cuffed ET tube for airway protection.

Recovery Cattle are, in the main, calmer anaesthetic patients than horses. Unlike horses, cattle normally spend a lot of time in sternal recumbency. Mostly, cattle recovering from anaesthesia will quietly regain a sitting position, sometimes needing support. Eg bale of hay behind the shoulder. They should be left undisturbed but supervised until they are able to stand steadily. Cattle will tolerate the endotracheal tube in place even once they are fully awake. ***Take care that the patient does not kink the tube or 'vacuum' up dust and straw if the ET tube is still in place.***

SHEEP
The risk factors wrt regurgitation, aspiration and bloat are the same for sheep as for cattle. Periods of food and water withdrawal should be 24 h and 12 respectively.

Premedication	Midazolam or diazepam	0.3 mg/kg
Induction	Thiopentone, alfaxalone or propofol to effect	
Intubate	Sternal recumbency, cuff the ET tube. Try 9 mm ET tube.	
Maintenance	isoflurane, halothane	

LOCAL ANAESTHESIA
= Local Analgesia = Regional Anaesthesia

"Local anaesthesia is defined as a deliberately induced and temporary loss of sensation in a defined body area without impairment of consciousness. This anaesthesia is produced by the introduction of an agent, the so-called local anaesthetic, which acts either on sensory nerve endings or nerve trunks and paralyses nerve function." (Westhaus and Fritsch, 1964)

Types of local anaesthesia (LA)
1. *Terminal anaesthesia* = LA drug acts directly on sensory nerve endings or end plates.
    - *Surface anaesthesia* = LA administered eg onto conjunctiva, into joint cavity
    - *Infiltration anaesthesia* = LA infiltrated into operative field
    - *Venous anaesthesia* = LA administered into the vein of a limb after application of a tourniquet
    - *Refrigeration anaesthesia*= low temperatures used to eliminate nerve function
2. *Regional (nerve trunk) anaesthesia* (nerve blocks) = LA injected endoneural (under the nerve sheath) or perineural to block the nerve trunk.
3. *Spinal anaesthesia* = a form of nerve trunk anaesthesia, as nerves are blocked within or at their exit from the spinal canal. LA injected into the space outside the dura mater (*epidural* space). Subdural injections (into the CSF) are less frequently used because of the risk of cranial spread of drug with possible respiratory paralysis.

The ease with which a nerve can be blocked by local anaesthetic depends on the *presence/absence* of, and the *thickness* of the *myelin sheath*. The *myelin sheath is relatively impervious to local anaesthetics* and the *drug can act only at the nodes of Ranvier* of a myelinated nerve. The large Group I nerves (including somatic motor nerves A-α) have thick myelin sheaths and are therefore last to be blocked. The thin Group III nerves are either unmyelinated or have thin myelin covering and are blocked first. Pain afferents (A-δ and C) are included in this group. For a large myelinated nerve to be blocked requires a higher concentration of drug or exposure to a greater length of nerve (ie a greater number of nodes of Ranvier). Thus, *it is possible to preferentially block the pain fibres and spare the motor nerves.*

History
Cocaine was first introduced clinically in 1884. Its toxicity and addictive properties lead to its use being discontinued except in surgery of the nasal cavity when its ability to produce intense vasoconstriction is useful.
Procaine was the next drug on the scene, in 1905. It is very slow in onset.
Research and development is directed towards producing a local anaesthetic that is longer acting and less toxic than those already available.

Current use

Recognition of the value of intra- and post-operative analgesia has led to an upsurge in interest in local anaesthesia. Maintenance of very light general anaesthesia, with surgery sites rendered analgesic with epidural anaesthesia, local anaesthetic nerve blocks or infiltration can reduce anaesthesia-related risk and improve recovery, especially in debilitated animals. The use of longer acting local anaesthetics means that the blocks will extend well into the post-operative phase, reducing the need for systemic analgesics.

Toxicity
Local anaesthetics affect all excitable tissues in the body, so toxicity can occur when sufficient amounts of the drug are *absorbed into the circulation*. *Inadvertent intravenous injection* is the most usual cause of toxicity but care must be taken with *injections of large*

volumes in very vascular tissues. The rate of absorption may exceed the rate at which the drug is metabolised.

CNS
- Firstly, *sedation*, then
- *excitement* (blocking of inhibitory pathways)
- overall *CNS depression* (blocking of all pathways)

CVS At concentrations required to treat dysrhythmias, there is little effect on cardiac muscle activity. At toxic doses, there is decreased electrical activity resulting is *sinus bradycardia* which can proceed to arrest.

Respiratory system There may be some *central depressant effects* even at subtoxic doses.

Local toxic effects. Large doses of some drugs can result in *local tissue damage*. Cytotoxicity is correlated with potency.

Accurate placement of minimal doses of local anaesthetics will minimise the risk of toxic side-effects.

Some clinically useful local anaesthetic drugs.

Lignocaine   Lignocaine is widely used in veterinary practice. It is available in injectable form, usually as a 2% solution, with or without adrenaline.   (Adrenaline causes vasoconstriction and hence delays absorption, thereby keeping the drug in contact with the nerves longer) Lignocaine rapidly diffuses through the tissues and easily penetrates the nerve sheath. Onset is rapid, duration 90 – 200 min.

It is useful when applied to mucous membranes. It is available in gel form, and as a spray.

Uses

Local anaesthesia.   *Total dose of lignocaine should not exceed 4.5 mg/kg.*
- Useful in surface, venous, infiltration anaesthesia, nerve blocks and epidural anaesthesia.
- Treatment of *cardiac dysrhythmias.* Can be used at a dose rate of 1 mg/kg IV to treat tachydysrhythmias. This dose can be repeated several times at intervals if necessary. It can then be delivered by infusion if required.
- *Systemic analgesia*   A continuous infusion of lignocaine can be used to provide pain relief.   (Loading dose of 2 mg/kg followed by infusion of 40 µg/kg/min.)

Bupivacaine (eg Marcain®)   *Total dose of bupivacaine should not exceed 2 mg/kg/4h.*

Bupivacaine is *slower to take effect than lignocaine*, but *lasts longer*. (180 – 600 min) It is available in solution at 0.25 – 0.75 %. Commonly used at 0.5%.   Long duration makes this drug useful for intra-operative and post-operative analgesia.

Bupivacaine is highly cardio-toxic.
Never administer bupivacaine IV.

Mepivicaine (eg Vetacaine®)   Duration 120 – 240 min.   Useful in local blocks for lameness diagnosis in horses.

Proxymetacaine HCl (eg Ophthetic®)   One drop of a 0.5% solution on the cornea results in analgesia in about 15 seconds which lasts for about 15 minutes. It is *not irritant* and *does not cause pupillary dilatation*.

Infiltration techniques (Terminal anaesthesia)
Apart from the initial injection, always try to *inject through desensitised tissue to mimimise pain*. Use the finest needle that is practical.

- inject while advancing the needle through the tissues, always drawing back before injecting.
- work proximal → distal along the line of innervation.

Intracutaneous infiltration   Producing a skin wheal with an intradermal injection of local anaesthetic gives an insensitive area of skin for future injections. Also, the wheal acts as a marker.

Linear infiltration   Infiltrate the line of the surgical incision. This can cause problems by altering the architecture of the tissues making tissue types difficult to identify.

Fan-wise infiltration   Infiltration of tissues around the surgical site to block the sensory nerves entering the operative area.

Pyramidal field   "   "

Ring block   The soft tissues are blocked around the circumference of a limb proximal to the surgery site. This procedure is best performed between two Esmarch's bandages.

Inverted "L" block   Two line blocks are carried out in the shape of an inverted "L", dorsal and cranial to, but not encroaching on the incision site. As the nerves run caudal and ventral, this block should render the surgical site insensitive.

Regional blocks
This type of local anaesthesia involves interruption of conduction in nerve trunks to block their fields of innervation. A good understanding of anatomy is necessary for accurate placement of local anaesthetic in close proximity to the nerve. The advantages of this technique are that the operative field is untouched and only small quantities of local anaesthetic are required.

Indications

Lameness diagnosis (especially in horses)

Surgical procedures carried out on *standing animals*, typically, cattle and horses. Eg paravertebral for standing caesarean section in cattle.

Analgesia (with general anaesthesia) for *surgery* and for *post-operative* analgesia in dogs, cats and horses.

Spinal anaesthesia
Epidural anaesthesia is described as *high or low*, *depending on how far cranial the anaesthetic travels*, rather than where it is placed. Eg in cattle, a small volume injection at C1-C2 space, will give a *low* epidural, the skin over the tail, croup and anus, vulva, perineum and the caudal thigh will lose sensation. The motor control of the hind limbs will be unaffected. A larger volume administered at the same site will travel further cranially and affect the hind limbs. Hence it is called a *high* epidural.

Should the anaesthetic travel too far forward, *respiratory paralysis* can result, necessitating positive pressure ventilation until the anaesthetic wears off.

If the lumbar and thoracic segments are blocked, *sympathetic* outflow may be affected and *hypotension* may result. It is advisable to have all animals having epidurals on *intravenous fluids* prior to injecting in case this problem should occur. Venous access may be difficult to procure after hypotension has occurred. Appropriate treatment would be rapid administration of IV fluids and an adrenoreceptor agonist with α-1 adrenoreceptor activity eg phenylephrine.

Low epidurals are used in dystocia in horses and cows. It is important in these cases that the hind limbs are not affected, as the animal must remain standing.

Subdural anaesthesia is less frequently used in veterinary patients. However, it is not unusual when placing a needle for epidural injection in cats or dogs to reach cerebrospinal fluid. The ensuing injection will then be in the subdural space. Should this occur, it is
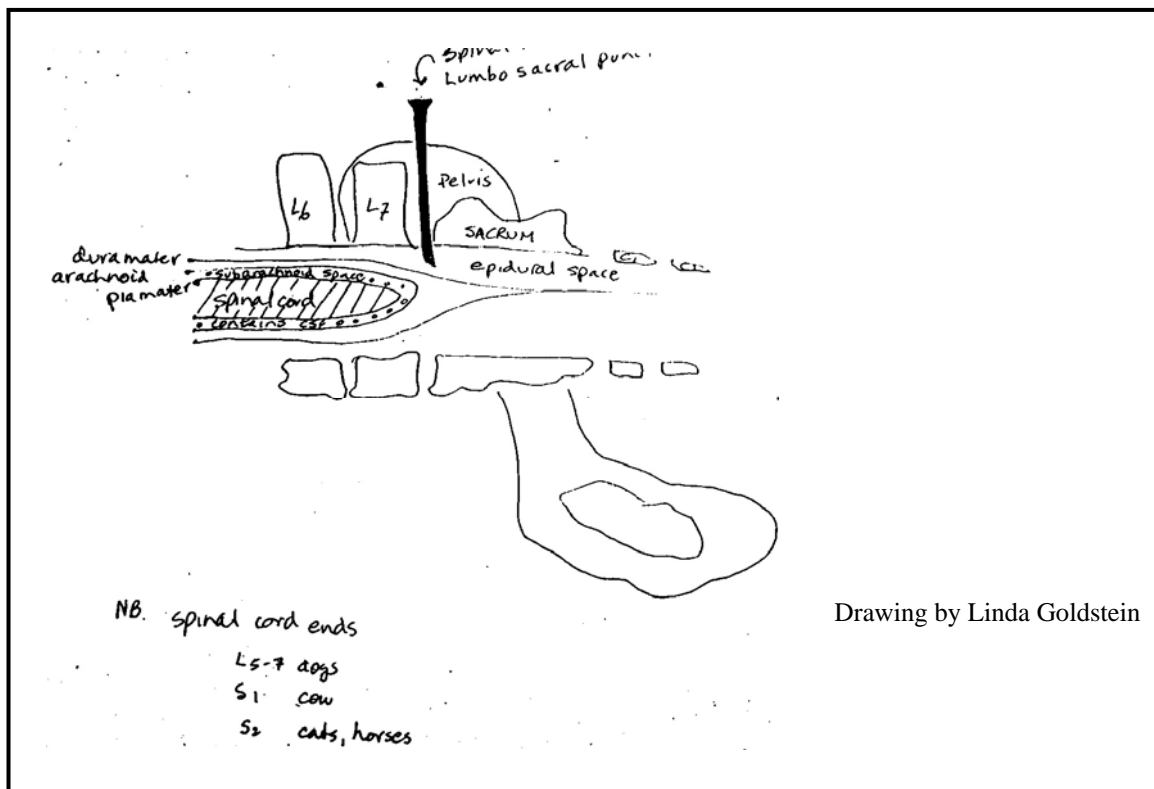
important to a) make up a suitable injectate (eg opioid without local anaesthetic) or b) inject half the prepared injectate.
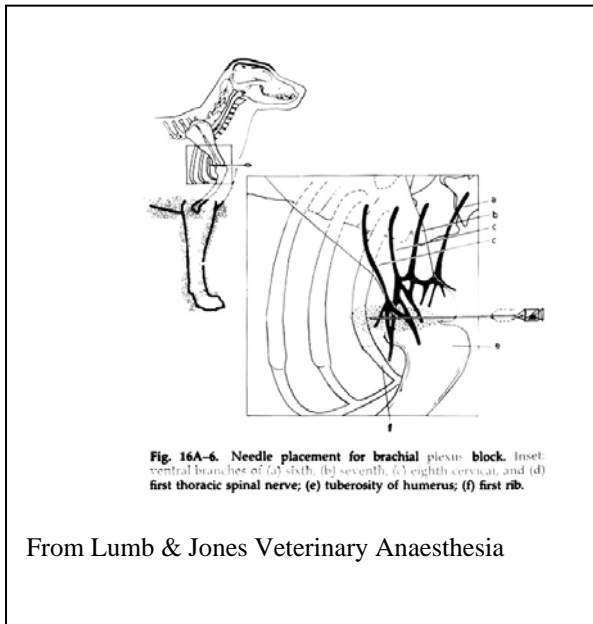
DOG
Epidural. There are two positions commonly used to maximally flex the lower spine and open the dorsal inter-vertebral spaces.

1. Dog in lateral recumbency with legs elevated so that the sagittal section of the dog is horizontal. The dogs hind legs are pulled well forwards

2. Dog in ventral recumbency with hind legs pulled forward to flex the lumbar spine.

Epidural injection must be performed as a sterile procedure. Clip, clean and drape the site, wash hands and wear sterile gloves. Palpate the iliac crests and imagine a line joining them. Approximately on this line, you will palpate the depression between the dorsal spinous processes of the seventh lumbar vertebra and first sacral vertebra. Use a short bevelled spinal needle with a stylette. Insert the spinal needle (with the stylette in place) perpendicular to the skin, into the L-S space. Advance the needle towards the spinal canal. A distinct pop may be felt as the needle penetrates the ligamentum flavum. The needle will need to be at a depth of 0.5 – 1.5 inches, depending on the size and condition of the dog. There is usually no cerebrospinal fluid at this level of the spinal canal, but pausing to see if CSF or blood appears is advisable. If there is no blood or CSF, attach a low resistance syringe and use air to test for resistance to injection. Minimally depress the plunger and release. If the plunger 'bounces' back, then there is resistance to injection and the needle placement is incorrect. The epidural space is under a slight negative pressure, and there should be no resistance to injection of air if needle placement is correct. Another way of testing that the tip of the needle is in the epidural space is to fill the hub of the needle with sterile saline or lignocaine once the needle is through the skin but before advancing towards the spinal canal. As the needle reaches the epidural space, the contents of the hub are sucked into the space. This technique works well in horses and cattle. If CSF is encountered, inject opioid only, or proceed with half of the local anaesthetic/opioid combination intended for epidural injections. If blood appears, you are in the vertebral sinus. Withdraw and start again.

Drawing by Linda Goldstein

Forelimb

Fig. 16A–6. Needle placement for brachial plexus block. Inset: ventral branches of (a) sixth, (b) seventh, (c) eighth cervical, and (d) first thoracic spinal nerve; (e) tuberosity of humerus; (f) first rib.

From Lumb & Jones Veterinary Anaesthesia

Brachial plexus   .   The area blocked is from the *foot to the elbow*. Use a 22G 3 inch needle. Direct the needle from *medial to the shoulder joint* towards the costochondral junctions and parallel to the vertebral column.   The needle will not penetrate the thorax. Inject the anaesthetic as the needle is withdrawn, aspirating before injecting at each site to be sure that intravenous injection does not occur. Inject 10 – 15 ml local anaesthetic. (Take care not to exceed 4.5 mg/kg lignocaine or 2 mg/kg bupivacaine.   Dilute with sterile saline to achieve the require volume.) The block will take effect slowly (20 minutes onset and 2 hours duration with lignocaine).

Nose and upper teeth.
Maxilliary   Blocking the infraorbital and palatine branches of the maxillary nerve produces a *sensory block of the maxilla, palate, upper teeth, nose and upper lip*. Use a 25G, 16 mm (5/8 inch) needle, and 0.25 – 0.7 ml local anaesthetic (0.5 ml in most dogs). The needle is bent to 45°, and inserted with the syringe at approximately 45° to the palate. This results in the needle being at 90° to the palate. The point of insertion is about ⅓ of the distance from the last molar to the midline. The needle is then walked off the edge of the hard palate, angled medially about 30° and inserted to the hub. After aspirating to ensure that the needle has not penetrated a blood vessel, inject the local anaesthetic.

Lower teeth and lip
Mandibuloalveolar Blocking this *nerve anaesthetises all the lower teeth, skin, and mucosa of the chin and lower lip*. The nerve is anaesthetised at the point of entry into the mandibular canal at the mandibular foramen. Use a 25G, 16 mm (5/8 inch), 0.25 – 0.7 ml anaesthetic. To identify the site of injection, draw a line from the angle of the jaw to the last tooth on the lower jaw. Draw a second line from the midpoint of the dip in the mandible, towards the lateral canthus of the eye. Where these two lines intersect is level with the mandibular foramen where you inject the local anaesthetic. The needle will pass medial to the mandible.

Maxillary Block

Mandibuloalveolar Block
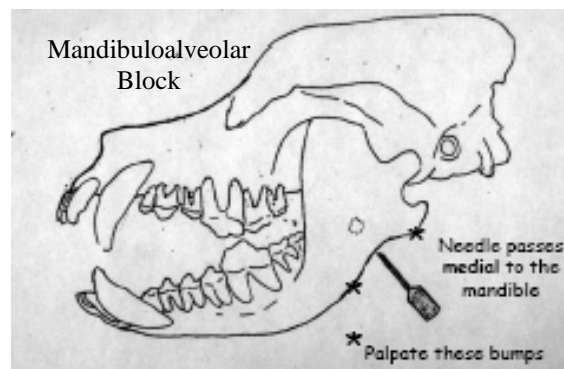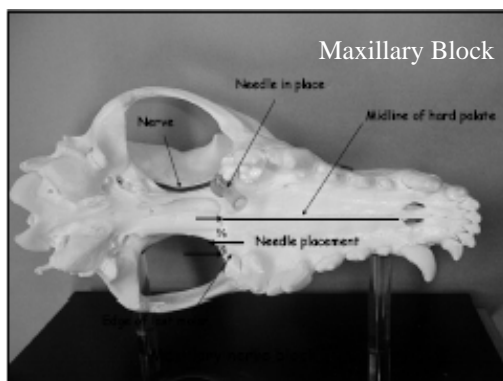
Eye

Ophthalmic division of the trigeminal nerve This block anaesthetises *the eye, orbit, conjunctiva, eyelids and forehead skin*. Use a fine, 25 mm (1") needle, 2 ml local anaesthetic. Insert the needle ventral to the border of the zygomatic arch, at the lateral canthus of the eye. The point of the needle should be 0.5 cm cranial to the anterior portion of the vertical ramus of the mandible. The needle travels medial to the mandible mediodorsal and caudal to reach the orbital fossa. Aspirate before injection.

Thorax

Intercostal nerve blocks Bupivacaine is the drug of choice as prolonged *pain relief is desirable after thoracotomy*. Because nerve "territories" overlap, block nerves cranial and caudal to incision site. For an incision between ribs 4 & 5, block the nerves between ribs 3 and 6. Use a fine 25 mm (1") needle. The volume at each site depends on the size of the dog. (small – 0.25 ml/site, large – 1ml/site.) Do not exceed 2mg/kg bupivacaine. The needle should be inserted at 90° to the skin surface, caudal to the rib, near the intervertebral foramen. It is very important to aspirate before injecting as the artery and vein are in close proximity to the nerve.

Needle placement for an Intercostal nerve block.

Digits This block is valuable during and after *surgery on the digits*. The digital nerves can be blocked medial and lateral to the first phalanx. Extend the toe to be anaesthetised with the left hand. Insert a fine needle subcutaneously on each side of the digit obliquely and proximally and inject 2 – 3 ml of local anaesthetic either side. (Less for a small dog)

Fracture Painful fractures can be made more tolerable in the conscious dog with an injection of *local anaesthetic into the fracture site*. Care must be taken with *sterility*.

CAT

As for dog, but smaller volumes.

CATTLE

Low or caudal epidural   *Blocks anus, perineum, vulva and vagina* and is very useful in *obstetric cases.* Insert needle into $C_1 – C_2$ space. Identify the space by lifting and lowering the tail. This joint is very mobile and is situated just anterior to the anal folds. The needle should be exactly midline and perpendicular to the skin. With the tail down, insert the needle through the interarcuate ligament and on to the floor of the canal. (2 – 4 cm depth) Test for position by a) having local anaesthetic in the hub of the needle to be sucked in when it hits the negative pressure within the canal or b) injecting air to establish lack of resistance. Use a 1½" needle, and inject 4 – 7 ml depending on size of the animal. Take care with the volume injected. Too large a volume will cause the cow to go down or to be very unsteady on the hind legs. Try 1 ml 2% lignocaine per 100 kg (maximal anaesthesia in 10 – 20 min, duration 30 – 150 min.). For longer duration, (up to 3 h) xylazine can be administered at 0.05 mg/kg diluted with sterile water to 5 ml. The side effect expected with IV or IM xylazine (sedation, CV disturbances etc) are routinely seen after epidural xylazine.

Paravertebral   *Blocks the skin, subcutaneous tissues, muscle layer and peritoneum.* This block is often used for *standing laparotomy* in cattle. The *viscera will not be desensitised*, so care must be taken not to stretch or tug viscera. The area to be blocked is supplied by the nerves from T13, L1, L2 and L3, both dorsal and ventral branches. These nerves run caudally and ventrally and can be blocked above and below the cranial edges of the transverse processes of vertebrae L1, L2, L3 and L4. Clip and surgically prepare the skin on the side of the cow to be blocked. Identify the transverse processes of L2, L3 and L4. L1 is short and can be palpated only in an emaciated cow. If it cannot be palpated, estimate its position considering that the processes are equidistant from each other. Imagine a line from the most prominent part of the transverse process perpendicular to the line made by the dorsal spinous processes. Come out along that line 5cm (one-matchbox length) from the line made be the dorsal spinous processes. This is the site of injection. Inject 2ml intradermally at the site of injection. This will serve as a marker as well as desensitising the skin for a stab with a small scalpel blade. Insert an 18 G, 4", short bevelled needle through the skin incision perpendicular to the surface of the skin. The needle should contact the transverse process. It can then be "walked" forwards off the anterior edge of the process. The needle can then proceed deeper, through the intertransverse ligament. Once beneath the ligament, 15 ml of local anaesthetic is deposited. The needle is then withdrawn to just above the process again, and a further 5 ml is deposited. Once all four sites are blocked, effects should be apparent within minutes if the placement was accurate. The area should be desensitized (test with a needle), the skin of the affected area should be warm (vasodilatation), and the cows spine should be bowed out towards the affected side (muscle relaxation).

Eye and eyelids
The **Peterson technique** results *in anaesthesia of the eye and the orbit and immobilisation of the eye. Paralysis (but not analgesia) of the eyelids* is achieved by blocking the *auriculopalpebral* branch of the facial nerve. *The Peterson technique* involves blocking the oculomotor, trochlear and abducens nerves and the three branches of the trigeminal nerve, ophthalmic, maxillary and mandibular. The nerves are blocked as they emerge from the foramen orbitorotundum, deep within the orbit. Use an 18G, 12.5 cm (5") needle, 7 – 15 ml local anaesthetic.
Surgically prepare the skin caudal and ventral to the eye.   Palpate the notch formed by the zygomatic and temporal process of the mandibular bone. (This is where the supraorbital process of the frontal bone meets the zygomatic arch.)   Inject a small subcutaneous bleb of anaesthetic in the notch, as far anterior and ventral as possible. Make a stab incision with a small scalpel blade. With the cows head fully extended and the frontal and nasal bones horizontal, direct the needle in a horizontal and slightly caudal direction. The needle will hit bone (the coronoid process of the mandible) when it must be adjusted cranially to pass

medial to the process. Pass the needle caudally and ventrally until it hits bone at a depth of 7.5 – 12 cm (3 - 4½"). Inject 15 ml at this site.
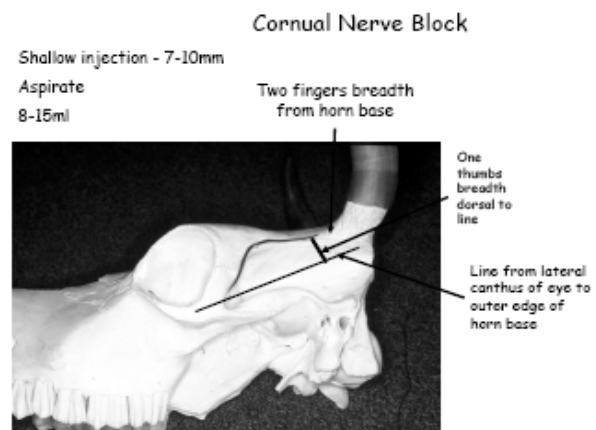
It common for the upper eyelid to still be sensitive after this block. Should this be a problem, the upper lid can be desensitised with an infiltration technique.

A *retrobulbar* block can be used for eye surgery. This block is simple to perform, but is somewhat risky. However, if the eye is to be enucleated, then the risk of damage to the eye is not so serious. Use 18G, 4 cm (1½"). The needle is placed at 2, 4, 8 and 10 o'clock, with the needle running close to the bony boundaries of the orbit. Inject 10 – 20 mls per site, some deep and the rest as the needle is withdrawn. This block, combined with infiltration of the upper and lower lids will give satisfactory anaesthesia of the eye for enucleation.

The *auriculopalpebral branch of the facial nerve* is blocked as it runs from the base of the ear along the facial crest of the frontal bone. The injection is made at the dorsal border of caudal end of the zygomatic arch, cranial to the base of the ear. 10 – 15 ml of solution is injected. This block *does not give analgesia to the eye or the eyelids*, but immobilises the eyelids and can be used to facilitate eye examination, or, *in combination with an eye block*, enucleation.

Cornual for dehorning

The site for injection to block the *cornual* nerve is located two fingers breadth anterior to the horn and a thumbs breadth dorsal to the line from the lateral canthus of the eye to the outer edge of the horn base. The nerve is 7 – 10 mm below the skin. Too deep an injection will be beneath the aponeurosis of the temporal muscle and may be ineffective. Aspirate before injection as the artery and vein are in close proximity to the nerve. Inject 8 – 15 ml, less in calves. A drooping upper eyelid is a sign that the block has worked.

Cornual Nerve Block

Shallow injection - 7-10mm
Aspirate
8-15ml

Two fingers breadth
from horn base

One thumbs breadth dorsal to line

Line from lateral canthus of eye to outer edge of horn base

Teat and udder

The most common reason for anaesthetising the udder or teats is for repair of wounds. Restraint of the cow will be necessary – probably the most effective being a tail hitch with sedation. The simplest and a very useful technique is to infiltrate the wound edges via the exposed surface of the wound itself. Injecting via the wound removes the need to go through the skin, which is very sensitive. The wound surface is much less sensitive. A ring block of the teat is effective, but less tolerable for the cow. Pinching the skin up before injection via a fine needle decreases the discomfort.

IV regional block for distal limbs surgery (Bier's Block)

This is a relatively simple technique that requires no detailed anatomical knowledge. Local anaesthetic is injected into any large superficial vein below an arterial tourniquet.   The distal limb will become analgesic and will remain so until the tourniquet is released. Adequate analgesia is achieved for amputation of the digits etc. The cow is sedated and the limb restrained. A tourniquet is applied above the carpus or the hock. The hind limb tourniquet will be more effective if a roll of bandage is placed in the depression between the tibia and the Achilles's tendon. Thirty ml of local anaesthetic is injected into a vein in adult

cattle. Complete analgesia occurs within 10 minutes, and sensation returns several minutes after release of the tourniquet. The tourniquet can be left in place for up to 1¼ hours. The success rate is higher if the limb is exsanguinated using an Esmarch bandage prior to injection of local anaesthetic.

HORSE

Lameness diagnosis using nerve blocks will be covered elsewhere in the course.

Epidural   Epidural anaesthesia can be used in the horse to block the tail, anus, perineum, vagina, vulva and rectum. The block is performed at the first intercoccygeal space. An 18G, 5 – 7.5 cm (2 – 3") spinal needle is used. It is imperative that the horse's hind limb function is not affected as horses will often panic if they are unable to stand properly. Six – 10ml will be appropriate in most cases. The horse must be restrained or sedated. The injection site can be identified by elevating the tail. The depression over the $C_1 - C_2$ joint can be palpated about 2.5 cm anterior to the start of the tail hairs. A skin bleb of local anaesthetic will desensitise the injection site. With the tail down, the needle can be inserted perpendicular to the skin or at an angle of 30º, angled cranially. Loss of resistance to injection is the most reliable way of ensuring accurate placement. The main indications for epidural anaesthesia in the horse are obstetrical manipulations and surgery of the perineum, vulva etc. The *block may take 20 – 30 minutes to take effect.*
Drugs used include local anaesthetics (eg 2% lignocaine 6 – 8 ml in a 450kg mare), alpha$_2$-adrenergic agonists (eg xylazine 0.17 – 0.25 mg/kg in saline to 6 – 10 ml) and opiates (eg butorphanol 0.04 mg/kg, administered with lignocaine)
A report by Grubb *et al*, 1992 described the use of a mixture of lignocaine (0.22 mg/kg 2% solution) and xylazine (0.17 mg/kg 2% solution) which gave satisfactory caudal epidural anaesthesia with a rapid onset (5.3 min) and long duration (330 min).

Nerve blocks on the horses head.
It is necessary to *sedate* and *twitch* horses before attempting these blocks. For correct needle placement of an infraorbital block, the needle may pass through the infraorbital nerve which will make the most stoical of horses jump. A combination of xylazine and butorphanol IV, followed by twitching 5 minutes later will improve patient tolerance and operator safety. **Do not use lignocaine with adrenaline.**

Infraorbital nerve   This block will *desensitise all the upper teeth except the last two, as well as the upper lip and nostril, roof of the nasal cavity and all related skin up to the infraorbital foramen*. Palpate the most rostral prominence of the facial crest and the caudal border of the nasomaxillary notch. Two finger-widths back from the midpoint of the line joining these two landmarks is the opening to the infraorbital foramen, under the levator labialis muscle. The ridge on top of the opening can be palpated. First anaesthetise the skin and soft tissues overlying the opening to the foramen with 2ml local. Insert an 18G 9 cm (3.5inch) needle through the skin, 1 cm in front of the palpable ridge and guide it into the foramen. The canal is approximately parallel to the surface of the horse's skull. When the needle is 5 cm (2") up the canal, cover the foramen with a finger to prevent anaesthetic escaping and inject 5ml lignocaine. Withdraw the needle and keep the foramen blocked for 5 minutes.

Mandibular nerve   Anaesthetising the mandibular nerve at the mandibular foramen *desensitises the whole of the lower jaw*. Use an 18G needle, 16 cm (6.5") long. Palpate the lowest point of the jaw, and draw a line to the lateral canthus of the eye. Extend the line of the occlusal surface of the cheek teeth to intersect with the first line. This point marks the location of the mandibular foramen on the medial aspect of the mandible. Once the point of insertion of the needle is identified **medial** to the lower border of the mandible, desensitise the skin with 2 ml local. Insert the 16 cm (6.5") needle to travel vertically in the cranio-

caudal plane, medial to the mandible, angled laterally to follow the medial surface of the mandible to the pre-measured depth. The mandibular foramen will be at approximately 12.5 cm (5") depth. Inject 20 ml local anaesthetic.
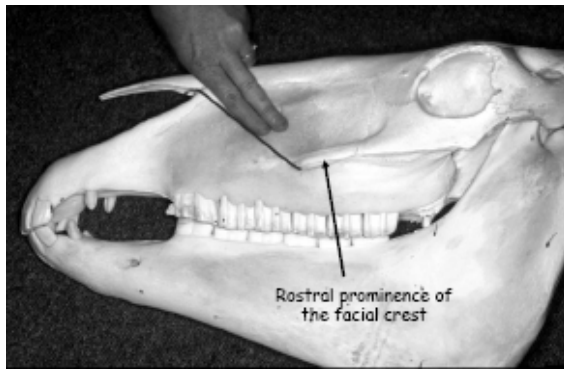
Landmarks for Infraorbital Nerve Block
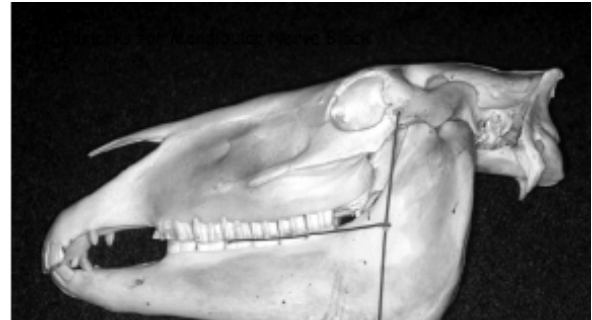
Landmarks for Mandibular Nerve Block

Supraorbital nerve   Blocking this nerve will desensitise the upper eyelid except for the lateral and medial canthi. This block is useful for eye examination and insertion of tubing for sub-palpebral lavage. The supraorbital foramen can be palpated on the supraorbital process of the frontal bone, 5 – 7 cm above the medial canthus of the eye. Use a fine, 2.5 cm (1") needle. Insert the needle 1.5 – 2 cm into the foramen, and inject 2 ml. Inject a further 1 ml as the needle is withdrawn from the canal and 2 ml subcutaneously.

Auriculopalpebral nerve   This block does not provide desensitisation. The orbicularis oculi muscles are paralysed, making this a useful block for eye examination. Use a fine 2.5 cm (1") needle, and 5 ml local anaesthetic. Palpate the depression caudal to the vertical ramus of the mandible at the temporal part of the zygomatic arch. Insert the needle and inject as the needle is withdrawn.

SHEEP AND GOAT

Cornual   There are cornual branches of both the lacrimal and infratrochlear nerves supplying the horn in goats. To block the cornual branch of the lacrimal nerve, the needle is placed as close as possible to the caudal ridge of the root of the supraorbital process to a depth of 1 – 1.5 cm. To block the cornual branch of infratrochlear nerve, the needle is placed as close as possible to the dorsomedial margin of the orbit, to a depth of 0.5 cm. At each site, inject 2 – 3 ml. (Adult dose)

Disbudding in kids can be carried out after blocking these two nerves, but with greatly reduced volumes of local anaesthetic. As disbudding of kids is carried out as early as three days old, the risk of administering toxic doses of local anaesthetic is high. It may be simpler and safer to induce general anaesthesia with an inhalation agent delivered by mask for disbudding.

Paravertebral   Paravertebral anaesthesia can be carried out in sheep and goats using the technique described for cattle. The landmarks are easier to palpate and therefore greater accuracy of needle placement can be achieved. The site of injections is 2.5 – 3 cm from the midline and the volume of injection is reduced to 3 – 5 ml ventral to the transverse process and 2 ml dorsal.

Intra-testicular   Anaesthesia can be achieved for castration by injection of local anaesthetic directly into the testes. Depending on the size of the animal, 2 – 10 ml is injected into each testicle. The site of the skin incision must be infiltrated.

Epidural   Provided aseptic technique is observed, epidural anaesthesia is a useful procedure for intra-abdominal, pelvic or hindlimb surgery.   Sheep will readily accept recumbency, and they are easily restrained.   The technique is very similar to that described for dogs. The sheep is restrained on its side with the lumbosacral spine in full flexion. The site should be clipped and cleaned. The cranial borders of the ilia are palpated. A line joining these crosses the dorsal spinous process of the last lumbar vertebra. The site for injection is just caudal to this process. The skin over the site is infiltrated with 2 – 3 ml anaesthetic. An injection of (3 – 4 ml in a 50 kg sheep) is sufficient for posterior analgesia and 5 ml should give paralysis of the hindquarters in 2 – 15 minutes. If a unilateral block is required, the animal should remain in lateral recumbency with the side to be blocked down. If a bilateral block is required, the sheep should be kept on its back. If lignocaine 1.5% with adrenaline 1:100,000 is used, the block should last for about two hours.

OXYGEN TOXICITY

Although oxygen is essential for efficient synthesis in all higher mammals, it is toxic in excess. Toxicity is dependent on

- Partial pressure
- Duration of exposure

Very high hyperbaric pressures of oxygen cause damage rapidly. In veterinary anaesthesia, 100% oxygen is mostly delivered at 1 atmosphere for relatively short periods of time, making oxygen toxicity rare.

There is wide inter- and intra-species variation. Small animals are, in general, more susceptible than large. Administration of 100% oxygen at 1 atmosphere is lethal in mice in 1 – 2 days, dogs in 2 – 3 days and monkeys in 2 weeks.

Oxygen toxicity and radiation damage share many characteristics and it can be surmised that both are due to free radical damage.

Toxic effects

"Physiological" effects

- Ventilation/perfusion changes
- Vasoconstriction
- $\downarrow$ buffering capacity of the blood

Lungs

Exudative phase

- Endothelial cell damage
- Interstitial and perivascular oedema
- Intra-alveolar oedema
- Alveolar collapse (absorption collapse)
- Capillary congestion

Proliferative phase

- Hyperplasia of septal cells
- Fibrosis and fibroplastic proliferation

Eye

- Retrolental fibroplasia
- Retinal vasoconstriction

Blood

- Depressed haemopoiesis

Myocardium

- Depressed contractility

Other

- Toxic effects on all tissues, organs and enzyme systems

Clinical implications

If a patient requires prolonged high oxygen therapy, it is safest to deliver a mixture of medical air and oxygen such that delivered oxygen concentration is 40 – 50 %.

However, it is important to deliver whatever it takes to maintain the partial pressure of arterial blood oxygen at 90 – 100 mm Hg so that the patient is not at risk of hypoxic tissue damage, especially to the CNS..

EUTHANASIA OF SMALL ANIMALS
Prepared by HLKeates & LJFilippich

Eu ≡ good   *Thanos* ≡ death

Euthanasia means the provision of a good death.   Veterinarians are called upon to euthanase small animals for a variety of reasons. For example: -
- To terminate suffering in a sick animal
- Owners may be unable or unwilling to pay for treatment that is essential for the animal's well being or long term survival
- Owners may be shifting house and are unable to take the animal to the new dwelling (eg unit, nursing home, retirement accommodation etc)
- The animal may have developed, through illness or altered management, intolerable behaviour (eg defaecating or urinating inside the house.)
- Owners may simply not be willing to keep the animal any longer.
- Aggressive behaviour

Although there may be occasions when the veterinarian can offer alternatives to euthanasia that the owner may accept, it is essential that the owner is treated with respect and supported whilst making a very difficult decision.

If euthanasia is to proceed, the veterinarian's responsibility is to provide a 'good death', one with minimal discomfort to the animal. If the animal is difficult to handle, heavy sedation, delivered prior to euthanasia, by the subcutaneous route may be advisable. If the owner wishes to remain with the animal whilst euthanasia is performed, the veterinarian should describe the process and the animal's likely reaction. For example, the following refers to euthanasia using an overdose of the anaesthetic agent, sodium pentobarbitone: -
- In most cases, the animal will lose consciousness over 20 - 30 seconds. If used at anaesthetic dose rates, sodium pentobarbitone takes about three minutes to have its maximum effect, but the euthanasia dose is much higher and death will occur very rapidly.
- In some cases, especially if the animal is agitated or excited, there may be a brief period of paddling or confusion. Sometimes, the animal will have one or more agonal breaths. Agonal breathing is the final and dramatic response of the respiratory centre to hypoxia. During euthanasia, agonal breathing is the result of circulatory failure. An agonal breath is a violent intake of breath, usually accompanied by opening of the mouth, flexing of the neck and shuddering, jerky body movements. This can be very disturbing to the owner, especially if they are not prepared.   It is important that the owner knows that the animal is unconscious ie unaware when it gives an agonal breath.
- Some animals will twitch after the circulation and ventilation have ceased. Again, the animal is unaware at this time.

Whatever method is chosen for euthanasia, the veterinarian must be certain that the animal is dead. The following routine check will ensure that euthanasia has been successful. Examine the patient thoroughly and determine: -
- absence of heart sounds
- absence of apex beat
- absence of femoral artery pulse
- absence of ventilatory effort
- widely dilated pupils
- pale or cyanotic mucous membranes

If you are in any doubt as to whether the animal is dead, wait a few minutes and recheck. You may need to administer more drug.

Sodium pentobarbitone overdose.   The most common method of euthanasia in small animals is to give an overdose of the anaesthetic, sodium pentobarbitone, which is

marketed in a very high concentration to be used exclusively for euthanasia. *These solutions should not be used to provide anaesthesia, as they are not sterile and frequently have additives which are toxic.* Table 1 gives details of some of the available solutions.

It is important to note that concentrated solutions of sodium pentobarbitone are highly alkaline and hypertonic. They are marketed for intravenous administration where immediate dilution in the blood reduces the alkalinity and the tonicity. *Injection undiluted into tissue or a body space (eg thoracic or abdominal cavity) may cause irritation and pain due to caustic burn or osmotic effect.*

There is a recommended dose rate for sodium pentobarbitone for euthanasia on the label of each of the marketed solutions. However, it must be remembered that some animals will require a larger dose and all animals must be carefully checked so that more solution can be administered if necessary.

IV administration. If the owners wish to be present during euthanasia, it is advisable to place a catheter connected to an extension tube so that handling of the animal is minimised. If the animal is agitated or difficult to restrain, a subcutaneous injection of an opioid analgesic and a tranquilliser 20 minutes before administration of sodium pentobarbitone will result in a calmer and more compliant patient.
When euthanasing a horse, bear in mind that accidental extravascular injection will result in pain and an agitated horse, and the prior placement of a catheter is an important step in this process. Administration of less than the dose required to render the animal unconscious can result in extreme excitement, staggering and falling. This is dangerous for the handler and

Intraperitoneal administration. There are occasions when administration by other routes is advisable: -
- ❑ very small patients (eg mice, rats, birds, kittens, puppies)
- ❑ animals with difficult or traumatised veins
- ❑ animals that will not tolerate an intravenous injection

If injecting into the peritoneal space, insert the needle through the *linea alba*, as the tissue in the ventral midline is less sensitive than the rest of the body wall.
*It is advisable that the euthanasia solution be diluted to reduce the alkalinity and osmolality prior to intraperitoneal injection. Dilute **at least** 1:5 with saline. This may be the method of choice for puppies and kittens as they rarely show signs of discomfort from mid-line abdominal needle placement or injection and they will quietly lose consciousness. Similarly, feral cats can be given intraperitoneal euthanasia solution by injection through the wire mesh of a cage. This is best achieved by inverting the wire topped cage so that the cat is sitting on the wire. It is usually possible to inject upwards into the cats abdomen. You may then need to give more euthanasia solution when the animal is unconscious.*

Injection into the kidney. It may be possible to stabilise a kidney through the body wall for injection directly into the renal tissue. This results in extremely fast uptake into the blood. *If you choose this technique, inject slowly, as rapid expansion of the renal capsule is very painful.*

Injection into the liver. By directing the needle into the anterior abdomen, euthanasia solution can be deposited into the hepatic tissue for very rapid uptake into the circulation. As with injection into renal tissue, inject slowly to avoid rapid hepatic capsule stretch.

Intrathoracic injection is not advised.

Intracardiac injection should be reserved for patients that are unconscious.

EUTHANASIA OF BIRDS.

LJ Filippich & HL Keates

As with small mammals, sodium pentobarbitone overdose is the most common method of euthanasia in birds.

Oral administration of sodium pentobarbitone into the crop using a crop needle is a simple and humane way to euthanase birds. The drug is rapidly absorbed and the bird rapidly becomes sedated without any excitement. Death occurs within 10 minutes.

Intravenous injection can be achieved via the jugular vein (most birds except ducks and pigeons) or into the medial tarsal vein (birds over 300g).

Intrahepatic injection of pentobarbitone (diluted to at least 1:5) can be achieved by inserting the needle at the tip of the xiphoid process of the sternum and directing cranially and somewhat laterally towards the upper right side of the abdominal cavity.

Alternatively, if the bird is distressed or difficult to restrain it can be first *anaesthetised with an inhalation agent* (eg halothane or isoflurane) delivered by mask before delivery of sodium pentobarbitone by any of the routes described above.

Table 1. **Examples of available euthanasia solutions: brand names, supplying company, concentration of sodium pentobarbitone, colour and pH of solution.**

Brand Name	Company	[Pentobarb. Na]	Other components	Dye	pH
Euthal	Delvet Pty Ltd	170 mg/kg	phenytoin 25 mg/ml	Brilliant blue	11 - .5
Euthanasia Fort Sol.	Apex Laboratories Pty Ltd	400 mg/kg	guiaphenesin 60 mg/ml	Blue	11 - 11.4
Lethobarb	Virbac, Pty Ltd	325 mg/ml		Green	11
Valabarb	Jurox Pty Ltd	300 mg/ml		Green (fluorescent)	10 - 11.5

Sodium pentobarbitone is a weak acid with a pKa of 8.1. Therefore it will be un-ionised (precipitated) in acidic solutions and ionised (soluble) in alkaline solutions.

Appendix 1.

Weight (kg)	Oxygen Uptake (ml/min)	PYHSIOLOGICAL VALUES Alveolar Ventilation (l/min)	Respiratory Minute Volume (l/min)		
1	10	0.16	0.23	-	0.32
2	17	0.27	0.38	-	0.54
3	23	0.36	0.52	-	0.73
4	28	0.45	0.65	-	0.91
5	33	0.53	0.76	-	1.07
10	56	0.90	1.29	-	1.80
15	76	1.22	1.74	-	2.44
20	95	1.51	2.16	-	3.03
25	112	1.79	2.56	-	3.58
30	128	2.05	2.93	-	4.10
35	144	2.30	3.29	-	4.60
40	159	2.54	3.64	-	5.09
45	174	2.78	3.97	-	5.56
50	188	3.01	4.30	-	6.02
55	202	3.23	4.62	-	6.46
60	216	3.45	4.93	-	6.90
65	229	3.66	5.23	-	7.33
70	242	3.87	5.53	-	7.74
75	255	4.08	5.83	-	8.16
80	267	4.28	6.11	-	8.56
85	280	4.48	6.40	-	8.96
90	292	4.68	6.68	-	9.35
95	304	4.87	6.96	-	9.74
100	316	5.06	7.23	-	10.12
150	429	6.86	9.80	-	13.72
200	532	8.51	12.16	-	17.02
250	629	10.06	14.37	-	20.12
300	721	11.53	16.48	-	23.07
350	809	12.95	18.50	-	25.89
400	894	14.31	20.44	-	28.62
450	977	15.63	22.33	-	31.27
500	1057	16.92	24.17	-	33.84
550	1136	18.17	25.96	-	36.34
600	1212	19.40	27.71	-	38.79

Appendix 2
Estimation of Alveolar Ventilation and Respiratory Minute Volume from Weight.

$$\text{Rate of uptake O}_2 \ (Q_{O2}) \quad = 10 \times W^{0.75} \text{ ml/min}$$

$$(W = \text{Wt in kg})$$

$$\text{Rate of production of CO}_2 \ (Q_{CO2}) \ = RQ \times Q_{O2} \text{ ml/min}$$

$$(RQ = \text{respiratory quotient} \approx 0.8)$$

$$\text{Alveolar fraction} \quad F_{ACO2} \quad \approx \ 0.05$$

$$(\text{ie alveolar gas is about 5\% CO}_2)$$

This means that 0.05 of the Alveolar ventilation (ml/min) is CO$_2$

$$\text{Thus, the total alveolar ventilation rate} \ = \quad Q_{CO2} \div F_{ACO2} = V_A \text{ ml/min}$$

$$\text{Respiratory tract dead space } F_D = 0.3 - 0.5 \text{ of RMV}$$

$$\therefore \ \text{RMV} \ (V_T) = V_A \div (1 - F_D)$$

eg 17 kg dog

$$Q_{O2} \quad = 83.7 \text{ ml/min} \qquad\qquad (\text{Wt}^{0.75} \times 10 = \text{O}_2 \text{ uptake in ml/min})$$

$$Q_{CO2} \ = 0.8 \times 83.7 \ \approx \ 67 \text{ ml/min} \qquad (RQ = 0.8)$$

$$V_A \quad = 67 \div 0.05 = 1340 \text{ ml} \qquad (\text{5\% alveolar gas is CO}_2)$$

$$V_T \quad = 1340 \div (1 - 0.3) \text{ to } 1340 \div (1 - 0.5) \text{ ml/min}$$
$$= 1{,}914 - 2{,}680 \text{ ml/min}$$

Appendix 3

ANAESTHESIA DOSE RATES FOR CATS AND DOGS   Brenda Dixon Trish Farry,
Helen Keates 2013
**The dose rates in this document are a <u>guide only</u>. Always consider the health
status and age of the patient before determining a dose rate. Always revise the
pharmacology of drugs you are unfamiliar with <u>before</u> you administer to the patient.**

TRANQUILIZERS AND SEDATIVES				
DRUG	**SPECIES**	**ROUTE**	**DOSE RATE**	**COMMENTS**
Acepromazine (2 mg/ml)	Cat	SC	0.05 mg/kg	unreliable
		IV	0.005 - 0.02 mg/kg	
	Dog	SC	0.01 – 0.05 mg/kg	
		IV	0.005 - 0.02 mg/kg	
Midazolam 5 mg/ml	Cat	SC, IV, IM	0.2 – 0.5 mg/kg	
	Dog	SC, IV	0.2 – 0.5 mg/kg	
Diazepam 5 mg/ml	Cat & Dog	IM, IV	Doses as for midazolam	Poorly absorbed SC. Give IM
OPIOIDS				
Methadone 10 mg/ml	Cat & Dog	SC	0.2 – 0.5 mg/kg	
		IV	0.1 – 0.2 mg/kg	
Morphine 5, 15, 30 mg/ml	Cat & Dog	SC	0.2 – 0.5 mg/kg	Will salivate, may vomit at high doses
		IV	0.1 – 0.2 mg/kg	
Butorphanol 10 mg/ml	Dog	SC, IV	0.1 – 0.2 mg/kg (up to 0.4 mg/kg)	
Buprenorphine 0.3 mg/ml	Cat & Dog	SC, IV	0.01 – 0.02 mg/kg	
Hydromorphone 2 mg/ml	Cat	SC	0.1 mg/kg	Cats will salivate, may vomit
	Dogs	SC	0.1 mg/kg	
Fentanyl 50 µg/ml	Cat & Dog	IV	1 – 5 µg/kg	
OPIOID REVERSAL				
Butorphanol 10 mg/ml	Cat & Dog	IV	0.01 mg/kg	Titrate
Naloxone 0.4 mg/ml	Cat & Dog	IV		Dilute 1:10 (to 0.04 mg/kg) and titrate using 0.5 – 1.0 ml increments
DISSOCIATIVE AGENTS				
Ketamine 100 mg/ml	Cat	SC	2 – 5 mg/kg	Combine with 0.5 mg/kg midazolam
		IV	1 – 2 mg/kg	Combine with 0.2 mg/kg midazolam

INDUCTION AGENTS				
Alfaxalone 10 mg/ml	Cat	IV	2 – 3 mg/kg	Titrate
	Dog	IV	1 – 2 mg/kg	
Propofol 10 mg/ml	Cat, Dog	IV	2 – 8 mg/kg	Frequent administration in cats can produce Heinz Body Anaemia
Thiopentone Make up dry powder to 2.5%	Cat. Dog	IV	10 – 15 mg/kg Titrate!	Don't use in sight Hounds or Belgian Shepherds. Irritant if injected extra-vascularly.
Ketamine 100 mg/ml + Midazolam 5 mg/ml	Cat, Dog	IV	Ketamine 5 mg/kg Midazolam 0.5 mg/kg	Mix in one syringe, deliver as bolus dose, can repeat ketamine dose if necessary.
PERIOPERATIVE ANALGESIA				
Methadone 10 mg/ml	Cat	IV	0.005 – 0.1 mg/kg	Titrate, repeated doses
		SC	0.3 – 0.5 mg/kg	4 – 6 hourly
	Dog	IV	0.1 – 0.2 mg/kg	Titrate, repeated doses
		SC	0.3 – 0.5 mg/kg	4 – 6 hourly
Morphine 5, 15 & 30 mg/ml	Cat	IV	0.005 – 0.1 mg/kg	Titrate, repeated doses
		SC	0.3 – 0.5 mg/kg	4 – 6 hourly
	Dog	IV	0.1 – 0.2 mg/kg	Titrate Incremental doses
		IV	0.1 – 0.2 mg/kg/h	Infusion
		SC	0.3 – 0.5 mg/kg	4 – 6 hourly
Fentanyl 50 µg/ml	Cat	IV	1 – 2 µg/kg	Titrate, repeated doses
	Dog	IV	2 – 5 µg/kg	
	Cat, Dog	Infusion	4 – 20 ug/kg/h	Intra-operative
		Infusion	4 - 8 µg/kg/h	Post-operative or conscious patient
Fentanyl transdermal patches	Cat,	Patch	4 – 6 µg/kg/h	Takes 6 – 12 h to take effect
	Dog		4 – 6 µg/kg/h	Takes 12 – 24 h to take effect
Ketamine 100 mg/ml	Cat, Dog	SC, IV	Loading dose 0.2 – 0.5 mg/kg Infusion 0.2 – 0.5 mg/kg/h	NMDA antagonist, opioid adjunct
Lignocaine 20 mg/ml	Cat, Dog	IV	Loading dose 0.5 – 1.0 mg/kg	
			Infusion 20 – 50 µg/kg/min	This is 1.2 – 3.0 mg/kg/h
Medetomidine 1 mg/ml	Cat, Dog	IV	Infusion 1 – 2 µg/kg/h	Good for dysphoria or as an adjunct to analgesia in appropriate patients
Epidural – see Appendix 13 of Anaesthesia				

ANTI-CHOLINERGIC AGENTS				
Atropine 0.6 mg/ml	Cat, Dog	SC, IV	0.02 – 0.04 mg/kg	Vagolytic effect lasts 30 min, treatment of bradycardia
Glycopyrrolate 0.28 mg/ml	Cat, Dog	SC, IV	0.011 mg/kg	Vagolytic effect lasts 2 – 3 h, treatment of bradycardia
TREATMENT OF HYPOTENSION ASSOCIATED WITH GENERAL ANAESTHESIA – TREATMENTS IN GENERAL ORDER. If the patient is bradycardic, treat with anticholinergic drugs first.				
Crystalloid	Cat	IV	5 ml/kg up to 10 ml/kg	Give bolus doses
	Dog	IV	5 ml/kg up to 20 ml/kg	
Voluven	Cat, Dog	IV	Bolus 5 ml/kg up to 20 ml/kg in 24 h	Artificial colloid, hetastarch
Metaraminol 10 mg/ml	Cat	IV	0.005 – 0.01 mg/kg	α-agonist, vasoconstriction Lasts 10 – 15 min Give incrementally. May see profound bradycardia
	Dog	IV	0.01 mg/kg	
Phenylephrine (Neosynephrine® 10 mg/ml)	Cat, Dog	IV	1 – 3 µg/kg/min	α-agonist, vasoconstriction Useful in cats with hypertrophic cardiomyopathy
Dopamine 40 mg/ml	Cat, Dog	IV	5 – 20 µg/kg/min	Chronotrope, inotrope, vasoconstrictor Renal protective at 2 – 3 µg/kg/min
Dobutamine	Cat, Dog	IV	2 – 10 µg/kg/min	Chronotrope, inotrope, decreases systemic vascular resistance
Noradrenaline (Levophed® 1 mg/ml)	Cat, Dog	IV	0.1 – 0.5 µg/kg/min Up to 2 µg/kg/min	Chronotrope, inotrope, potent vasoconstrictor
Vasopressin 20,000 m Units/ml	Cat, Dog	IV	60 – 240 m Units/kg/min	Potent vasoconstrictor
Adrenaline 1 mg/ml	Cat, dog	IV	0.05 – 0.4 µg/kg/min	Potent chronotrope, inotrope, vasoconstrictor

ASSOCIATED TREATMENTS				
Regurgitation				
Esomeprazole	Cat, Dog	IV	1 mg/kg SLOWLY/24 h	
Metoclopromide	Cat, Dog	IV	1 – 2 mg/kg/24 h as an infusion	
Antibiotics				
Cephalothin	Cat, Dog	IV	22 mg/kg 2 hourly during surgery	
Ampicillan	Cat, Dog	IV	22 mg/kg 8 hourly	
Metrionidazole	Cat, Dog	IV	10 mg/kg SLOWLY twice daily	Cover bag, light sensitive
Enrofloxocin	Cat, Dog	SC	5 mg/kg once daily	

Appendix 4
VETS4012 SURGERY PRACTICAL SESSIONS – NOTES FOR THE ANAESTHETIST
VETERINARY CLINICAL FACILITY, GATTON TUESDAYS AM

ANAESTHESIA
At your anaesthesia/surgery practical classes, you will be required to anaesthetise a pig. You will work in teams of four, with two students performing the tasks of the anaesthetist and two performing surgery. The anaesthetists may need help from the rest of the team until the pig is anaesthetized.
Pigs
Treat your patient with respect and minimise any stress the pig may be under.
Requirements.
Bring *a stethoscope, a watch, and a pen.* Please be early. The sessions are scheduled for 8.00AM and we will hold a tutorial/discussion before the prac commences.
Records.
There are anaesthetic record forms available for you to record your pre-anaesthetic findings, drugs and doses, anaesthetic machine settings, heart rates, respiratory rates etc.
Prepare you anaesthetic machine
All stations have circle absorber breathing systems. Most have the vaporizers out of circle (VOC, the rest have vaporizers in-circle (VIC)). Identify the following: -
- Soda lime absorber/s
- Two one-way valves
- One spill valve (pop-off valve)
- Flow meter
- Reservoir bag
- Breathing tubes that make a circle (of sorts)
- Vaporizer

Assemble and test your anaesthetic machine (pp 123-5). Convince yourself that there is a one-way valve between the patient and the reservoir bag in either direction. Determine whether you have an in- or out-of-circle vaporizer. There are many ways to set these machines. The following **suggestions** may help you get started.
- Vaporizer-in-circle – Start with an oxygen flow rate of 2 - 3 $\times$ O_2 uptake and vaporizer set on just over half. (Goldman - ¾, Stephens – $^5/_8)$ Turn the vaporizer down with time.
- Vaporizer-out-of –circle – Start with an oxygen flow rate of 0.75 - 1.0 $\times$ Alveolar ventilation and vaporizer on about 1.5%.

Remember –
- ❖ *These settings are just to get you started – as the anaesthetist, you must adjust what you deliver to the pig in response to the depth of anaesthesia. Keep monitoring!!!*
- ❖ *If the oxygen flow rate is > the patient's oxygen requirement, the spill valve must be open.*

**In first semester, we will prepare your premedicant drugs for you, but you must demonstrate that you have calculated doses and volumes accurately. .

Preparation and Premedication.
You must prepare <u>everything you will need before you premedicate your pig.</u>
Now, get ready for the next step. DO NOT PROCEED until you have prepared the following: -
- **Induction agent (in syringe, labeled and ready)**
- **Several endotracheal tubes (appropriate sizes, cuffs checked), tube ties, syringe for cuff inflation and a stylette to insert in the tube to facilitate intubation.**
- **Laryngoscope (in your hand, checked and in working order)**
- **Anaesthetic machine assembled, tested by YOU, vaporizer filled and you know how it works and what setting to choose!!! (chart on wall for O_2 uptake, alveolar ventilation, respiratory minute volume)**
- **Tutor at the ready**

Premedicate your patient by intramuscular injection in the muscles of the cranial aspect of the upper hind limb. Try a very fine needle so it will not hurt eg 23g, ¾ "26g, ½". Stay with the pig once you have injected and take it to the surgery as soon as it is sedated. This will have an effect very rapidly. Carry the pig to your work station and apply a mask delivering oxygen over the pigs snout. Your tutor may decide to deliver the inhalation anaesthetic agent (isoflurane) at this time.

Suggested drugs and dose rates: -
Zoletil 100 (zolazeopam 50 mg/ml + tiletamine 50 mg/ml)   *4 mg/kg*
Butorphanol (10 mg/ml)   *0.15 mg/kg*

Palpate the patient's trachea, and select a tube that will fill the lumen. The length of the tube is important – it should go from the patient's nose far enough through the larynx for the cuff to be in the trachea, but not through the thoracic inlet. The point of the shoulder is a useful landmark. Select a few tubes, smaller and larger than the one you think is OK. One of these should fit. Pigs have relatively small tracheas. Inflate the cuffs to check for leaks. Lubricate the tubes. Have a tube tie ready.

Catheterization of a peripheral vein.
Your tutor will assist you to secure a venous catheter. Ear veins and limb veins are useful in pigs.

Induction.
Induce anaesthesia with intravenous alfaxalone 10 mg/ml to effect. This will probably be less than 0.5 mg/kg.   Calculate about 1 mg/kg and draw up this amount in your syringe, but **remember – you are going to give the drug intravenously, slowly to effect ie by titration!!!** The required amount of drug/kg varies widely from animal to animal, and titration is the only way to get it right for THIS patient. **It is unlikely that you will need the entire volume you have drawn up. You may not need to administer alfaxalone at this time.** Alternatively, induce with isoflurane.
You want the patient just deep enough to allow you to intubate and connect to the anaesthetic machine.
Important fact:- alfaxalone takes about 20 – 30 seconds to take effect, and the animal begins to recover from a single dose within a few minutes. This means, you have to develop the skill of injecting at an appropriate rate – too fast and they are rapidly too deep; too slow, and they are recovering as fast as you inject!!   Titration with either alfaxalone or isoflurane means ***checking depth before administering more drug***. Don't panic – you'll work it out!!

Intubation.
Your tutor will advise and help you. Use a laryngoscope. We want you to view as many larynx's/pharynx's as possible before you graduate!

Maintenance.
Once your patient is anaesthetized, turn on the flow meter, check that the spill valve is open, turn on the vaporizer and connect your patient to the breathing system. ***Never connect a patient to a breathing system until you have turned on the oxygen flow.***

Monitoring.
Throughout the surgical exercise, the **anaesthetists MUST stay with the patient and monitor**. Check pulse rate and character, respiratory rate and character, eye reflexes, jaw tone and be aware of any response to surgery.   **Keep good records.**

Most common problems at surgery pracs. : -
- Endotracheal tube placed in the oesophagus   Anaesthesia lightens or patient's breathing is obstructed. Patient may become cyanotic.
- High flow rate in machine with vaporizer-in-circle. Anaesthesia lightens
- Large patient, hot day, vaporizer-in-circle, low O_2 flow rate. Anaesthesia **very** deep
- Soda lime exhausted. Patient apnoeic with very pink gums Use the available capnograph to measure the pCO_2 levels.

We know that most of you have never done most of the tasks we have set you before you attend these pracs. We are there to help you. However, try to work out as much as you can before you call us. ***Look up your notes during the prac and do your best to familiarize yourself the information therein.***

Read all relevant material (for both anaesthesia and surgery) before you arrive at the prac!

Appendix 5

MONITORING ANAESTHESIA AT PRACTICAL SURGERY CLASSES

HOW CAN I TELL HOW DEEP THE PATIENT IS?

MOVEMENT/MUSCLE TONE
Spontaneous voluntary movement = very light or awake!
Movement in response to pain = very light   (Pinch a toe to test)
Tone of muscles (limbs, jaw) becomes less as the anaesthetic deepens. Test jaw tone regularly so that you are able to detect changes.

MUCOUS MEMBRANE COLOUR
Mucous membranes should be pink.

❖ *Cyanosis.* We do not have the ability to detect colour changes in the early stages of hypoxia. Only a pulse oximeter or blood gas analysis can do this. By the time you can see cyanosis, things are serious. Remember, blue means deoxygenated haemoglobin – so your patient needs oxygen. If you are delivering a high concentration of oxygen to the patient, ventilate the patient manually and call a tutor. Check tube placement.

❖ *Injected mucous membranes* can be caused by hypercapnia. If this is a possibility, check the status of your soda lime and that your breathing circuit is put together correctly. If you have a **non-rebreathing system**, check that your fresh gas flow rate is adequate. If your patient is hypercapnic, it needs ventilation, not a higher O_2 flow rate.

RESPIRATION
Apnoea can mean light or deep or lots of other things.

❖ At very *deep planes of anaesthesia*, tidal volume will usually progressively decrease and eventually, the animal will be apnoeic. Decrease in tidal volume rather than rate may signal that the patient is too deep.

❖ Animals that are intubated when *very lightly anaesthetized* often breath hold. Check the colour of the mucous membranes and the pulse characteristics. If these parameters are OK, stimulate the animal to take a breath by pressing firmly on the costo-chondral junctions. If 60 seconds pass without the animal taking a breath, inflate the lungs by squeezing the reservoir bag. Usually, one or two breaths per minute will suffice for the few minutes that it takes for the animal to start breathing on its own. Occasionally, the animal is apnoeic because it has received an overdose of induction agent. Then positive pressure ventilation will be necessary for longer.

❖ Hypercapnia may result in stimulation of respiration in the early stages, but eventually, the animal will become apnoeic (with injected mucous membranes.) Check the soda lime, the breathing circuit and the fresh gas flow rate (if you have a non-rebreathing circuit). Anaesthetised animals mostly will not respond to high CO_2 levels.

The **character of the respiratory movements** is one of the most valuable signs of depth.

❖ *Jerky, irregular breathing*, or even breath holding may indicate that the patient is light and responding to the surgery.

❖ *Slow, regular, shallow breathing* usually means deep.

❖ A *big inspiratory effort, with the thorax 'sucking in'* over the heart area means obstructive breathing. Is the reservoir bag moving? If not, the animal is likely to be totally obstructed. Is the endotracheal tube in the trachea? CHECK! Is the tube patent? Kinked? Do you need a tutor? (yes!)

CARDIOVASCULAR SYSTEM
Pulse
Feel pulses at every opportunity!! Assess *each* of these four parameters *separately* every time: -

❖ *Vessel tone*
❖ *Pulse amplitude*
❖ *Rate*
❖ *Rhythm*

Small arteries give you valuable information. If you can feel a tongue pulse at the start of surgery but not at the end – maybe the blood pressure has dropped.

In the early stages of anaesthesia, the pulse is often fast with a large amplitude and is sometimes irregular.

If the animal is deep, the pulse is likely to be harder to find (low tone and/or amplitude) and slow.

Heart sounds

Continuous monitoring of heart sounds is desirable. If you are listening to the heart and regularly recording the rate, you should have early warning of dysrhythmias or declining rate.

Palpebral Reflex/Eye Position

A lightly anaesthetized animal will usually have a brisk palpebral reflex ie touch the palpebrae, and the eye will blink. In most species, as depth of anaesthesia increases, the reflex is obtunded until it is lost. Be careful not to touch the cornea, as even a very deeply anaesthetized animal is likely to have a corneal reflex.

The position of the eye changes with depth. As the animal looses consciousness from the induction agent, the eye will roll down. As anaesthesia deepens further, the eye will roll back up, and eventually the pupil will dilate.

A dog with no palpebral reflex, blankly staring ahead with dilated pupils is likely to be deep.
BUT - never decide on depth of anaesthesia on the basis of eye signs alone!!!

ELECTRONIC MONITORS

All stations in the teaching surgery have a monitor. All monitors have the capacity to do pulse oximetry. Most have the facility to measure blood pressure oscillometrically or using a Doppler flow detector. Five stations have the capacity to sample and analyse circuit gases. These monitors will give respiratory rate and inspired and end tidal concentrations (or partial pressures) of CO_2, oxygen, and anaesthetic agent eg isoflurane. Make sure that you acquire the skills to connect these monitors to the patient. You must be able to interpret the results and respond appropriately. The tutors are there to help you, but in the end, it's up to you. You will be examined on the use of these monitors.

REMEMBER – THESE ARE JUST GUIDELINES AND STARTING POINTS – ANYTHING CAN HAPPEN.

MONITOR EVERYTHING ALL THE TIME *AND USE ALL THE AVAILABLE INFORMATION* TO DECIDE WHERE YOUR PATIENT IS ON THE CONTINUUM FROM
AWAKE TO DEAD!!!

If in doubt, call a tutor!

☠

Appendix 6

INFLATION OF AN ENDOTRACHEAL TUBE CUFF

Select a tube of a diameter and length you think will be suitable. Then select at least one smaller and one larger. Test the cuffs for leaks and lubricate the cuff ends of the tubes.

The cuff must be inflated *just enough* to seal the trachea.

What if the cuff is not inflated enough?
- *It may not be possible to perform IPPV*
- The patient may aspirate saliva, regurgitated gastric contents
- The patient may entrain atmospheric air as they inhale. This will decrease inspired isoflurane and oxygen concentrations
- Isoflurane may escape to the atmosphere on exhalation (OH&S risk)

What if the cuff is over-inflated?
- Pressure from the cuff on the tracheal mucosa will reduce perfusion to these tissues and may cause tissue necrosis. If scar tissue is formed, contraction may occur eventually leading to reduced tracheal lumen.
- As the tube warms to body temperature, it softens. The pressure in the cuff may be enough to deform and close the tube.
- The cuff may migrate forward and obstruct the opening of the tube.

How to inflate the cuff to an appropriate level: -
1. Select a syringe and fill with air.
2. Connect the syringe to the pilot tube of the endotracheal tube.
3. Close the spill valve
4. *With your ear close to the patient's mouth*, squeeze the reservoir bag to increase the pressure in the breathing circuit and the patient's lungs. About 20 cm wather pressure will suffice. There will be a manometer in line. You may have an assistant to squeeze the bag for you, in which case you need to have your head turned to the reservoir bag so you know when there is positive pressure applied. *Listen for a leak.*
5. If there is no leak, you need not inflate the cuff. However, you should check again in 10 – 20 minutes so ensure that a leak does not occur as a result of muscle relaxation caused by the inhalation anaesthetic agent.
6. If there is a leak, inject a small volume of air into the cuff, and test again. Test and inflate until no air escapes around the cuff.
7. Now, deflate slightly until there is a slight leak. Inject just enough air to again seal the tube. This step is included to make sure you have not over-inflated.

The whole process should not take more than 4 -5 breaths.

8. ***NOW OPEN THE SPILL VALVE AND LEAVE IT OPEN***

Appendix 7

a)   How to check a circle absorber with the vaporizer out of circle.

1.   Check the oxygen supply. Turn on the reducing valve on the oxygen cylinder and check the gauge.   Always have a spare cylinder.
2.   Check the emergency flush button. Press the emergency flush button (usually red, near the common gas outlet) and you should hear a rapid flow of oxygen
3.   Check the oxygen flow meter. Turn the flow meter as high as it will go to make sure it can reach and sustain a high flow. Now make sure it will sustain a 2 l/min flow.
4.   Check the gas & vapour supplies for leaks.
a)   Turn the flow meter to 2 l/min. *Briefly* occlude the common gas outlet. The bobbin/ball of the flow meter should drop because of back pressure. If it doesn't, there is a leak. (This works for flow meters in which the gas flows up a tube and the column of gas supports a ball or bobbin)
b)   Now turn the vaporizer on. Repeat step 2a). This will establish if there is a leak through the vaporizer. *Now turn the oxygen flow meter off.*
5.   Check that the vaporizer is full.
6.   Check the CO_2 absorber. Check the colour.   (After the patient has been connected for 10 – 15 minutes, check that there is a warm zone in the canister.)
7.   Assemble the breathing circuit. Attach a hose from the common gas outlet to the absorber. Attach the inspiratory and expiratory limbs of the circle. Attach the reservoir bag.
8.   Check the circle for leaks. Occlude the y-piece where the patient's endotracheal tube will connect. Close the spill valve. Fill the reservoir bag until the wrinkles just disappear. ***Turn the oxygen off.*** Wait for 10 – 20 seconds. If there is a leak, the wrinkles will reappear in the bag.
9.   Check the spill valve function. Following straight on from 8, keeping the spill valve closed and the Y-piece occluded, release the pressure by opening the spill valve. The reservoir bag will deflate if the valve releases correctly. ***Do not close the spill valve again.***
10.   Check that the uni-directional (one-way) valves work. Partly fill the reservoir bag.   (i) Attach a small reservoir bag to the y-piece to act as the patient's lungs. Alternately squeeze the two bags to test the activity of each valve independently or
 (ii) Squeeze the reservoir bag to test the inspiratory valve and blow into the y-piece to test the expiratory valve (Do not put your mouth on the y-piece).

**ALWAYS LEAVE THE SPILL VALVE OPEN AT THE END OF THE CHECK.
PERFORM THIS CHECK BEFORE EACH PATIENT.**

Never attach the patient to any breathing system without first turning the oxygen flow meter on.

Never have two breathing systems or two vaporisers on one machine.

b) How to check a Komesaroff Circle Absorber.

1.   Check the oxygen supply. Turn on the reducing valve on the oxygen cylinder and check the gauge.   Always have a spare cylinder.

2.   Check the oxygen flow meter. Turn the flow meter as high as it will go to make sure it can sustain a high flow.

3.   Check the emergency flush button. Press the flat top of the flow meter and you should hear a rapid flow of oxygen.

4.   Check the vaporiser. Make sure that the dial on the top moves easily.   There should be liquid anaesthetic in the glass jar, to a depth of at least one centimetre.

5.   Check the CO_2 absorber. Check the colour.   (After the patient has been connected for 10 – 15 minutes, check that there is a warm zone in the canister.)

6.   Assemble the breathing circuit. Attach the inspiratory and expiratory limbs of the circle and the reservoir bag.

7.   Check the circle for leaks. Occlude the y-piece where the patient's endotracheal tube will connect. Close the spill valve. Fill the reservoir bag until the wrinkles just disappear. ***Turn the oxygen off.*** Wait for 10 – 20 seconds. If there is a leak, the wrinkles will reappear in the bag.

8.   Check the spill valve function. Following straight on from 7, keeping the spill valve closed and the Y-piece occluded, release the pressure by opening the spill valve. The reservoir bag will deflate if the valve releases correctly. ***Do not close the spill valve again.***

9.   Check that the uni-directional (one-way) valves work. Partly fill the reservoir bag.   (i) Attach a small reservoir bag to the y-piece to act as the patient's lungs. Alternately squeeze the two bags to test the activity of each valve independently or
 (ii) Squeeze the reservoir bag to test the inspiratory valve and blow into the y-piece to test the expiratory valve (Do not put your mouth on the y-piece).

ALWAYS LEAVE THE SPILL VALVE OPEN AFTER YOUR CHECK.

Perform this check before each patient.

.NEVER ATTACH THE PATIENT TO ANY BREATHING SYSTEM WITHOUT THE OXYGEN FLOW METER TURNED ON

c) How to check a Stephens Circle Absorber.

1.   Check the oxygen supply. Turn on the reducing valve on the oxygen cylinder and check the gauge.   Always have a spare cylinder.

2.   Check the oxygen flow meter. Turn the flow meter as high as it will go to make sure it can sustain a high flow.

3.   Check the emergency flush button. Press the emergency oxygen button (usually red) and you should hear a rapid flow of oxygen.

4.   Check the vaporiser. Make sure that the dial on the top moves easily. There should be liquid anaesthetic in the glass jar, to a depth of at least one centimetre. Check that the metal sleeve inside the glass jar is pushed up – not down near the surface of the liquid anaesthetic. (Sleeve down results in a higher vaporiser output)

5.   Check the CO_2 absorber. Check the colour.   (After the patient has been connected for 10 – 15 minutes, check that there is a warm zone in the canister.)

6.   Assemble the breathing circuit. Attach the inspiratory and expiratory limbs of the circle and the reservoir bag.

7.   Check the circle for leaks. Occlude the y-piece where the patient's endotracheal tube will connect. Close the spill valve. Fill the reservoir bag until the wrinkles just disappear. ***Turn the oxygen off.*** Wait for 10 – 20 seconds. If there is a leak, the wrinkles will reappear in the bag.

8.   Check the spill valve function. Following straight on from 7, keeping the spill valve closed and the Y-piece occluded, release the pressure by opening the spill valve. The reservoir bag will deflate if the valve releases correctly. ***Do not close the spill valve again***.

9.   Check that the uni-directional (one-way) valves work. Partly fill the reservoir bag.   (i) Attach a small reservoir bag to the y-piece to act as the patient's lungs. Alternately squeeze the two bags to test the activity of each valve independently or
 (ii) Squeeze the reservoir bag to test the inspiratory valve and blow into the y-piece to test the expiratory valve (Do not put your mouth on the y-piece).

ALWAYS LEAVE THE SPILL VALVE OPEN AFTER YOUR CHECK.

Perform this check before each patient.

NEVER ATTACH THE PATIENT TO ANY BREATHING SYSTEM WITHOUT THE OXYGEN FLOW METER TURNED ON.

Appendix 8 Bovine anaesthesia practical session

This practical class has been replaced with a sheep anaesthesia class. These notes have been left in for your reference.

BOVINE PARAVERTEBRAL BLOCK

Block *nerves T13, L1, L2 and L3* cranial to the *transverse processes of L1, L2, L3 and L4.* L2 is the first palpable transverse process caudal to the last rib.

Remember, the nerves travel in a caudo–lateral direction and the transverse processes of the lumbar vertebrae run cranio–lateral.

Cows usually have 6 lumbar vertebrae, but palpation of all the transverse processes may be difficult.

- *Imagine a line from the most prominent part of the transverse process of a lumbar vertebra to the midline, perpendicular to the spine*. Do this for the processes of L2, L3 and L4 and *estimate where this line would be for L1*. (L1 is rarely palpable.)
- Identify the point *5 cm from the midline* on each of these imaginary lines.
- At these points, inject *2 – 3ml local* anaesthetic using a 26 G needle. Ideally, leave a visible skin bleb.
- Using a size 15 scalpel blade*, stab* through the skin at these sites.
- *Insert a 6" 18G short bevelled needle* through the incision perpendicular to the surface. The needle should *hit the anterior edge of the transverse process. Walk the needle off the front of the process*, and feel it pass through the inter-transverse ligament.
- *Inject 10 –1 5ml beneath the ligament*.
- Withdraw the needle, and inject *5 – 7 ml above the ligament*.

Signs that your block has been successful
- *Warm skin* in the affected area
- *Cow flexed* away from the affected side
- *No response* to needle prick on affected side.

DO NOT THROW OUT THE STAINLESS STEEL PARAVERTEBRAL NEEDLES. There are not enough needles for one per injection. Handle only the hub of the needle, and place it back in the sterile packet for the next person to use. At the conclusion of the procedure, rinse the blood out of the needles and return them for sterilization.

BOVINE EPIDURAL ANAESTHESIA
- *Identify the C1 – C2 space*, between the dorsal spinous processes of C1 and C2. The C1 – C2 joint is the most mobile joint at the top of the tail when the tail is moved up and down. This should be just anterior to the anal folds.
- Using a 1.5" needle, preferably with a short bevel*, insert the needle perpendicular to the skin* until it hits the floor of the spinal canal. Withdraw slightly.
- *Check position* by
    - Injecting air or saline. NO resistance indicates correct placement.
    - Fill needle hub with saline or lignocaine once the needle is through the skin but *before* advancing the needle towards the spinal canal. The drop of liquid will be "sucked" in by the negative pressure in the epidural space.

- *Inject 4 – 5 ml local anaesthetic* into the epidural space. (eg 1 ml 2% lignocaine per 100 kg.)

Signs that the block has been successful -
- Tail and escutcheon are *insensitive* to needle prick.
- Tail is *without tone*.

BOVINE GENERAL ANAESTHESIA

Cattle are 10 times more sensitive to xylazine than horses and dogs!!!

PREMEDICATION
Xylazine   (Deep sedation)
Intramuscular   0.2 - 0.4 mg/kg   (1 – 2 ml/100 kg of 20 mg/ml)
Intravenous   0.1 – 0.15 mg/kg   (0.5 – 0.75 ml /100 kg of 20 mg/ml)

INDUCTION
Ketamine
Intravenous   2.2 mg/kg
If necessary, top-up with one-third to 1/3 – ½   dose of ketamine.

OR

Thiopentone
Intravenous   1 gm/200kg after xylazine
If necessary, top-up with 0.5gm increments.

INTUBATION
Intubate the trachea with the cow in sternal recumbency, head elevated.
By palpation, insert a 26 – 30mm cuffed endotracheal tube.

Appendix 9 Equine anaesthesia practical session

This practical class is no longer offered. These notes have been left in for your reference.

Choose a suitable time and place to induce anaesthesia. Warn all others in the vicinity that your horse is about to become recumbent BEFORE you administer the induction agent.

Once your horse in anaesthetised, take care of its eyes – protect the lower eye by lifting the head instead of dragging, putting the head on a cloth with the eyelid closed. Protect the upper eye with Lacrilube®. DON'T put your finger on the cornea.

1. Examine your patient.
2. Place a jugular catheter and secure it with super-glue.
3. Premedicate.
4. Induce anaesthesia.
5. Whilst your horse if anaesthetised, each member of your team should insert an endotracheal tube

Monitor depth of anaesthesia
- take heart rate/pulse rate, palpate peripheral pulses, auscultate the heart and lungs, mucous membrane colour & refill
- record respiratory rate, observe the character of the respiration
- note eye position, presence/absence of nystagmus, palpebral reflex (not corneal!)
- check anal tone (use someone else's biro!)
- check muscle relaxation
- insert an arterial catheter into the dorsal pedal artery on the lateral side of the upper limb, below the hock joint. Connect to a pressure transducer for direct arterial blood pressure measurement. Record systolic, mean and diastolic pressures. Respond appropriately to changes in BP.
- Clip three areas on the thorax and place ECG 'dots'. Connect ECG electrodes and carefully monitor.
- Attach a pulse oximeter to the tongue. Interpret results and respond appropriately.
- Attach gas sampling tube to the ET tube. Interpret gas analysis and respond appropriately.

Recoveries can be dangerous to horse and handlers. Make sure everyone is aware of your horse's imminent attempts at standing.

There are some suggested regimens for you to use on the next page.

All horses are to be **weighed** and have their **mouths washed out**.
Clip and prepare the skin over the jugular before placing a catheter.
***Always have a ½ dose of your induction agent available.
***You may wish to give ¼ to ½ dose of xylazine and/or a half dose of acepromazine as your horse's anaesthesia lightens to prevent premature attempts at standing.

Pre-sedation – all horses
0.03 mg/kg Ace
0.1 mg/kg methadone
0.5 mg/kg xylazine

All in one syringe IM
This is useful for fractious horses.

Horses on inhalation agents	**Weight (Kg)**	**Dose**	**Volume**
Horse 1			
Catheter			
Xylazine 0.5 mg/kg			
Guiafenesin to effect (25 – 100 mg/kg)			
Thiopentone 5 mg/kg as a 5% solution			
Intubate			
Isoflurane + oxygen			

Horse 2
Catheter
Romifidine   50 – 80 ug/kg
Diazepam   0.02 – 0.05 mg/kg
Ketamine   2.2 mg/kg
Intubate
Isoflurane + oxygen

Horses on Triple drip
Horse 3
Catheter
Xylazine   0.5 - 1 mg/kg
Diazepam   0.02 -0.05 mg/kg
Ketamine   2.2 mg/kg
Intubate
Triple drip

Horse 4
Catheter
Romifidine   50 – 80 ug/kg IV
Diazepam   0.02 – 0.05 mg/kg IV
Ketamine   2.2mg/kg IV
Intubate
Triple drip

Triple drip
500ml bottle of 10% Guiafenesin
Add: -
1 g ketamine
500 mg xylazine
Administer to effect (Try 1 ml/kg/h)

Appendix 10   EQUINE FIELD ANAESTHESIA

Horses are inherently difficult anaesthetic patients. *The death rate associated with anaesthesia in horses is approximately 1%, excluding colics.* Causes of death include spontaneous cardiopulmonary arrest and euthanasia because of fractures incurred during induction or recovery, muscle or nerve damage incurred by the recumbent horse.

Horses are naturally flight animals *ie* a horses natural reaction to a situation it perceives to be dangerous is to flee. Horses often panic and attempt to escape if they begin to lose their balance. Thus, it is usual to take a horse from being able to stand steadily and unaided to unconsciousness very rapidly. The usual method of anaesthetising small animals is by titration, but this is not the case with horses. The process of rapid induction of anaesthesia is inherently risky as the dose chosen may be too high (or too low) for the individual needs of the patient. Added to this is the fact that a horse is a large animal with a high centre of gravity and it may fall very heavily. Fractures are not uncommon. It is essential to have horses well sedated and with good muscle relaxation when anaesthesia is induced. *Induction of anaesthesia is a high risk time for horse and handlers. Position yourself and the horse carefully.*

Body weight contributes to problems in anaesthetised horses. Firstly, recumbent horses are difficult to manoeuvre. Secondly, compression of muscles and nerves can lead to *muscle necrosis and nerve damage* which may not be manageable and may necessitate euthanasia of the horse (a horse may panic and further damage itself if it does not have full use of its limbs). Muscle necrosis results in the release of myoglobin which can cause renal damage. It is very important to choose a suitable flat area for anaesthesia and to have the anaesthetised horse on a soft surface eg mattress. If the horse is lying on its side, pull the lower fore limb forwards to minimise the chance of radial nerve damage. Choose halters carefully so that there are no knots or buckles that can damage soft tissues. A flat rope halter is best.

A horse's diaphragm slopes caudally and dorsally and the horse has a very high volume gastrointestinal tract in relation to its body weight. Thus, when a horse is placed on its side, the lower lung lobes are compressed and when the horse is on its back, most of its lung tissue is compressed. (Healthy adult horses rarely lie flat on their sides and never lie on their backs.) Once a horse has been anaesthetised, you must assume its ventilation is severely affected. *All anaesthetised horses should be given supplemental oxygen. It is advisable to have the necessary equipment to provide positive pressure ventilation when anaesthetising horses.*
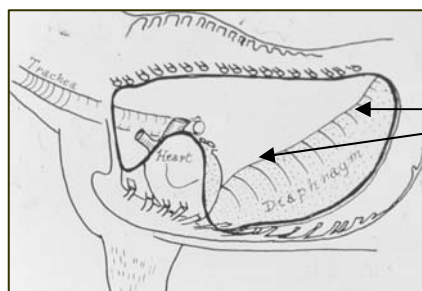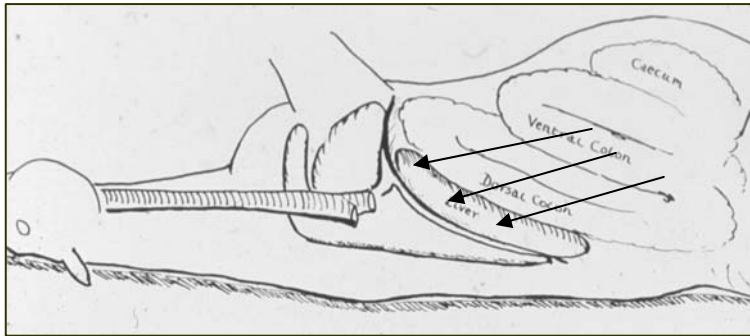

Standing horse.
Note the sloping diaphragm.
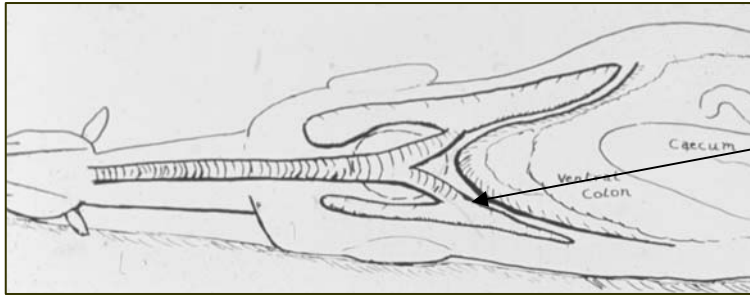
Horse lying on its side. Note compressed lower lung lobes.

Horse lying on its back. Note the abdominal contents compressing the contents of the thorax.

It is important to make sure that the eyes of an anaesthetised horse are not damaged or allowed to dry out. Avoid dragging the horses head along the ground and apply sterile ophthalmic lubricant to the corneas regularly.

The safety of personnel is of utmost importance when dealing with anaesthetised horses. Always stay behind the horses back if possible, don't stand between its legs.

Signs of depth of anaesthesia.

The *pulse* should be monitored for vessel tone, amplitude of pulse wave, rate (beats/minute) and rhythm at least every 5 minutes. Pulses can be felt on the horses face and on the lower limbs. Rates vary, but usually lie between 25 and 40 beats/min.

Mucous membrane colour and rate of refill should be monitored regularly. Observe gum colour before firmly pressing the mucosa with a finger to blanch the gums. Note the time for the colour to return. If the refill time is more than 2 seconds, you should be concerned about the adequacy of peripheral perfusion. Blue means *serious* lack of oxygen in the peripheral blood – **GIVE OXYGEN.**

Where possible, *blood pressure* should be measured. Mean blood pressure should be maintained at 70 mm Hg or higher. Low blood pressure must be treated. (Decrease delivery of anaesthetic drugs, give IV fluids, use positive inotropes eg dobutamine. Dobutamine is frequently administered as an infusion for the maintenance of blood pressure.)

Respiratory rate should be counted every 5 minutes and the size and character of respiratory movements noted. Simply counting the respiratory rate and watching chest and bag movements does not represent adequate monitoring of ventilation. Monitoring devices such as pulse oximeters and capnographs are very useful in monitoring anaesthetised patients. Blood gas measurements are also very valuable. Horses often breathe very poorly under anaesthesia and assisted ventilation may be

necessary. Rate of < 6 breathes/min are not uncommon. Rates of < 4 breathes/min almost certainly mean inadequate ventilation. You should begin manual ventilation.

Eye position and movement give some indication of depth of anaesthesia. A lightly anaesthetised horse usually has nystagmus. The eye positions are often asymmetrical and at surgical depth at least one eye is usually rolled forwards. Eyes should be kept closed and moist. An open, central, dry eye means very deep anaesthesia.

The presence/absence of a *palpebral reflex* should be established at intervals. Gently touch the eyelids, taking care not to touch the cornea. The eyelids should slowly close. A brisk response, coupled with brisk nystagmus indicates the horse is light. Absence of palpebral response and nystagmus indicates deep anaesthesia.

The presence/absence of a *response of the anus* to touch gives useful information. Absence of tone indicates deep anaesthesia.

As with all anaesthetised patients, the depth of anaesthesia must be ascertained by gathering all the information possible. No one parameter will give the answer.

Appendix 11   HORSE DOSE RATES

Tranquillizers/Sedatives

Acepromazine   0.02 – 0.05 mg/kg
This means 0.8 – 2 ml of a 10 mg/ml solution for a 400 kg horse. I rarely use more than 2 ml or 20 mg per horse. Onset of action is slow (20 – 30 min). Duration of action is 4 – 6 h.
Acepromazine is a phenothiazine derivative tranquilliser and it is a useful anxiolytic drug in horses.   Side effects include ataxia, vasodilation (and therefore hypotension) and flaccid extrusion of the penis (with potential for damage - avoid acepromazine in breeding stallions).   Acepromazine has some cardioprotective action during anaesthesia (decreases the incidence of dysrhythmias). It provides no analgesia and it is often used in combination with the α_2 – adrenergic agonist drugs or opioids.

Benzodiazepines
Diazepam   0.02 – 0.04 mg/kg
This means 1.6 – 3.2 ml of a 5 mg/ml solution for a 400 kg horse.
Diazepam is used mainly as an adjunct to anaesthesia rather than a stand-alone sedative drug in horses. It causes *ataxia* and is *best avoided* if the anaesthetic is less than 20 minutes from finished.   If diazepam is administered prior to induction of anaesthesia, it should *immediately precede the induction drug (or be mixed in the same syringe with eg ketamine).*
Diazepam produces good sedation in foals at a dose rate of 0.1 – 0.25 mg/kg.
Midazolam   0.01 – 0.05 mg/kg
Midazolam has a faster onset of action than diazepam, although the duration and recovery times are similar.

α_2-adrenergic agonists   These drugs are used for their sedative, muscle relaxatant and analgesic properties. Typically, the horse will adopt a '5-point stance' (4 legs + head almost on the ground) within 5 minutes of IV administration.   Dose dependant ataxia is a side effect of these drugs, but is less pronounced with romifidine than with xylazine or detomidine.   As in small animals, the α_2-adrenergic agonists cause intense vasoconstriction with resulting hypertension and profound bradycardia. This lasts over half an hour.   Once the vasoconstriction and profound bradycardia pass, the horse becomes hypotensive.   GIT motility is reduced, urine output is increased and temperature control is lost.   These drugs can be used in combination with ketamine and glycerol guiacolate ether as an infusion to prolong anaesthesia after an α_2-adrenergic agonist/ketamine induction.   These combinations are known collectively as 'Triple Drip'.

Sedative dose rates: -
Xylazine   *(Rompun®, Xylazil® etc 10 or 100 mg/ml)0.5 – 1 mg/kg IV (20 – 40 min)*
Detomidine   *(Dormosedan® 10 mg/ml)*   *10 – 20 µg/kg IV (60 – 120 min)*
Romifidine   *(Sedivet® 10 mg/ml)*   *50 – 120 µg/kg IV*

Guiafenesin   *25 – 100 mg/kg (usually 10% solution ie 100 mg/ml).*   This means 0.25 – 1 ml/kg eg 100 – 400 ml for a 400 kg horse. *Guiafenesin is extremely irritant and MUST be administered via a correctly placed intravenous catheter.* These large

volumes can be administered by 50 ml syringes as serial bolus doses or via an infusion set. Guiafenesin is a centrally acting muscle relaxant drug (NOT a muscle paralysing drug). It is a useful adjunct to anaesthesia in that it causes sedation and muscle relaxation. Guiafenesin is known to cause haemolysis. This is less likely if the drug is administered slowly by infusion than by bolus doses.

Opioids   Morphine, pethidine, methadone and butorphanol are the most frequently used opioid analgesic drugs in equine practice. Morphine, pethidine and methadone are pure µ-opioid agonists but butorphanol is an agonist/antagonist. This means that butorphanol has a ceiling effect – increasing doses may not give increasing analgesia. Horses can exhibit excitement when administered opioids. Therefore, opioids are usually administered in combination with tranquilisers or sedatives. Morphine, methadone and butorphanol can be administered by IV, IM or SC injection but pethidine MUST NOT be administered IV (histamine release → vasodilation and hypotension). In the main, these drugs are administered by IM injection.

Table 2.   Dose rates for opioid analgesic drugs. *Adapted from Hall, Clarke & Trim, 2001.*

Opioid drug	Dose and route
Morphine	0.05 – 0.1 mg/kg IV or IM
Methadone	0.1 mg/kg IV or IM
Pethidine	1 – 2 mg/kg *IM ONLY*
Buprenorphine	0.004 - 0.006 mg/kg IV or IM
Butorphanol	0.05 – 0.1 mg/kg

Induction agents
Ketamine   2.2 mg/kg IV Ketamine is a cyclohexamine derivative. It causes dissociation between the thalamocortical and limbic systems. This group of drugs has cataleptic, analgesic and anaesthetic properties without hypnosis. Thus, the patient exhibits spontaneous involuntary muscle movement and high muscle tone. Patients usually have their eyes open and have active palpebral, laryngeal and pharyngeal reflexes. Analgesia is extremely good. Patients under ketamine can show spontaneous muscle movement to the extent of convulsions. Therefore, drugs in this group **should always be used in combination with drugs that afford good muscle relaxation**. It is common to use ketamine with the α_2-adrenergic agonists (eg xylazine, detomidine or romifidine) or benzodiazepines (eg diazepam or midazolam). Cardiovascular and respiratory depression are minimal, and, in combination with a drug with suitable muscle relaxing properties, inductions and recoveries are usually smooth. Recovery is largely due to metabolism by the liver and excretion by the kidneys, so, although some cumulation occurs, incremental doses have little effect on recovery times.

Thiopentone   5 – 15 mg/kg IV (dependent on premedication)   Thiopentone is a barbiturate. Used as a sole anaesthetic agent, thiopentone provides a precipitous induction and, at times, a stormy recovery. However, with appropriate premedication, it is used successfully. Thiopentone causes dose related respiratory and cardiovascular depression, but at low doses (in combination with tranquillisers, sedatives and/or opioid

analgesics) these effects are minimal. Thiopentone is highly irritant with the pH of a 2.5% solution being 10.5 – 11.0. Thus, it should always be administered via a correctly placed venous catheter. Any solution injected perivascularly should be diluted with saline or dilute lignocaine hydrochloride solution. *If thiopentone is administered after an α_2-adrenergic agonist, the dose rate of thiopentone is dramatically reduced eg to 5 mg/kg). It must also be noted that α_2-adrenergic agonists cause increased vein to brain circulation time, and loss of consciousness following an α_2-adrenergic agonist/thiopentone induction may take up to 90 secs.*

Propofol   2 mg/kg following premedication with an α_2-adrenergic agonist provides approximately 10 minutes of anaesthesia with horses standing within 30 minutes. Propofol is a phenol derivative. This dose rate constitutes a very large induction volume and slow administration results in poor quality induction. Respiratory depression results in hypoxaemia.

Zoletil (zolazepam & tiletamine) This combination of drugs (a benzodiazepine & a cyclohexamine) administered after premedication with an α_2-adrenergic agonist provides reasonable short term anaesthesia of slightly longer duration than xylazine/ketamine/diazepam.

Alfaxalone   Preliminary trials with alfaxalone in horses have been promising for short duration anaesthetics.

Maintenance
Although anaesthesia can be prolonged with incremental doses of induction drugs, this is associated with increasingly poor recoveries and greater physiological disturbance than a single induction dose of drug.

Ketamine   Anaesthesia can be prolonged by administering a ¼ – ½ of the induction dose as required. Further incremental doses of α_2-adrenergic agonist (eg xylazine 0.2 mg/kg) may be necessary to maintain muscle relaxation and to avoid violent recoveries. It is best not to go beyond one ketamine 'top-up'.

Thiopentone   As with ketamine, administering further increments of thiopentone is associated with poor recoveries. Incremental doses of 1 mg/kg can be given but the total dose should not exceed 11 mg/kg (induction dose usually 6 – 7 mg/kg). Xylazine 0.2 mg/kg administered IV as the horse begins to lighten should improve the quality of recovery.

Triple drip   Ketamine + an α_2-adrenergic agonist added to glycerol guiacolate ether (guaifenesin) has become known as 'Triple Drip'. Whilst this is a useful mix of drugs for prolonging anaesthesia, it is associated with poor physiological stability and poor recoveries if used for long periods of time.

Table 1.   Suggestions for making up "Triple Drip" for maintenance of anaesthesia by infusion.

Drug combinations	Drug concentrations (mg/ml)	Infusion rate
Xylazine Guiafenesin (10 %) Ketamine	1 100 2	Approx 1 ml/kg/h
Detomidine Guiafenesin (10 %) Ketamine	0.01 - 0.02 100 2	Approx 1 ml/kg/h
Romifidine Guiafenesin (10 %) Ketamine	0.06 100 2	Approx 1 ml/kg/h

Inhalation agents   Halothane (MAC 0.88%)   Isoflurane (MAC 1.31%)   Inhalation agents are frequently used for maintenance of anaesthesia in horses. Halothane and isoflurane must be delivered using agent specific precision vaporisers and specifically designed large animal anaesthetic breathing circuits (large animal circle absorber with the vaporiser out of circle or Water's to and Fro with the vaporiser in the fresh gas flow).

Whilst both of these agents cause cardiac and pulmonary depression, isoflurane is associated with better cardiovascular stability than halothane. As isoflurane has a lower blood/gas solubility coefficient that halothane, the recovery from isoflurane is much faster. Whilst it is common to turn the halothane vaporiser off shortly before the end of a procedure, it is wise to leave an isoflurane vaporiser on until the surgery has been completed and the area prepared for the patient to wake up.

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SEDATIVE COMBINATIONS *(*Ref Taylor, PM & Clarke, KW   1999 *Handbook of Equine Anaesthesia* Saunders)

Acepromazine + α₂ agonist

Acepromazine	0.02 – 0.05 mg/kg
Xylazine	0.5 – 0.6 mg/kg
Acepromazine	0.03 mg/kg
Detomidine	0.01 mg/kg
Acepromazine	0.03 mg/kg
Romifidine	0.05 mg/kg

Acepromazine + opioid

Acepromazine	0.02 – 0.05 mg/kg
Butorphanol	0.02 – 0.04 mg/kg
Acepromazine	0.05 – 0.1 mg/kg
Methadone	0.1 mg/kg

α₂ agonist + opioid

Xylazine	0.5 – 1.0 mg/kg
Butorphanol	0.02 mg/kg
Detomidine	0.01 – 0.015 mg/kg
Butorphanol	0.02 – 0.03 mg/kg
Romifidine	0.05 mg/kg
Butorphanol	0.02 – 0.03 mg/kg
Xylazine	0.5 mg/kg
Methadone	0.1 mg/kg
Detomidine	0.01 – 0.015 mg/kg
Methadone	0.1 mg/kg

Acepromazine + opioid + α₂ agonist

Acepromazine	0.04 – 0.06 mg/kg
Butorphanol	0.01 - 0.02 mg/kg
Detomidine	0.01 – 0.015 mg/kg
Acepromazine	0.04 – 0.06 mg/kg
Methadone	0.05 – 0.1 mg/kg
Detomidine	0.01 – 0.015 mg/kg

Appendix 12   EMERGENCY DRUGS FOR HORSE ANAESTHESIA

Adrenaline	0.02 mg/kg 2 ml/100 kg of 1 in 1,000 Repeat if no response.
Atropine	0.01 – 0.02 mg/kg IV, IM This is 1.6 – 3.3 ml/100 kg of 0.6 mg/ml.
Dopamine	0.25 – 5 μg/kg/min
Dobutamine	0.25 – 5 μg/kg/min
Doxapram	0.2 mg/kg This is 1 ml/100 mg of a 20 mg/ml solution.
Ephedrine	0.03 – 0.1 mg/kg, single dose
Furosemide	1 mg/kg This is 2 ml/100 kg of 50 mg/ml.
Glycopyrrolate	0.0001 - 0.005 mg/kg This is 0.5 – 2.5 ml/100kg of a 0.2 mg/ml solution.
Lignocaine	0.5 mg/kg slowly IV This is 2.5 ml/100 kg of a 2% solution. This can be repeated up to a total dose of 1.5 mg/kg
Methyl prednisalone (Soludelta cortef)	5 mg/kg This is 5 $\times$ 100 mg vials/100 kg

Appendix 13   Suggestions for pain management in cats and dogs.

1.   MILD PAIN.
Non-steroidal anti-inflammatory drug's (NSAID's) Indicated where inflammation plays a major role eg post-surgery.

	DRUG	DOSE	DURATION	ROUTE	SPECIES
1.	Meloxicam (Metacam®)	0.2 mg.kg, 1st day then 0.1 mg/kg	24 h	SC/IV or PO (liquid)	Dog
		0.15 mg/kg SC 1st dose then 0.05 mg/kg PO /24 h, max 3 days)			Cat
2.	Ketoprofen (Ketofen®)	2 mg/kg 1st dose then 1 mg/kg	24 h	SC/PO	Dog, Cat

Don't use with other NSAID's. Use with care with geriatric patients. Contraindicated in patients with renal failure.

3.	Carprofen (Rimadyl®)	4.0 mg/kg/day divided doses(4 – 7 days)12 hr then 2.0 mg/kg/day divided doses		IV/SC/PO	Dog
4.	Aspirin	10-20 mg/kg	8 – 12 hr	PO	Dog
		10 mg/kg *single dose only*	48h	PO	Cat
5.	Paracetamol	15-25 mg/kg	8 h	PO	Dogs,

<u>**PARACETAMOL IS TOXIC TO CATS!!**</u>

6.   Tramadol   1 – 5 mg/kg BID – TID Per Os

7.   Gabapentin   3 – 10 mg/kg Per Os

2.   MODERATE PAIN

1.   Buprenorphine (Temgesic®)   0.01 – 0.02 mg/kg   8 – 12 hr   SC, IM, IV   Dog,Cat
        Buprenorphine is an agonist/antagonist and therefore is thought to have a 'ceiling'effect. This means that you cannot
        satisfactorily titrate the dose of the drug against the analgesia it gives. However, recent research indicates that in human
        patients, the ceiling effect occurs with respect to ventilation and not to analgesia (Dahan *et al, 2006)*

2.   Buprenorphine + NSAID (see above)

3.   Tramadol 1.0 - 5.0 mg/kg BID to TID Per Os

4.   Fentanyl patches See Severe pain management P 121.

5.   Butorphanol 0.2 – 0.4 mg/kg   SC/IV DOGS, 0.1 – 0.2 mg/kg SC/IV CATS. (Low doses IV) Effective for 2 – 4 h

6.   Paracetamol + codeine. Use according to the dose rate for paracetamol (above). **NOT CATS**
        Panadeine® (paracetamol 500 mg + codeine 8 mg)
        Panadeine Forte® (paracetamol 500 mg + codeine 30 mg)

7.   Dextropropoxyphene (32.5 mg) + paracetamol (325 mg) (Digesic®, Capadex®) **NOT CATS**
        Dose for dextroproxyphene 0.5 – 1 mg/kg PO 8 hourly.

3.   SEVERE PAIN   Many of the treatments described on this page are suitable for _continuously monitored patients_ _only_
Supplemental oxygen therapy may be indicated for patients administered opioid drugs. Keep naloxone on hand.

1. Incremental doses of methadone or morphine to effect. 0.1 mg/kg IV at 10 minute (morphine) or 5 minute (methadone) intervals until the patient is comfortable. Up to 1 mg/kg.

2. Morphine continuous infusion   0.1 – 0.2 mg/kg/hr   IV   Dog
                                                                            Start with a SC loading dose of 0.3 – 0.5 mg/kg or titrate using IV boluses of 0.1 mg/kg at 5 – 10 min intervals until the patient is comfortable. (Titrated dose reaching 1 mg/kg is not unusual) Adjust the infusion rate as necessary.
             Take care with cats – 0.1 mg/kg SC repeated once if necessary 15 min later, then 0.05 – 0.1 mg/kg/h.

3. Fentanyl continuous infusion   1 – 6 µg/kg/h   IV   Start with loading dose of 2 µg/kg

4.   Epidural analgesia. Morphine and /or local anaesthetic should last 12 – 24 h.

5.   Fentanyl transdermal patch. Where possible, apply the patch the evening before surgery. However, patches applied in early AM should be effective by that evening.in cats   Prepare the shaved skin by washing with water only and drying. Bandage over the patch and label the bandage with details of patch application. Caution – heating pads on patch, fever or inflamed skin can increase release and absorption of fentanyl)
          Patches are available as 12, 25, 50, 75 and 100 µg/h. Apply patches to provide 2 – 4 µg/kg/h depending on patient requirements.
          Patients will require additional pain management until the patch is effective.

6.   Local blocks, bupivacaine infiltration. Don't exceed 2 mg/kg/4 h bupivacaine, 4.5 mg/kg lignocaine.

7.   Ketamine low dose SC or IV infusion. Try 0.2 – 0.5 mg/kg/hr. This requires major dilution. Loading dose required 0.2 – 0.5 mg/kg SC. Should be used in conjunction with an opioid.

8.   Lignocaine IV infusion. Loading dose 2 mg/kg followed by 40 - 80µg/kg/min .

9.   Morphine/lignocaine/ketamine IV infusion   (Morphine 0.24 + lignocaine 0.3 + ketamine 0.06) mg/kg/h .   (Tranquilli _et al,_ 2007)

10. NSAID's in combination with any of the above
Despite our best endeavours, there will be times when rescue opioid medication will be necessary. Eg bolus doses of morphine or methadone.

Appendix 14   Morphine administered as an infusion.

PREPARATION OF A MORPHINE SOLUTION FOR ADMINISTRATION AS A CONTINUOUS INFUSION

Add 4 × 30mg vials morphine to a one litre bag of saline

ie 120mg/1000ml = 0.12mg/ml

Or

Add 2 × 30mg vials of morphine to a 500ml bag of saline

Ie 60mg/500ml = 0.12mg/ml

THE USE OF NALOXONE TO COUNTERACT MORPHINE OVERDOSE.

Naloxone is a pure opioid antagonist and can be used to relieve over-dose symptoms where necessary. Its duration of action is very short, and more than one administration may be necessary if the duration of action of the agent to be reversed is greater than that of naloxone.

Dog & cat   0.04 mg/kg IV, IM, SC

It is possible to carefully titrate naloxone against unwanted side effects to reduce side effects and maintain analgesia.

Narcan® is 0.4 mg/ml of naloxone.
Take 0.1 - 0.25 ml Narcan® and dilute to 10 ml with saline. (For cats & small dogs, 0.1 ml Narcan, large dogs 0.25 ml Narcan.. Dilute further for very small animals.)
Titrate 1 ml/min until undesirable side-effects have subsided.

Monitoring a patient on a continuous rate infusion.

Unless you actually measure plasma levels, you can only be guided by behavioural signs and some measurable clinical variables.

Signs that the morphine plasma levels are too high.

- Hypoventilation ($\downarrow$ respiratory rate, $\downarrow$ respiratory depth, cyanosis, $\downarrow$ oxygen saturation, hypercapnia)
- CNS depression (unresponsiveness, sleep,)
- CNS stimulation (agitation)
- Hypothermia
- Bradycardia
- Hypotension

Signs that the animal is in pain.

- Tachycardia
- Tachypnoea
- Pale mucous membranes (peripheral vasoconstriction)
- Vocalization
- Agitation
- Abnormal posture
- Reluctance to move
- Unresponsiveness

You will notice that some descriptors appear in both lists of signs – assess carefully!!

Patients on morphine continuous rate infusions require careful and constant monitoring.

Appendix 15

Epidural Anaesthesia in the Cat and Dog
Brenda Dixon, Clinical Anaesthetist, School of Veterinary Science, UQ

Epidural anaesthesia' is the term used to describe the introduction of drugs into the epidural space to produce anaesthesia, analgesia or both. It is a safe, simple and highly effective technique for perioperative pain management in suitable candidates. In dogs and cats, it is most easily performed at the lumbo-sacral space.

Various drugs can be used – the most common of these being a narcotic analgesic in saline or local anaesthetic, or a local anaesthetic alone. Local anaesthetics produce both motor and sensory blockade, while the narcotic analgesics produce analgesia by acting on the large number of opioid receptors in the pain tracts in the dorsal horn of the spinal cord.

The cranial spread of the drugs is volume dependent, so strict adherence to dose rates is necessary to prevent excessive cranial spread and thus possible respiratory muscle paralysis when a local anaesthetic is used. Dose rates should be calculated on estimated lean body weight so as not to overdose. Obese dogs have the same size vertebral canals as their lean counterparts and may even have fat deposits occupying the epidural space.

Epidurals are described as low or high volume and the volume is what determines how far cranial the drug solution will reach. A low volume epidural (0.2 mLl/kg) will extend to the mid-lumbar region and a high volume epidural (0.3 mL/kg) will extend to the mid-thoracic region.

Indications for a low volume epidural
Hindlimb procedures
Pelvic procedures
Perineal surgery (including anus)

Indications for high volume epidural
Laparotomy
Spinal surgery
Extensive soft tissue surgery
Thoracotomy (may not be high enough)

The onset and duration of action depend on the pharmacological properties of the chosen drugs. The most commonly used narcotic analgesic is morphine at a dose rate of 0.1 - 0.2mg/kg. Recently, the use of epidural buprenorphine at a dose rate of 0.004 mg/kg has been reported. The local anaesthetics commonly used are bupivacaine (0.5%) and lignocaine (2%). *All drugs to be administered into the epidural space must be **free of preservatives** and must be from single use vials.*

Preparation
♦   Calculated dose, ready in syringe and warmed
♦   Spinal needle (short bevelled needles with a stylette filling the lumen)
1½ " (small – medium cat or dog), 2¾"(medium – large dog) or 3" (very large dog)
♦   Low resistance syringe (traditionally glass)
♦   Area clipped and surgically prepared
♦   Sterile gloves

Technique
♦   Palpate the iliac prominences & the lumbar dorsal spinous processes (L6, L7 & S1). There is a paplable dip at the spot!
♦   Position the animal in 1) sternal recumbency with hind limbs drawn forwards or 2) lateral recumbency, hindlimbs pulled forwards, pelvis 'square' to the table.
♦   Needle perpendicular to the skin at the lumbosacral space
♦   Advance slowly, feel 'pop' as the needle passes through the *ligamentum flavum*.
♦   Remove stylette & wait to ensure no blood or CSF flows out.
♦   Blood means you have hit the central venous plexus – pull out and start again.
♦   CSF means you are about to do a spinal block – use half prepared dose. (can give the same morphine dose)

In most dogs, the meningeal sac ends before the L/S space, so it's unlikely that the needle will be in the CSF. However, in cats, the meningeal sac usually finishes at the sacrum – so sub-arachnoid penetration is more likely.

♦ If no CSF or blood flows, give a small trial injection of air. If there is no resistance to injection (no bounce back of the plunger), proceed with the injection slowly, over 1 minute. Stop if there is resistance to the injection. If there is resistance to the injection of air, advance the needle further. The needle may not have penetrated the *ligamantum flavum*. If you hit bone, 'walk around' to advance the needle. If in doubt, pull out and try again.

If a local anaesthetic is used, the patient must be receiving IV fluids. Increase the flow rate of IV fluids to compensate for the loss of vasomotor tone to the hind quarters and the possible development of hypotension.

Common side effects include: -

1. Change in heart rate (increase or decrease). If this is immediate, then it is usually due to insufficient warming of the drugs, injecting too fast or injecting against resistance. If this is delayed, it is probably due to hypotension as a result of loss of vasomotor tone, and should respond to fluid loading.
2. Motor disturbance can occur for up to 14 hours after injection and is related to residual effect of the local anaesthetic.

Less common side effects include: -

1. Morphine induced pruritis, urinary retention and dysphoria
2. Retarded hair regrowth at the L/S site. This is termed 'follicular arrest'. Hair should regrow within 6 months.

Contraindications: -

1. Uncorrected hypovolaemia
2. Anatomical abnormalities, infection or trauma to the L/S area
3. Skin infections at L/S area

In conclusion, epidural anaesthesia is a useful technique with regards to perioperative pain control. It is well worth the effort to gain the necessary skill to perform this procedure.

Drug	Dose	Onset of action	Duration of action
Morphine (5 mg/ml)	0.1 - 0.2 mg/kg	20 - 30 min	Up to 24 hours
Buprenorphine (0.3 mg/ml)	0.004 mg/kg	20 – 30 min	Up to 24 hours
Lignocaine 2% Not more than 5 mg/kg.	Depends on whether high or low volume. Make up the rest of the volume after narcotic analgesic is added.	0 - 15 min	60 - 120 minutes
Bupivacaine 0.5% Not more than 2 mg/kg		20 - 30 min	4 - 6 hours

Appendix 16
Anaesthetising the Geriatric Patient
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Introduction
The definition of a geriatric patient is one that has reached approximately 75-80 % of their expected lifespan. This as a chronological age will therefore differ for different species, and breeds within that species. An alternative definition states that any dog or cat over 8 years is to be considered geriatric irrespective of breed.
Anaesthetising the geriatric patient can be extremely stressful for the Veterinarian and a high risk procedure for the patient.   Anaesthesia of the geriatric patient can be achieved successfully, but only when one has taken the necessary time to thoroughly evaluate the patient; perform any necessary investigations; have an appreciation for the normal aging physiological process; be aware of any concomitant disease processes; have on hand suitable drugs with a thorough understanding of their clinical usage as well as having the appropriate anaesthesia equipment and monitoring devices available.
Old age in itself is not a contraindication to general anaesthesia, but it will increase the relative risk of the procedure compared with anaesthesia of the young healthy patient and so the risk benefit ratio must be evaluated and considered. There are few absolute contraindications to general anaesthesia and old age is not one of them.
Physiological changes associated with aging
Aging is a progressive physiological process that results in an unavoidable deterioration in organ system function in the absence of any particular disease process. Generally across all organ systems there is a reduction in functional mass resulting in a decrease in the functional reserve of that organ.
Cardiovascular System
There are major progressive age related anatomical changes that are seen in the heart; these include fibrosis of the myocardium, myxomatous valvular degeneration (MVD) potentially leading to valvular incompetence, and ventricular wall hypertrophy. Variable degrees of myocardial fibre atrophy result in a reduced pump ability of the heart and a subsequent decrease in cardiac output. Heart rate may be affected if pacemaker cells are involved. Fibrosis of the endocardium and valves leads to a decrease in compliance. The vascular tree thickens and loses elasticity resulting in an increased resistance to left ventricular output and causing progressive ventricular hypertrophy due to the increased afterload. As this hypertrophy progresses with a resultant decrease in chamber elasticity, the aging heart is more dependent on atrial contraction for diastolic ventricular filling. The maximal chronotrophic (heart rate) response during physiological stress is reduced in the aging heart due to β-receptor attrition and reduced receptor affinity. An increase in vagal tone is also seen as well as a reduction in blood volume.
Myxomatous degeneration of the atrio-ventricular valves (MVD) is very common in the geriatric patient. MVD is both age and breed related, with both old dogs and smaller breeds being the most commonly affected. 93% of dogs between the ages of 9 and 12 are affected when all degrees of severity of MVD are included , increasing to 100% of dogs over the age of 12, 58% of dogs over 9 years have advanced disease. These statistics include large dogs as well as small and we know that the incidence of MVD in large dogs is less (with the Doberman pinscher being the exception) making the prevalence in the smaller breeds even higher.
Collectively we see a reduction in cardiac reserve resulting in a decreased ability to compensate for anaesthesia related cardiovascular changes, making these patients particularly vulnerable to inappropriate drug choices or relative overdoses.   The geriatric animal commonly has concomitant acquired myocardial degenerative disease and this will significantly increase the risk of cardiac arrhythmias under general anaesthesia particularly in the presence of hypoxia. Drugs which increase the risk of arrhythmias e.g. thiopentone,

α2 adronergic agonists (xylazine, medetomidine) and high doses of ketamine are therefore best avoided.

Respiratory System

The aging process in the lung results in progressive reductions in lung elasticity, thoracic wall, intercostal and diaphragmatic muscle strength, chest wall compliance, elastic recoil, protective reflexes and the number of functional alveoli. As the thorax becomes more rigid and less compliant, vital capacity, total lung capacity, respiratory minute volume and tidal volumes are all reduced. Gas trapping occurs due the reduction in lung elasticity and results in an increase in ventilation/perfusion mismatching and thus decreasing the ability for effective gas exchange, particularly oxygenation. This produces a decrease in PaO_2 (arterial oxygen) with age, but accompanying this is a reduction in oxygen consumption.

The net effect of all of these changes is a reduction in respiratory reserve, which becomes particularly important when combined with the depressant effects of opioids and anaesthetic agents. It is therefore important to pre-oxygenate the geriatric patient if possible or at a minimum to provide oxygen by mask during the induction of anaesthesia. Monitoring the haemoglobin oxygenation saturation (SpO_2) by pulse oximetry of a heavily sedated patient would also be prudent, and oxygen provided if the SpO_2 falls below 95%. Due to the loss of lung elastin, lungs are more prone to barotrauma (volutrauma) and so particular care is needed during intermittent positive pressure ventilation (IPPV) to use minimal inspiratory pressures.

Central Nervous System

Aging results in a reduction in cerebral mass due to neuronal degeneration. There is also a reduction in cerebral perfusion and a general depletion of neurotransmitters e.g. dopamine, noradrenalin, tyrosine and serotonin, as well as a decrease in receptor affinity for neurotransmitters. This results in an enhanced effect of sedative and anaesthetic drugs. The MAC (minimum alveolar concentration) of inhalational anaesthetic agents decreases linearly with age as do the requirements of local anaesthetics, opioids, barbiturates and intra-venous induction agents. It is therefore prudent to use lower doses in the geriatric patient.

There is also an increase in sympathetic nervous system activity with an increased sympathetic outflow but a reduction in parasympathetic nervous system activity. A down regulation of all β-receptors is also seen.

Renal

There is a decrease in functional kidney mass due to a reduction in the number of functional nephrons (in humans by the age of 80 years there can be up to a 50% loss of functional nephrons). This results in a reduction in GFR (glomerular filtration rate) directly proportional to the loss of nephrons. There is also a reduced renal blood flow secondary to the decreased cardiac output. The aged kidney is less responsive to ADH (anti-diuretic hormone) and therefore has a reduced ability to conserve Na and concentrate urine. The reduced renal blood flow makes them more susceptible to renal failure in the presence of renal ischaemia e.g. hypotension or hypoxia.

The geriatric patient cannot maximally retain Na or water under conditions of volume depletion. They are therefore less tolerant of hemodynamic insults such as dehydration, hypovolaemia and haemorrhage. Their ability to correct fluid, electrolyte and acid base disturbances is also diminished. They have a reduced ability to excrete an overzealous Na or water load and so excessive fluid loading should be avoided as should fluids high in Na e.g. 0.9% saline, unless specifically indicated or congestive heart failure can result. Hypotension, hypovolaemia, hypoxia and hypercapnia will also exacerbate the reduction in renal blood flow and should also be avoided.

Anaesthetic drugs that are dependent on renal excretion e.g. ketamine may have a longer half life in the geriatric patient and so should be used cautiously and conservatively, and dose and frequency of dosing altered accordingly.

Hepatic

There is a reduction in functional liver mass (again in the human patient a 50% reduction in liver mass can accompany old age), hepatic blood flow and enzyme activity. This can result in a prolonged metabolism and excretion of drugs dependent on hepatic conjugation leading to a more profound and longer duration of effect. Drugs that are therefore primarily liver metabolized should be avoided if possible (difficult as almost all drugs are liver metabolized) or reduced doses with longer dosing intervals instituted. As liver function deteriorates there is also a greater susceptibility towards hypoproteinaemia, impaired clotting and hypoglycaemia.

Body Composition

There is a reduction in skeletal muscle mass and an increase in the lipid fraction of the body with aging. There is also a reduction in total body water, with decreases in intra-cellular water content and plasma volume. Therefore when intra-venous drugs are administered, there is a smaller volume of distribution and resultant higher plasma concentrations than expected if doses are not reduced accordingly. The increased fat content of the body can result in a higher proportion of highly fat soluble drugs redistributed to body fat and a slower complete elimination time from the body. A reduction in serum albumin is seen and will result in an increased unbound fraction of drugs in the plasma thus producing a higher active component of the drug than was expected and therefore a more exaggerated effect. There is also a reduction in their basal metabolic rate which makes the geriatric patient more prone to hypothermia. Shivering can dramatically increase oxygen requirements by 2-3 times and so increases their vulnerability to hypoxia particularly in recovery if oxygen demands are not met.

Assessing the geriatric patient prior to general anaesthesia

A thorough physical examination is essential in the geriatric patient, including careful cardiac auscultation. A discussion with the owner is essential to help identify any specific health issues e.g. exercise intolerance, coughing, increased thirst, reduced/increased appetite, recent weight loss etc, can pinpoint specific areas of concern to direct further investigations. Any abnormalities should be investigated. Serum biochemical analysis, complete blood count and urine analysis is mandatory. If any cardiac abnormalities are noted e.g. murmurs, gallop rhythms, dysrhythmias, exercise intolerance, pulse deficits, coughing etc then a cardiac work-up is essential including, if possible, an echocardiogram performed by a cardiologist (this will be the most useful investigation in the identification of the specific nature of the cardiac disease present) and possibly chest radiology and electrocardiogram (ECG). It is vital to identify the specific cardiac disease present as different cardiac disease/degenerative processes can require quite different anaesthetic regimens, and the relative risk of the procedure will be reduced if the most suitable regimen is employed.

Any significant, pre-existing abnormalities should be identified and corrected if possible prior to general anaesthesia. Hydration state should be assessed and dehydrated or hypovolaemic patients should have volume restored prior to general anaesthesia or severe hypotension is likely to result due to the vasodilating effects of many of the drugs used. This should be done slowly and conservatively in the geriatric patient.

Anaesthetising the geriatric patient

Premedication/sedation

Sedation may not be necessary in quiet or debilitated patients. Sedatives will however alleviate stress and decrease the required doses of anaesthetic drugs. Opioids will also provide pre-emptive analgesia for painful procedures. Opioids are the mainstay for sedating or premedicating the geriatric patient, often providing suitable sedation on their own. They produce minimal cardio-vascular depression and little to no respiratory depression in appropriate doses.

Benzodiazepines (diazepam, midazolam) can be useful sedative/anxiolytics in these patients as they produce little or no cardio-vascular or respiratory depression at clinical dose rates, but are notorious for producing dysphoria. Midazolam administered intravenously at

micro doses can be very useful in providing adequate sedation in very old/ill cats but dysphoria is always a possibility when benzodiazepines are used.

Acepromazine (ACP) due to its α_1-adrenergic receptor blockade can produce vasodilation and will exacerbate hypotension particularly when used in combination with other anaesthetic drugs that also cause vasodilation. Generally it is best to avoid its use in the geriatric patient but micro doses can be useful particularly in very anxious dogs when an opioid on its own has not provided the required sedation. Do not give to hypovolaemic or dehydrated patients. It will also exacerbate hypothermia. It is a poor and unreliable sedative in cats generally and therefore best avoided in the geriatric feline.

Non-steroidal anti-inflammatory drugs (NSAID) are best avoided in the immediate peri operative period in the geriatric patient. It is mandatory before instigating NSAID therapy in these patients to evaluate liver and kidney function. Do not give prior to or during a general anaesthetic as the protective prostaglandin effect in the low perfusion state in the kidney is lost and renal damage in the presence of hypotension will be exacerbated.

Induction of anaesthesia

Always pre-oxygenate the patient if possible. Do not overly stress the patient to achieve this aim however. Always as a minimum provide oxygen during the induction process, remember that an ill-fitting face mask can dramatically increase machine dead-space and result in rebreathing. Providing oxygen will significantly increase the animal's PaO_2 (arterial oxygen partial pressure) and therefore increase the time period before the animal becomes hypoxic with respiratory arrest/depression. If the animal has suspected or known cardiac issues an ECG and a Doppler blood pressure monitor are best applied before induction of anaesthesia so that continuous myocardial electrical activity and systemic blood pressure can be closely monitored during this physiologically vulnerable time.

Thiopentone is best avoided as it is highly arrythmogenic, highly protein bound, its' elimination time will be greatly increased with a failing liver and it has a relatively low therapeutic index (the therapeutic index relates to how "safe" a drug is, referring to the multiples of the normal dose of a drug that will produce death. Hence the lower the number, the more potentially dangerous the drug).

Propofol is a potent vasodilator and profound hypotension may result with its use. A 20% – 40% reduction in blood pressure can be expected with induction doses. It is best to avoid using propofol in the geriatric patient but particularly in those animals that are dehydrated or hypovolaemic. It does however have an alternative route of metabolism other than the liver and so is a very useful drug in animals with acute/overt liver failure or in animals with porto-systemic shunting e.g. severe cirrhosis. It has a very short half-life and is non-cumulative, providing a very 'crisp' wake-up and so can be useful in animals where the maintenance of their airway may be compromised in the early recovery period e.g. brachycephalics or patients with intractable vomition. It is best to limit its use to those patients where the benefits outweigh this risk of profound hypotension. Use minute doses to effect, combining with an opioid such as fentanyl and a benzodiazepine such as midazolam (i.e. balanced anaesthetic technique) can dramatically decrease the required dose and therefore significantly reduce its unwanted side effects. Propofol is also a profound respiratory depressant and severe hypoventilation to the point of apnoea is commonly seen with its use. Be aware of this and be prepared to implement positive pressure ventilation post intubation. All of these unwanted side-effects will be exacerbated by giving large bolus doses and this is therefore best avoided.

Alfaxan®, the newest formulation of alfaxalone solubilised in cyclodextrin provides very good cardio-vascular stability and is non-arrythmogenic. It is often the best choice in the geriatric patient. It is liver metabolised and so should be avoided in liver failure patients. It is a very 'safe' drug with a high therapeutic index (therapeutic index in the rat recorded at 30). It also produces dose related respiratory depression. A patient that is minimally sedated may get neuroexcitatory effects on induction with Alfaxan but this dissipates as the patient reaches a deeper plane of anaesthesia. Hyperaesthesia can be seen in the early recovery period, particularly after a short period of anaesthesia, so it is best to recover these patients in a quiet, unstimulated environment. This is generally not seen with longer general anaesthesia times.

The ketamine/benzodiazepine combination for induction can provide good cardiovascular stability, with patients generally maintaining good blood pressure due to the stimulation of the sympathetic nervous system and catecholamine release. Ketamine's effects on the cardiovascular system include increases in cardiac output, heart rate, systemic blood pressure, pulmonary arterial pressure and central venous pressure. The direct effect on the myocardium is depression however and care should be avoided in animals with high existing sympathetic tone, severe heart disease, or systemic or pulmonary hypertension. The benzodiazepines produce dose related respiratory depression and are liver metabolised. Midazolam makes a much better choice than diazepam as it has a significantly shorter half life. Ketamine is renally excreted and the dose needs to be reduced in kidney failure patients. Ketamine should also be avoided in the patient with intra-cranial disease, head trauma, seizuring, and increased intra-ocular pressure or suspected raised intra-cranial pressure as it can induce seizuring and can raise intra-cranial pressure.

Maintenance of anaesthesia
The inhalant anaesthetic agents are easily titrated. Halothane is best avoided due to the sensitization of the myocardium to circulating catecholamines. Isoflurane or sevoflurane make good choices. All inhalant anaesthetics produce dose related cardio-vascular and respiratory depression and so the minimum dose possible should be delivered. Combining with an opioid e.g. fentanyl infusion, will not only provide analgesia but also allows for a significant reduction in the required inhalant, helping to preserve cardiovascular function. This will however potentially exacerbate respiratory depression and so it is imperative to be able to measure the animal's adequacy of ventilation i.e. capnography, and IPPV instituted if required. Local anaesthetic techniques such as regional blocks, epidural anaesthesia etc, are also excellent techniques to employ to provide analgesia and significantly reduce the required inhalant. Isoflurane is probably the most popular choice for the geriatric. It is virtually non-metabolised being almost completely excreted unchanged via the lungs. It has relatively low blood solubility and consequently is rapidly excreted on cessation of delivery, producing rapid recoveries, particularly important in the airway-compromised patient. Isoflurane produces minimal myocardial depression; hypotension may be seen but is due to vasodilation and is dose related. It is indicated in animals with renal or hepatic disease. Arrhythmias are rarely seen as there is minimal sensitisation of the myocardium to circulating catecholamines and so it also makes a good choice for animals with cardiac disease. Sevoflurane and desflurane are expensive and have no real significant advantage over Isoflurane to warrant the extra cost (in the author's opinion).

Drug doses

	Dogs	Cats
Acepromazine mg/kg	0.01-0.02 S/C 0.005 – 0.01 IV	Acp is not an effective sedative-
Midazolam mg/kg	0.1 – 0.5 S/C, IV	0.1 – 0.5 S/C, IV
Diazepam mg/kg	0.1 – 0.5 S/C, IV	0.1 – 0.5 S/C, IV
Ketamine mg/kg	- -	2 - 5 S/C for sedation 1 – 2 IV for sedation
Methadone mg/kg	0.5 S/C 0.1 – 0.2 IV	0.3 – 0.5 S/C 0.05 – 0.1 IV
Butorphanol mg/kg	0.1 – 0.2 S/C	0.1 – 0.2 S/C
Hydromorphone mg/kg	0.1 – 0.2 S/C	0.1 – 0.2 S/C
Buprenorphine mg/kg	0.01 – 0.02 S/C	0.01-0.02 S/C
Fentanyl µg/kg	2 – 5 IV	2 -5 IV

Fentanyl CRI: 4 – 10 µg/kg/hr (capnography essential as respiratory depression is likely with the higher doses)
Propofol mg/kg   0.5 mg/kg incremental doses IV to effect for induction (may use up to 4 – 8 mg/kg total dose). (Care with use, best to combine with fentanyl and midazolam as this will substantially reduce the required dose of Propofol and therefore reduce the potential hypotension, see text)
Alfaxan® mg/kg   0.25 mg/kg (dogs), 0.5 mg/kg (cats) incremental doses to effect IV for induction (may use up to 1 -2 mg/kg total dose in dogs and 3 – 5 mg/kg in cats).

Ketamine/benzodiazepine   Give up to 0.5 mg/kg of midazolam or diazepam IV and then titrate ketamine at 1 – 2mg/kg incrementally IV to effect for induction (may use up to 10 mg/kg). Will not be suitable for all patients, see text.

Dopamine   5 – 20 µg/kg/minute for blood pressure support.

Phenylephrine   1 -3 µg/kg/minute for blood pressure support.

S/C = sub-cutaneous   IV =intra-venous   CRI = constant rate infusion

Fluid therapy and general support

Aggressive fluid therapy and fluid overloading should be avoided if possible in the geriatric patient. Even in the hypotensive, dehydrated or hypovolaemic patient fluids should be administered more judiciously than in the young healthy patient. A balanced electrolyte replacement fluid is best used over 0.9% NaCl to minimise the salt load administered. Peri-operative fluids should be specifically targeted at correcting specific deficits and maintaining adequate perfusion without delivering excessive electrolyte or fluid loads. If hypotension occurs under general anaesthetic conservative fluid loading may be instituted but drug therapy should be considered early in the treatment protocol to prevent excessive fluid administration. Some cardiac conditions will not tolerate any fluid loading as pulmonary oedema can result (see specific conditions).

 The geriatric patient is highly susceptible to hypothermia and so particular diligence in this area is required. Placing the animal in a warm environment from the time of premedication, using warm water beds, warm water bottles, warm air blankets, warming intravenous fluids, utilising in-circle humidifiers etc can help minimise the heat loss associated with heavy sedation/general anaesthesia. Be careful however as animals are more susceptible to thermal burns under general anaesthetic; wrap all warming apparatus to avoid direct skin contact.

Oxygen therapy is always advantageous in the geriatric patient. Preoxygenation is especially important for the respiratory or cardiac compromised patient. All animals should receive oxygen during the induction of anaesthesia, an appropriate sized face mask is the easiest way to achieve this, and oxygen may also be required in the recovery period. Nasal oxygen lines (small feeding tubes) can be placed intra-nasally and secured carefully with Supa-glue® before wake-up and provide an excellent non-invasive way to provide oxygen insufflation to the recovering patient, two can be placed if necessary. Ensure that the animal can maintain adequate haemoglobin oxygen saturation (SpO_2 of 95% or above) on room air before discontinuing oxygen therapy. Pulse oximetry should also be utilised in the heavily sedated patient to evaluate their need for oxygen therapy. Hypoxia is best avoided and can precipitate cardiac arrhythmias and even cardiac arrest if severe.

It is imperative in the geriatric patient to be extra diligent with dosing calculations. Weigh the patient, always do the maths, use smaller doses than would be used in young healthy patients. Always titrate, and leave longer times between induction dosing increments allowing for prolonged circulation times due to the lower cardiac output. A carefully chosen anaesthetic protocol, meticulous physiologic support and vigilant monitoring are required for a successful outcome.

Monitoring

Vigilant monitoring is mandatory in the geriatric patient. Minimum monitoring should include pulse oximetry, blood pressure and capnography. An ECG is also essential in the patient with cardiac disease or one undergoing major surgery. Urinary catheterisation is also useful in the patient with renal compromise or one that is having major surgery as urine output can be monitored allowing for early recognition and therapy for low/no output states.

 In human mortality studies, 30% of deaths associated with general anaesthesia were attributable to inappropriate use of monitors, specifically pulse oximetry and capnography. This has led to the compulsory use of these monitors in all human patients undergoing general anaesthesia. The ACVA (American College of Veterinary Anaesthetists) is now recommending that the minimum monitoring for all anaesthetised small animal patients should include blood pressure, SpO_2, $ETCO_2$ and an ECG. It has already become mandatory in some American states. Most of the multi-function monitors now available will perform all these monitoring tasks and are well worth the investment.

Approach to anaesthesia for some common disorders seen in the geriatric patient
1.   *Cat with hypertrophic cardiomyopathy (HCM)*
HCM is a disease of diastole; the hypertrophied ventricular walls interfere with adequate filling during diastole but also can cause a left ventricular outflow tract obstruction during systole. The aims in these patients are to provide adequate pre-load, produce a mild reduction in myocardial contractility and to maintain afterload. The left ventricular outflow obstruction will be increased if there is an increase in myocardial contractility (as can occur with the administration of ketamine or positive inotropes) ; if there is a reduction in preload (hypovolaemia); or if there is a decrease in afterload (hypotension and hypovolaemia). In the presence of a decreased afterload an animal with HCM can also develop systolic anterior motion of the mitral valve leading to valvular incompetence predisposing to pulmonary oedema. The cat with HCM may not tolerate any fluid loading as left sided congestive heart failure may result. If there is a contributing disease of hyperthyroidism then it is best to stabilise the patient on appropriate medical therapy for a minimum of 2 weeks prior to general anaesthesia. The cardiac disease from hyperthyroidism is somewhat reversible.
Example of a suitable protocol:
Pre-medication: Methadone or hydromorphone $\pm$ midazolam. No ketamine as it can increase inotropy by sympathetic stimulation and no acepromazine as it can reduce afterload by vasodilation. If it is a fractious cat, an α_2 adrenergic agonist may be given in micro doses (i.e. medetomidine 1 – 5 µg/kg IM) as it will give reasonable behavioural control and aid in the maintenance of blood pressure and afterload. Arrhythmias are very unlikely with these small doses.
Induction: Preoxygenate with oxygen if possible, Alfaxan® very slowly to effect.
Maintenance: Isoflurane plus fentanyl infusion if longer procedure. No ketamine.
Fluids: No fluids for short procedure. Conservative maintenance fluids for lengthy procedure.
Blood pressure support: Phenylephrine infusion.
Monitoring: SpO$_2$, Doppler, capnography, and ECG.

2.   *Animal with renal insufficiency/ chronic renal failure.*
A thorough assessment of the patient is absolutely essential prior to anaesthesia, including clinical exam, blood work (serum biochemical analysis and haematology) and urine analysis. Any significant abnormalities should be corrected, including packed cell volume, hydration status and electrolyte abnormalities, particularly hyperkalaemia. Ideally an animal should not be anaesthetised with a packed cell volume (PCV) less than 20; splenic engorgement is a common anaesthesia drug effect and this can result in a further reduction in the PCV and this can therefore impact significantly on the oxygen carrying capacity of the blood. A blood transfusion should be given in this circumstance prior to the procedure. Ideally an animal with renal insufficiency/chronic renal failure should be placed on fluids 12 hours prior to anaesthesia, but 4 – 6 hours at a minimum. Fluid rates need to be conservative.
 The aim for these patients is to preserve blood pressure and renal perfusion. The maintenance of renal blood flow and the avoidance of hypovolaemia, hypotension and renal vasoconstriction are necessary for the successful management of these patients. If there is a reduction in blood pressure and/or renal perfusion then renal shutdown can occur and acute renal failure may result. Monitoring of urine output in the early recovery period is ideal in these patients so early intervention can be instituted in low or no output urine states. It is also important that the animal is not discontinued from fluids until it is drinking and eating sufficiently well to avoid dehydration and decompensation at home. It is also essential to ensure that the animal has appropriate analgesia without excessive sedation. Pain and over sedation can both decrease food and water intake.
Example of a suitable protocol:

Premedication: Opioid ± midazolam. Ketamine in small doses for fractious cats would be appropriate.
Induction: Alfaxan® to effect.
Maintenance: Isoflurane, fentanyl infusion if major surgery.
Fluids: Twice maintenance rate during anaesthesia.
Blood pressure support: Conservative fluid boluses can be given. A dopamine infusion can be instigated if necessary, higher end doses however can decrease renal perfusion by causing renal vasoconstriction.
Monitoring: SpO_2, capnography, and Doppler. It would be ideal to place an indwelling urinary catheter for urine output monitoring. Direct blood pressure measurement would be preferable for a long general anaesthetic or a major surgical procedure.

3.   Diabetic animal
Unless it is an emergency is best not to anaesthetise the diabetic patient unless it is well controlled. It is best to delay elective procedures until this aim has been achieved. All patients need to be starved the morning of surgery. If the patient is on an insulin glargine treatment protocol then the morning dose can be missed and insulin treatment instigated as soon as the animal is awake and eating again. If the animal is on caninsulin then a half dose can be administered the morning of surgery and frequent blood glucose monitoring performed during the course of the day. The aims for the diabetic patient are to prevent hypoglycaemia, to achieve a fast return to their normal feeding/insulin routine and to get them home as soon as possible. The stress of hospitalisation may interfere with diabetic control. Diabetic cats are often very old with concomitant renal insufficiency, so the principles of anaesthetising the animal with renal insufficiency will also apply.
Example of a suitable protocol:
Premedication: Opioid ± midazolam, low dose ketamine would be appropriate for the fractious cat.
Induction: Ketamine/benzodiazepine or Alfaxan® slowly to effect.
Maintenance: Isoflurane, add in fentanyl for major procedures.
Fluids: Twice maintenance, 5 % glucose fluids can be administered if the animal has had insulin that morning (do not administer at more than maintenance rate).
Blood pressure support: Some fluid loading would be appropriate, dopamine infusion.
Monitoring: SpO_2, capnograph, Doppler, frequent blood glucose monitoring particularly during the peak effect of the insulin.

4.   Dog with mitral regurgitation.
A thorough cardiac assessment is essential in the dog with suspected mitral regurgitation, including if possible an echocardiogram by a cardiologist, to assess the severity of the disease process. A chest radiograph is useful to assess the size of the left atrium and to detect the presence of any pulmonary oedema. If pulmonary oedema is present it is best to delay an elective procedure until diuretic therapy can be instituted and there is resolution of the pulmonary oedema.
 The aims for the patient with mitral regurgitation are the avoidance of fluid overloading, the prevention of any exacerbation of the regurgitant fraction and the maintenance of heart rate and myocardial contractility. Fluid loading can result in pulmonary oedema. The regurgitant fraction may be increased if there is a significant increase in afterload. The animal with a very large left atrium and pulmonary vasculature congestion as seen on chest radiographs is obviously at acute risk of developing left-sided congestive heart failure. Any animal with mitral regurgitation however, irrespective of where they are in the compensatory process is also at risk of developing pulmonary oedema with any degree of fluid loading and blood pressure support/treatment should be by early instigation of appropriate drug therapy and not fluid loading.
Example of a suitable protocol:
Premedication: Opioid ± midazolam.
Induction: Preoxygenate if possible. A balanced induction technique using small doses of fentanyl , midazolam and Propofol would be appropriate (e.g. fentanyl 3ug/kg, midazolam

0.2 mg/kg and then propofol to effect, it would be expected that only a very small total dose of propofol would be needed to intubate i.e. total dose of 1 mg/kg), or Alfaxan® to effect.
Maintenance: Isoflurane, fentanyl for major procedure.
Fluids: No fluids if short procedure, conservative maintenance fluids if major procedure.
Blood pressure support: No fluid loading, dopamine infusion.
Monitoring: SpO_2, capnography, Doppler and ECG. Direct blood pressure measurement would be preferable for prolonged general anaesthesia or for a major surgical procedure.

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