



**Association of Veterinary Anaesthetists**
**&**
**European College of Veterinary Anaesthetists**

# Training Day

**29th May 2002**

# Pre-anaesthetic Assessment of the High Risk Patient

**University College Dublin**
**Ireland**

# Timetable: Wednesday 29th May 2002

***AVA / ECVA Trainee day***

***Pre-anaesthetic assessment of the high risk patient***

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|---|---|---|
| *08.45 - 09.15* | *Registration, University Industry Centre, University College Dublin, Belfield, Dublin* | |
| **09.15 - 10.00** | **Hester McAllister** | **Imaging the abdomen** (page 1) |
| **10.00 - 10.45** | **Steve Haskins** | **Arterial blood gas analysis** (page 13) |
| *10.45 - 11.10* | *Tea / Coffee* | |
| **11.10 - 11.55** | **Dan Holden** | **Clinical pathology** (page 21) |
| **11.55 - 12.40** | **Eddie Clutton** | **E.C.Gs** (page 37) |
| *12.40 - 14.05* | *Lunch, Main Restaurant, 1st floor* | |
| **14.00 - 15.00** | **Hester McAllister** | **Imaging the thorax** (page 42) |
| *15.00 - 15.30* | *Tea / Coffee / Commercial exhibition* | |
| **15.30 - 16.30** | **All speakers** | **Case discussions** |

| | | |
|---|---|---|
| **19.00 - 22.00** | **Social gathering** | **Belfield Bar, Montrose Hotel** |
| | **(All delegates. Light supper 20.00. Free)** | |

# ABDOMINAL IMAGING

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## RADIOGRAPHY

Abdominal radiography requires at least one recumbent lateral and a ventro-dorsal study. Usually sedation and restraint using sandbags is all that is required. The sedative chosen should cause no increase in the size of the spleen or cause GIT distension and the animal should be rendered relaxed. Some sedative combinations are contra-indicated for GIT studies and others do not ensure adequate muscle relaxation.

### Lateral View

True lateral positioning requires pads placed under the sternum and pads placed between the stifles to avoid pelvic tilt. The centring point is in the mid abdomen halfway between the top of the last rib and the iliac crest. In large dogs two views may be required, one centred on the cranial abdomen and one caudally. The largest cassettes necessary to include the whole anatomical area should be used.

Occasionally both right and left lateral recumbent studies are useful to evaluate the movement of gas or ingesta in the stomach or intestine.

### Ventro dorsal view

The animal is placed in dorsal recumbency in a symmetrical position in a foam or radiolucent trough. The hind legs are drawn caudally and the centring point is at the umbilicus.

Contrast procedures are usually elective and careful preparation is required in order to obtain as much diagnostic information as possible and to ensure the removal of any extraneous faecal or surface material which may confuse the final assessment. Where large volumes of abdominal fluid are suspected little information can be gained from radiography.

### Dorso-ventral study

This position is sometimes used during contrast studies of the stomach and when several dyspnoeic or distressed animals are being radiographed.

**Lateral decubitus view**

The animal is in **left** lateral recumbency and the x-ray beam is directed horizontally and centred at the umbilicus. The cassette is placed along the dorsum of the back. Particular care must be taken in regard to radiation safety. Its use is generally restricted to those cases where free intra-abdominal gas/air is suspected and requires confirmation. The gas rises and lies under the right cranial abdomen.

**Ultrasonographic Technique**

3.5-7.5MHz transducer depending on the dogs size. Clip closely and apply an acoustic coupling gel. Spirit may be useful to clear greasy or dirty skin. The animal can be imaged in lateral or dorsal recumbency or if necessary in the standing position. May have to image from the recumbent or dependent aspect in order to avoid gas or locate small volumes of fluid.. Very useful for ultrasound guided aspiration of the fluid from a specific or localised site

## Radiological evaluation

Evaluate the quality of the film.

Review the abdominal structures for changes in size, contour, opacity, outline and position.

The presence of intra-abdominal fat improves the radiographic contrast and permits the viewer to identify the various organs.

Should be able to identify the liver, spleen, kidneys (50%), bladder (if full of urine), intra-abdominal prostate, stomach, small intestine, caecum and colon.

Poor abdominal contrast is a feature of young animals and athletic dogs. Pathological causes of poor contrast are the presence of intra-abdominal fluid and peritonitis.

**Intra-abdominal fluid/ascites**

Blood
Urine
Bile
Transudate
Exudate

Depending on the volume of fluid present some or all of the following may be seen

- There is a lack of contrast in the abdomen.
- Homogenous soft tissue/fluid opacity throughout the abdomen
- Poor serosal definition of the intestines
- Inability to identify the margins of the liver , spleen or bladder.
- The intestinal gas shadows lie in the centre of the abdominal cavity
- Pendulous abdominal wall margins
- Small volumes of fluid give an indistinct appearance to the centre of the abdomen .

**Peritoneal fluid Ultrasonography**

Ultrasonography is the diagnostic imaging method of choice
Small volumes of fluid can be identified Ultrasound does not characterise the fluid type. Abdominocentesis is necessary for the definitive diagnosis as to the type of fluid will require cytological analysis.

**Peritonitis**

Secondary to trauma to the abdominal wall or rupture of an abdominal organ. Secondary to pancreatitis or pancreatic neoplasia. Seen with free urine or bile in the abdomen

**Radiography**

- A localised loss of serosal detail in the abdomen.
- Granular or patchy increase in opacity.
- In the later stages abdominal fluid may accumulate and the patchiness may no longer be appreciated.
- Abdominal distension is occasionally seen.
- Free intra-abdominal air
- Static intestinal contents. Regional ileus at the site of the perforation
- Build-up of intestinal contents proximal to the perforation site

**Ultrasonographic findings**

- Anechoic areas outlining the serosal surfaces of abdominal structures
- May see a swirling-snowy appearance
- Fibrin tags may be identified
- May be localised or generalised

**Bile peritonitis**
Ultrasonography

- The gall bladder may or may not be seen

**Peritoneal haemorrhage**
Ruptured spleen, ruptured liver, avulsed blood vessel, erosion of a vital structure by a neoplasm
**Neoplasia-carcinomatosis, melanoma**
The intra-abdominal fluid may be due to a ruptured neoplasm or neoplastic seeding.

**Pancreatitis**:-ultrasonography

- Regional ileus in proximal duodenum
- Hypoechoic or anechoic fluid in the immediate vicinity of the duodenum
- Swollen tissue medial to the duodenum

**Retroperitoneal fluid**
Haemorrhage or urine
Radiography

- Poor retroperitoneal detail
- Ill defined renal shadows
- Displaced renal silhouette
- Displaced colon

**Intra-abdominal free gas**
As a result of abdominal wall trauma, intestinal perforation or post-laparotomy. Left lateral decubitus view may be helpful.
**Radiography**

- separation of the diaphragm from the liver.
- gas shadows identified surrounding and enhancing the renal silhouette
- unusual gas patterns -bubbles and streaks- scattered throughout the abdomen and not associated with the gastro-intestinal tract.

**Ultrasonography**
Difficult to appreciate. Scan the upper or non-dependent region and may see a reflective hyperechoic area. If fluid is present as well, may see disseminated hyperechoic speckles

**Hernias**
Ventral hernia: abdominal structures lying outside of the abdominal cavity and under the skin.

Inguinal hernia: may contain fat, bladder, uterus or intestine

Perineal hernia: bladder absent in caudal abdomen. Contrast cystography may be useful

Hiatal hernia: protrusion of part of the stomach through the oesophageal hiatus of the diaphragm.

**Liver**
Normally lies within the costal arch. Seen as a sharp triangular opacity peeking out beyond the last rib margin. Cranial boundary is the diaphragm and caudally it is bordered by the stomach. Any change in size or position of the liver is often signalled by changes in the position or outline of the stomach or diaphragm.

**Anatomical variations**
- Deep chested dogs Afghan: the liver is usually within the costal arch.
- Barrel chested dogs-basset, staff bull terrier; the liver tends to lie beyond the costal margin.
- Obesity: on the right lateral study - the ventral lobes may project caudally
- Barium may be useful when localisation of the stomach is proving difficult.

**Hepatomegaly**- some causes include. neoplasia, right heart failure, metabolic disease-Cushings

- Hepatic silhouette lies caudal to the costal arch.
- The liver margins are rounded or nodular.
- The gastric axis is displaced caudally.
- A localised hepatic mass may displace part of the stomach e.g. pylorus or fundus.
- Barium may be useful when localisation of the stomach is proving difficult.

**Reduced liver size**
May be associated with Congenital port-systemic shunts, cirrhosis and diaphragmatic hernias.
Radiography

- Inability to identify the ventro-caudal liver margin
- Gastric axis displaced cranially

**Porto-systemic shunts**: congenital or acquired

- small hepatic silhouette

Contrast radiography has been used to confirm the clinical diagnosis.

**Portal venography**: requires general anaesthesia and laparotomy.

1. Introduce cannula into a jejunal mesenteric vein.
   5-10 cc of a water soluble contrast agent and take the radiograph towards the end of the injection.

2. Inject contrast directly into a small splenic vein or directly into the splenic pulp (spleno-portography) This procedure may be carried out percutaneously. This techniques disadvantage is that it may not identify a shunt which is located caudal to this site.

Normally see the outline of the portal vein and its branches in the liver.
Abnormal : contrast is shunted directly into the caudal vena cava or azygous veins and other less common venous systems. Shunts may be intrahepatic (ductus venous) or extrahepatic. If the shunt is cranial to the body of T13 it is likely to be intrahepatic.

Many shunts can be diagnosed using ultrasonography when the abnormal vessel may be identified and if Doppler is available the abnormal flow be also be noted. As the liver is small it is often necessary to have the animal sedated and occasionally anaesthetised.
Recently nuclear scintigraphy has become the gold standard technique for PSS diagnosis. Usually sedation is all that is required for this technique.

**Choleliths**: occasionally identified as a radio-opacity in the cranio ventral hepatic region. Many choleliths are radiolucent.

**Dystrophic calcification** is occasionally seen in the liver

**Spleen**

Radiography

Ventro-dorsal view: usually identify the splenic head in the left cranial abdomen, lateral and caudal to the stomach.

Right lateral recumbent view: splenic tail is seen as a soft tissue triangle on the mid-ventral abdomen caudo-ventral to the pylorus and caudal to the stomach

Left lateral recumbent view: the spleen is not consistently seen.

Ultrasonography

The splenic echotexture is hyperechoic when compared with the liver and kidneys. It has a granular appearance and only a few vessels are identified.

**Splenomegaly**: common and may not be of any clinical significance.

May be caused by anaesthesia, anaemia, neoplasia, haematomas, cysts or abscesses and in association with gastric torsion

- Soft tissue mass mid-ventral abdomen.

**Splenic rupture**

- Poor serosal definition locally due to haemorrhage or metastatic seeding

Ultrasonography

- Echogenic fluid oscillating in the abdomen
- Fibrin tags on serosal surfaces
- Disruption of the normal splenic outline

**Splenic Torsion**: uncommon

- Spleen seen on the left lateral aspect of the abdomen on the V-D view. Ltrasonographic examination demonstrates the so called 'starry sky' appearance' Thrombosis of the splenic vessels is also seen.

## Urinary Tract

**Cystography** usually is an elective procedure and an enema should be given one hour prior to the procedure. Deep sedation is usually required. Plain studies should be taken prior to the contrast procedure, to establish the exposure technique and to ensure the contrast is necessary and that the enema has worked. Occasionally GA may be necessary.

Positive contrast cystography is the technique of choice particularly when bladder or urethral rupture is suspected.

**Procedure:** Left lateral recumbency. Withdraw the catheter to the bladder neck in the female or to the level of the tuber ischium in the male dog. Catheterise the bladder and remove the urine, a three-way valve is useful.

**Contrast:** Iodinated contrast diluted to 20% , e.g. Hypaque 75. Low osmolar non-ionic contrast agents such as Iohexol or Iopamidol may also be used but they are more expensive.
Dilute the contrast to 10% w/v with normal saline and introduce the contrast slowly. Depending on the size of animal 30 ml (small dog or cat) to 300 ml (giant dog) is the range of volume which may be safely introduced. The bladder should be palpated per abdomen until distended. No resistance to the contrast should be felt, if any back-pressure is appreciated the procedure should be immediately stopped and the animal rolled 360°, take ventro-dorsal and lateral radiographs. This is the diagnostic method of choice for evaluating the bladder and urethral wall integrity or prostatic and urethral reflux.

**Double Contrast Cystography** is particularly useful when intraluminal filling defects are suspected.

**Procedure:** Left lateral recumbency. Catheterise the bladder and remove the urine. Introduce approximately 5-15 ml of contrast 20% w/v. Then inflate the bladder with 50 to 200 ml room air, depending on the animal's size or stop once back-pressure is felt on the syringe. Great care must be taken not to create air bubbles as these produce strange artefacts. Roll the animal 360° and take ventro-dorsal and lateral radiographs.

**Negative Contrast Cystography**: Pneumocystograpy is only of used when one is trying to localise or differentiate the bladder from abdominal masses in the caudal abdomen. Recently traumatised animals or animals with cystitis or prostatitis should not have negative cystography as fatal air embolism has been described.

**Procedure:** Left lateral recumbency. Catheterise the bladder remove the urine. Introduce 50 to 150 ml of air slowly or until back pressure is felt on the syringe. Take immediate studies.

**Ultrasonography of the Bladder**
7.5MHz transducer
Ideally the bladder should be full
Avoid prior urinary catheterisation
The bladder wall layers may be identified with high resolution transducers
May need to inflate the bladder with saline
**Artefacts**
Slice thickness- simulates sediment
Acoustic enhancement in the far tissues
Reverberation –from the intestine or free intra-luminal air

**Cystic calculi**

- Hyperechoic areas moving within the bladder lumen
- Marked acoustic shadowing

**Cystitis**
The bladder may be difficult to image as it is usually small if the animal is painful. Check the prostate carefully.

- The urine is usually echogenic
- Hyperechoic floccules oscillate within it
- The bladder wall may be thickened
- Discrete polypoid structures projecting from the mucosa
- Irregular contour to the mucosa

**Uroperitoneum:** Radiology is the imaging method of choice
Rupture of the bladder
Rupture of the ureters within the peritoneal cavity
Rupture of the urethra
Bladder rupture
Ultrasonography is not definitively diagnostic

**Bladder rupture**
Contrast will leak into the abdominal cavity. But must take the radiograph as quickly as possible If the intrapelvic urethra is ruptured the contrast will leak into the periurethral tissues

**Intraluminal haemorrhage**

Ultrasonography

- The anechoic urine becomes hypoechoic
- Snow storm effect in real time
- Clots may form hypoechoic masses
- Clots free in the lumen will move with ballottement
- Clots may be attached to the mucosal wall

**Bladder Neoplasia**

- Hypoechoic mass attached to the bladder wall
- Usually base wide
- Normal bladder wall layers are disrupted
- Usually localised at the bladder neck

**Intravenous Urography**

**Procedure:**-Intravenous catheter - wide bore. Ideally an enema two hours prior to procedure is preferable when the study is elective, in emergency cases this may not be possible. Plain studies to ensure the abdomen is clear.

General anaesthesia is required. Place the animal in dorsal recumbency and restrain using sandbags and ties.

**Low volume - rapid infusion technique.**

The contrast agent is preferably a higher percentage 60-70% or 420 mg/$I_2$ per ml. Dosage is 420 mg/lb bw to a maximum of 32 gm total. The contrast is heated to body temperature to improve its viscosity.

Suitable medicaments should be available in case of anaphylactic reactions.

The contrast is injected rapidly and studies are taken at 0, 1, 5, 10, 15 and 20 minutes. If both kidneys and ureters are outlined, left and right lateral studies should be taken.

**High Volume - Drip Infusion:** Contrast is made up in a dilute solution - 150 mg $I_2$/ml - commercially available. Total dose is 1200 mg $I_2$ per kg bw infused over 10-15 minutes intravenously.

**Normal ureters**
**Radiography**
Normally course caudally and may see peristaltic contractions on sequential studies.
Ultrasonography
Transverse scan is best for orientation.Requires high resolution transducer
Identify two vesicoureteral junctions and with Doppler may see two ureteral jets

**Ectopic Ureter**
Usually requires an Intravenous Urogram or retrograde urethrography

Ultrasonography
Recently ultrasonographic diagnosis has been used. Instill normal saline into the bladder to change the specific gravity
With ureteral ectopia may only find one papilla and ureteral jet
Colour Flow Doppler makes it easier still as pick up the ureteral jet as a turbulent flow

**Retrograde vagino-urethrography:** A useful technique to outline vaginal masses and often used to identify ectopic ureters. However, it is not 100% successful in identifying the latter condition..

An enema should be given at least one hour prior to the procedure. Deep sedation is required but often a GA is necessary. Lateral recumbency. A cuffed pre-filed Foley catheter is introduced into the vulva and inflated.

**Contrast Agent:** Urographin 150 60%. Dosage 1ml contrast/kg bw maximum.
The contrast is gently introduced and a radiograph taken immediately.

**Retrograde Urethrography:** Male dog. Useful for evaluation of the integrity and patency of the urethra. Deep sedation, lateral recumbency. Plain studies should be taken in the standard lateral position with the hindlimbs taken caudally. A second study should be performed with the hindlimbs drawn cranially and the central beam aligned to the penile urethra. Catheterise the urethra to the base of the os penis. Approximately 5 to 15 ml of positive contrast diluted to 30% should be introduced gently and stopped immediately if back-pressure is appreciated in the syringe. An immediate lateral study should be obtained.

**Urethra:-** Radiography is the imaging method of choice
Obstruction
Rupture
Neoplasia

## References

(1) Principles of Veterinary Radiography Douglas, Herrtage and Williamson. 1989. 4th Edition. Lea & Febiger.
(2) B.S.A.V.A. Manual of Veterinary Radiography and Radiology. B.S.A.V.A. Publication. 1995. Editor - Robin Lee.
(3) Diagnostic Radiology and ultrasonography of the Dog and Cat Kealy and McAllister. April 2000 3rd Edition. W.B. Saunders.

# Arterial Blood Gas Analysis

**Steve Haskins, DVM, DACVECC**

**Collection, storage, or measurement issues**

Blood must be collected anaerobically. Exposure to air allows equilibration of gases between the air and the blood. This will lower the partial pressure of carbon dioxide and change the partial pressure of oxygen in the sample toward that of room air (150 mmHg at sea level). Small air bubbles which sometimes occur during blood sampling should be avoided, but if they occur, should be immediately expelled from the sample as soon as the sample is collected. Strong negative pressure when sampling should be avoided since this may pull gases out of solution and decrease the measured values.

Arterial acid-base values reported for normal individuals.

| | Human[Martin L, 1999] | Dog[DiBartola, 2000] | Cat[DiBartola, 2000] |
|---|---|---|---|
| pH | 7.40 (7.35-7.45) | 7.41 (7.35-7.46) | 7.39 (7.31-7.46) |
| PaCO2 | 40 (35-45) | 37 (32-43) | 31 (26-36) |
| Base deficit | 0 (-2 to +2) | -2 (+1 to -5) | -5 (-2 to -8) |
| Bicarbonate | 24 (22-26) | 22 (18-26) | 18 (14-22) |
| PaO2 (sea level) | 95 (80-105) | 92 (80-105) | 105 (95-115) |

Mixed, jugular, and cephalic venous acid-base values compared to arterial for normal dogs (Ilkiw JE, 1991).

| | Arterial | Mixed venous | Jugular vein | Cephalic vein |
|---|---|---|---|---|
| pH | 7.40 ± 0.03 | 7.36 ± 0.02 | 7.35 ± 0.02 | 7.36 ± 0.02 |
| PCO2 | 37 ± 3 | 43 ± 4 | 42 ± 5 | 43 ± 3 |
| Base deficit | -2 ± 2 | -1 ± 1 | -2 ± 2 | -1 ± 1 |
| Bicarbonate | 21 ± 2 | 23 ± 2 | 22 ± 2 | 23 ± 1 |
| Total CO2 | 22 ± 2 | 24 ± 2 | 23 ± 2 | 24 ± 2 |
| PO2 | 102 ± 7 | 53 ± 10 | 55 ± 10 | 58 ± 9 |

The collector should be sure from where the sample is actually obtained. Arterial, central venous, and peripheral venous samples generate different measured values and must be interpreted in the context of their source. Arterial blood sampling is important if the purpose of the measurement is to evaluate pulmonary function and blood oxygenation. It is not so important if the purpose of the investigation is to evaluate acid-base status. In the normal patient, venous blood acid-base values are similar to arterial values and can usually be interpreted using the same guidelines as for an arterial sample. Under certain circumstances, however, the interposed tissue bed induces substantial disparities between arterial and venous blood analyses. Sluggish peripheral blood flow (hypovolemic shock; cardiac arrest), impaired carbon dioxide carriage (anemia, administration of carbonic anhydrase inhibitors) increase venous PCO2.

The sample should be analyzed immediately after collection to minimize *in vitro* metabolism which will decrease oxygen and pH, and increase carbon dioxide values. Important changes may occur with room temperature storage times of as little as 10 minutes (Kelman, 1966). If the analysis cannot be

made immediately, the sample should be stored in ice water. Ice without water is not sufficient; air pockets around the syringe insulate it from the cooling effect of the ice.

Dilution of the blood sample by anticoagulant will change the measured values. Heparin has a pH of about 5.8, a bicarbonate of about zero, a PCO2 of about 5 mmHg and a PO2 of about 165 mmHg. The dead space of a 3 ml syringe and needle is about 0.1 ml. This represents a 9% dilution of a 1 ml blood sample and a 3.2% dilution of a 3 ml blood sample. A 10% dilution of whole blood with heparin slightly increases the pH by about 0.02 units, decreases the PCO2 by about 7 mEq/L, decreases the bicarbonate concentration and increases the base deficit by about 3 mEq/L; and decreases the PO2 when it is above 165 and slightly increases it when it is below 165. Obviously the less the dilution, the more the sample will represent the status of the patient.

The effect of temperature on acid-base variables

| °C | °F | pH | PCO2 | $HCO_3^-$ | PO2 |
|---|---|---|---|---|---|
| 25 | 77 | 7.58 | 24 | 22 | 37 |
| 30 | 86 | 7.5 | 30 | 22.7 | 51 |
| 35 | 95 | 7.43 | 37 | 23.5 | 70 |
| 37 | 99 | 7.40 | 40 | 24 | 80 |
| 40 | 104 | 7.36 | 45 | 24 | 97 |

Blood gases are measured at the temperature of the blood gas analyzer water bath - usually 37 °C. When the animal's body temperature is different from that of the water bath, there will be in vitro pH and blood gas changes associated with the change in temperature of the blood sample as it equilibrates with that of the water bath in the surrounding measuring electrodes. The pH decreases as the blood is warmed due to the increased ionization of $H_3O^+$ and the decreased solubility of carbon dioxide (increases the partial pressures of the gas). Base deficit and bicarbonate are calculated from pH and PCO2 and do not change with *in vitro* changes in blood temperature. Correcting for *in vitro* temperature changes will more accurately reflect the actual values in the patient at the time the blood sample was taken. It would seem to be a more accurate way to report a series of acid-base values when the body temperature of the subject is fluctuating. There is, however, some debate about whether or not to correct for these *in vitro* temperature changes and then implementing therapy based upon these corrected values. Dogs (Ohmura A, 1979) and human infants (Matthews AJ, 1984) experiencing deep hypothermia maintained better hemodynamics if the $PCO_2$ is maintained at about 40 mm Hg as measured at 37 °C without temperature correcting to the temperature of the patient. The problem is correction of the values to the patient's temperature and then use normothermic standards by which to evaluate the results and initiate therapy. Appropriate values for hypothermic or hyperthermic patients have not been established for all temperature levels.

**The Partial Pressure of Carbon Dioxide ($PCO_2$)**

The arterial $PCO_2$ ($PaCO_2$) is a measure of the ventilatory status of the patient and normally ranges between 35 and 45 mmHg. A $PaCO_2 < 35$ mmHg indicates hyperventilation; a $PaCO_2 > 45$ mmHg indicates hypoventilation. A $PaCO_2$ in excess of 60 mmHg may be associated with excessive respiratory acidosis and hypoxemia (when breathing room air) and is usually considered to represent sufficient hypoventilation to warrant ventilation therapy. $PaCO_2$ values below 20 mmHg are associated with severe respiratory alkalosis and a decreased cerebral blood flow which may impair cerebral oxygenation.

Venous $PCO_2$ is usually 3 to 6 mm Hg higher than arterial in stable states. It is variably higher in transition states, during anemia, and carbonic anhydrase inhibitor therapy. It is a reflection of tissue $PCO_2$, which represents some combination of arterial $PCO_2$ and tissue metabolism.

perfusion ↓ (shock) Carriage ) $CO_2$ problems.

**Causes of hypercapnia**

| |
|---|
| Hypoventilation |
| Neuromuscular disorder |
| Medullary dysfunction (excessive depths of anesthesia; intracranial disease) |
| Cervical disease or neuromuscular disease |
| Airway obstruction |
| Large airway obstruction (laryngeal paralysis; tracheal collapse) |
| Small airway obstruction (chronic airway disease; bronchoconstriction) |
| Thoracic or abdominal restrictive disease (pleural fibrosis; abdominal enlargements) |
| Pleural space filling disorder (air; fluid) |
| Pulmonary parenchymal disease (terminal) |
| Inappropriate ventilator settings |
| Dead space rebreathing |
| Recent bicarbonate therapy |

$PaCO_2$ may also be estimated by measuring the carbon dioxide in a sample of gas taken at the end of an exhalation. The analyzer should be calibrated and, periodically, should be correlated to an actual $PaCO_2$ measurement. The accuracy of these instruments can be impaired by many causes. The presumption in correlating end-tidal $PCO_2$ with $PaCO_2$ is that alveolar and capillary $PCO_2$ are equilibrated. End-tidal $PCO_2$ is usually somewhat lower than $PaCO_2$ (1-4 mm Hg in humans and dogs, and 10-15 mm Hg lower in horses), but the difference is usually inconsequential for clinical purposes. Capnography allows the anesthetist to evaluate adequacy of ventilation, and many other problems as well. Whenever possible, the capnographic waveform should be displayed as it will reveal more information than just the end-tidal $CO_2$ measurement.

Hypercapnia may be caused by hypoventilation or dead space rebreathing.

**Alveolar gas composition**

Atmospheric air at sea level primarily contains nitrogen and oxygen, small amounts of water and carbon dioxide, and very small amounts of other gases and pollutants (Table 8). The partial pressure of oxygen is calculated as barometric pressure (760 at sea level) x 21% = 160 mm Hg. Inspired air becomes fully humidified when it is breathed into the alveoli. The vapor pressure of water in fully saturated air at 38 °C is 50 mm Hg (Table 5). The net partial pressure of the inspired oxygen is then calculated as barometric pressure (760 mm Hg at sea level) minus water vapor (50 mm Hg at 38°C) x 21% = 150 mm Hg. Venous blood is continuously delivers carbon dioxide to the alveoli and removes oxygen from the alveoli as it traverses the capillary bed. The carbon dioxide and oxygen diffuse down a partial pressure gradient until the partial pressure between the alveolus and the capillary blood is equilibrated. Alveolar gas composition, compared to atmospheric, has a higher water vapor and carbon dioxide, and a lower oxygen, partial pressure and concentration (Table 8). Alveolar $PO_2$ ($P_AO_2$) can be calculated by the alveolar air equation:

$$P_AO_2 = ([P_b - P_{H2O}] \times \%\ \text{oxygen}) - PaCO_2\ (1/RQ)$$

where $P_b$ = barometric pressure; $P_{H2O}$ = partial pressure of water of saturated air at body temperature; and RQ is respiratory quotient, the ratio of carbon dioxide production to oxygen consumption. ($[P_b - P_{H2O}] \times$ % oxygen ) calculates the partial pressure of inspired oxygen ($P_IO_2$). The $P_AO_2$ can be calculated for any altitude by inserting the appropriate barometric pressure for that altitude into the formula for $P_IO_2$ (Table 7). The $P_AO_2$ can be calculated for any inspired oxygen concentration by substituting the appropriate oxygen concentration into the $P_IO_2$ formula. The $P_IO_2$ at sea level and 21% oxygen, is invariably about 150 mm Hg. If an average RQ in a critically ill patient is about 0.9, 1/RQ = 1.1. The alveolar $PO_2$ equation can therefore be shortened to:

$$P_AO_2 = 150 - (PaCO_2 \times 1.1)$$

At sea level and 21% oxygen, $P_AO_2$ is calculated to be about 106 mm Hg.

**Oxygen partial pressure vs hemoglobin saturation**

Blood oxygen can be expressed in three different ways: the partial pressure of oxygen dissolved in the plasma ($PO_2$; units = mm Hg), the percent saturation of the hemoglobin ($SO_2$; units = %), and the whole blood oxygen content ($ContentO_2$; units = mls of oxygen per 100 mls whole blood).

The $PO_2$ is the partial pressure (the vapor pressure) of oxygen dissolved in solution in the plasma and is measured with a blood gas machine with a silver anode/platinum cathode system in an electrolyte

solution (polarography) separated from the unknown solution (the blood) by a semipermeable (to oxygen) membrane. The normal $PaO_2$ at sea level ranges between 80 and 110 mm Hg. Hypoxemia is usually defined as a $PaO_2 < 80$ mm Hg (at sea level). It is generally accepted that patients with a $PaO_2$ of less than 60 mm Hg require treatment. There are only two treatments: 1) increase the inspired oxygen concentration; 2) mechanical ventilation; or some combination of the two.

The $SO_2$ is the percent saturation of the hemoglobin. It can be measured with a bench top oximeter or a pulse oximeter, or it can be extrapolated from the measured $PO_2$ via a standard oxyhemoglobin dissociation curve. Oximetry is based upon the pattern of red to infra-red light absorption of hemoglobin: oxyhemoglobin, reduced hemoglobin, methemoglobin, and carboxyhemoglobin each absorb light differently. One wave length of light, preferably one that maximizes the difference between the hemoglobin species of interest and the others, is required to identify each species of hemoglobin. Pulse oximeters use only two wave lengths (660 and 940 nm) and are designed to measure oxygenated and unoxygenated hemoglobin. If methemoglobin or carboxyhemoglobin are present in high concentrations, they will absorb light and will impact the measurement made by the pulse oximeter. Due to the biphasic absorption of methemoglobin at both the 660 and 940 nm wavelengths, abnormal accumulations of this hemoglobin species tends to push the pulse oximeter reading toward 85% (underestimating measurements when $SaO_2$ is above 85% and overestimating it when below 85%). Carboxyhemoglobin absorbs light like oxyhemoglobin at 660 nm but hardly at all at 940 nm and this would increase the apparent oxyhemoglobin value.

The percent saturation of the hemoglobin is related to the $PaO_2$ and defines the oxyhemoglobin dissociation curve (fig. 12; Table 10). The hemoglobin is near fully saturated when the $PaO_2$ is 100 mm Hg. Increasing the $PaO_2$ to 500 mm Hg barely increases the percent saturation. A $PaO_2$ of 80 mm Hg is associated with an $SaO_2$ of approximately 96% , which is the level below which defines hypoxemia and a $PaO_2$ of 60 mm Hg is associated with an $SaO_2$ of approximately 91%, which corresponds to a level of hypoxemia below which warrants treatment.

**Venous admixture**

Hypoxemia is usually defined as a $PaO_2$ of less than 80 mm Hg. Hypoxemia may be caused by a decreased inspired oxygen concentration, by hypoventilation, or by lung disease. Diminished pulmonary oxygenating efficiency in lung disease is caused by venous admixture.

Venous admixture represents all of the ways in which blood can pass from the right ventricle to the left ventricle without being properly arterialized. Some of this occurs normally via the bronchial circulation, the thebesian veins which drain from the myocardium directly into the left ventricle, and via suboptimal arterialization of blood as it traverses the low V/Q regions of the lung. Venous admixture can be increased by four physiologic mechanisms: 1) an increased number of low V/Q lung units, 2) by atelectasis or alveolar filling (no ventilation but perfused lung units), 3) by diffusion impairment, and 4) by anatomic right-to-left shunts. Of these four, the first two are by far the most common.

The quantification of the magnitude of venous admixture provides an assessment of the oxygenating efficiency of the lung . Venous admixture can be identified several different ways.

1) The alveolar - arterial $PO_2$ gradient (A-a $PO_2$) is the difference between the calculated alveolar partial pressure of oxygen ($P_AO_2$) and the measured arterial partial pressure of oxygen ($PaO_2$) . The A-a $PO_2$ is normally about 10 mm Hg when breathing 21% oxygen at sea level and is about 100 mm Hg when breathing 100% oxygen. If the calculated A-a $PO_2$ is greater than 15 mm Hg when the animal is breathing room air or greater that 150 mm Hg when the animal is breathing 100% oxygen, the animal has venous admixture. The greater the A-a $PO_2$ , the greater the venous admixture. The expected A-a $PO_2$ at intermediate inspired oxygen concentrations has not been established and must be extrapolated between 10 and 100 mm Hg; values suggestive of venous admixture should be extrapolated between 15 and 150 mm Hg (Table 11).

2) There are only four gases of note in the alveoli (nitrogen, water vapor, oxygen, and carbon dioxide). Barometric pressure and the partial pressures of nitrogen and water vapor do not change. Since capillary blood flow adds carbon dioxide to and removes oxygen from the alveolar gases at a rate that is comparable to metabolic production and consumption of these two gases, respectively, the alveolar $PCO_2$ and $PO_2$ are approximately reciprocally related and must total an approximately fixed value of 150 mm Hg at sea level. Assuming an A-a $PO_2$ of about 10 mm Hg, the $PaO_2 + PaCO_2$ added value for arterial blood should be about140 mm Hg (range 120 to 160 mm Hg). Added $PaO_2$ and $PaCO_2$ values of less than 120 mm Hg indicate a reduced lung oxygenating efficiency; added values above 160 (breathing 21% oxygen at

sea level) are physiologically impossible (there is a measurement error). The further the added value is below 120 mm Hg, the greater is the venous admixture. This assumes that the patient is breathing 21% oxygen at sea level. If the patient is not breathing an enriched oxygen mixture or is not at sea level, the original alveolar air equation and the calculation of A-a $PO_2$ must be used to estimate venous admixture.

3) For the same reasons, but from a slightly different perspective, the relative change of the $PaCO_2$ and the $PaO_2$ can be used to asses's lung oxygenating efficiency. If one assumes a central normal $PaO_2$ of 100 mm Hg, a central normal $PaCO_2$ of 40 mm Hg, an RQ of 1.0, and 21% inspired oxygen at sea level, the magnitude of change in $PaCO_2$ would be expected to cause an equal and opposite change in $PaO_2$. If the $PaCO_2$ , for instance, decreases 20 points from 40 to 20 mm Hg, the $PaO_2$ would be expected to increase 20 points from 100 to about 120 mm Hg. Likewise if the $PaCO_2$ increases 40 points from 40 to 80 mm Hg, the $PaO_2$ would be expected to decrease 40 points from 100 to about 60 mm Hg. If the measured $PaO_2$ is more than 20 mm Hg below that predicted by this calculation, venous admixture is present. The further the measured $PaO_2$ is below the predicted value, the greater the venous admixture.

4) When breathing 21% oxygen, changes in $PaCO_2$ have an important impact on $PaO_2$ and must be taken into account when calculating the expected $PaO_2$. With progressively higher inspired oxygen concentrations, changes in $PaCO_2$ have a progressively less important effect on $PaO_2$ and, for clinical purposes, can legitimately be ignored at higher inspired oxygen concentrations. A common rule-of-thumb method which is especially helpful when the animal is breathing an enriched inspired oxygen concentration, is that the anticipated $PaO_2$ should be at least 5 times the inspired oxygen concentration. If the patient is breathing 50% oxygen, for instance, the anticipated $PaO_2$ should be at least 250 mm Hg. In normal lungs, the $PaO_2$/inspired $O_2$ ratio is > 5. Values between 3 and 5 represent mild oxygenating inefficiency; values between 2 and 3 represent moderate lung inefficiency; and values below 2 represent severe venous admixture.

5) The most popular expression of lung oxygenating efficiency in the literature nowadays is the $PaO_2/FiO_2$ ratio. This approach is essentially the same as approach # 4 except that instead of dividing by a whole number (% inspired oxygen; room air = 21%), one divides by a fraction (the fractional inspired oxygen concentration - $FiO_2$; room air = 0.21). In normal lungs the $PaO_2/FiO_2$ ratio is > 500 mm Hg. Values between 300 and 500 represent mild oxygenating inefficiency; values between 200 and 300 represent moderate lung inefficiency; and values below 200 represent severe venous admixture.

6) If mixed venous blood (pulmonary artery) can be obtained, the venous admixture can be calculated by the following equation.

$$Q_S/Q_T = (CcO_2 - CvO_2) / (CcO_2 - CaO_2)$$

where $Q_S/Q_T$ = venous admixture expressed as a percent of cardiac output; $CcO_2$ = oxygen content of end-capillary blood; $CvO_2$ = oxygen content of mixed venous blood; and $CaO_2$ = oxygen content of arterial blood. Oxygen content (ml/dl) is calculated by the following formula.

$$ContO_2 = (1.34 \times Hb \times SO_2) + (0.003 \times PO_2)$$

where $SO_2$ is percent hemoglobin saturation with oxygen (which is usually derived from a standard oxyhemoglobin dissociation curve), and $PO_2$ is the partial pressure of oxygen in the respective blood sample. Venous admixture is normally less than 5% of the cardiac output; values over 10% are considered to be increased. Venous admixture may increase to over 50% in diffuse lung disease.

**Whole blood oxygen content and oxygen delivery**

Oxygen content is dependent upon both hemoglobin concentration and $PO_2$. Oxygen content is calculated by the formula above or may be measured with a galvanic cell quantitative analyzer. Like hemoglobin saturation, the relationship between oxygen content and $PO_2$ is defined by a sigmoid curve[43]. The $PaO_2$ at which human hemoglobin is 50% saturated ($P_{50}$) is about 27 mm Hg. $P_{50}$ is a common way to define the position of the curve; whether it is shifted to the left (a lower $P_{50}$ value due to higher hemoglobin affinity for oxygen) or to the right (a higher $P_{50}$ value due to lower hemoglobin affinity for oxygen). The $P_{50}$ for canine hemoglobin is about 29 mm Hg and so table 10 is close enough for clinical purposes for the dog. The $P_{50}$ for feline hemoglobin, however, is about 36 mm Hg and so this table is not accurate for the cat. For mean $PO_2$ values of 621, 151, 60, and 41, for instance, we measured hemoglobin saturations to be 100, 97.8, 79.1, and 61.2, respectively. Corresponding oxygen content values (assuming a hemoglobin concentration of 15 g/dl) would be 22.0, 20.1, 16.1, and 12.4 ml/dl, respectively. This represents a rightward shift of the oxyhemoglobin dissociation curve for the cat compared to the dog. Like hemoglobin saturation, oxygen content does not increase much when the $PO_2$ is raised above 100 mm Hg; the hemoglobin is mostly full and further increases in content are attributed mostly to an increase in dissolved

oxygen in the plasma.

Hemoglobin concentration is quantitatively the most important contributor to oxygen content. Anemia is a far more potent cause of decreased oxygen content than is hypoxemia. Hypoxemia is primarily important in that it is the partial pressure of oxygen that provides the driving pressure responsible for the flow of oxygen molecules from the plasma to the mitochondria. Content is important as a reservoir of oxygen to buffer the decrease in $P_{O_2}$ that would occur when oxygen molecules diffuse out of the plasma.

The treatment for excessive anemia is an infusion of a hemoglobin-containing solution. However, in the interim, between recognition of severe anemia and implementation of the transfusion, does an enriched inspired oxygen concentration benefit the patient? Clearly, even 100% inspired oxygen does not increase the oxygen content very much in the anemic patient and not nearly enough to provide all of the oxygen requirements of the patient. Anemia causes problems for the patient when the blood oxygen content becomes insufficient to meet the metabolic requirements of oxygen consumption. It is not, however, necessary to meet the entire oxygen consumption requirements of the animal with the oxygen therapy. It is only necessary to increase the oxygen content enough to move the animal from a little below the "death line" to a little above it. Sometimes a little bit of help can make a lot of difference to a patient and oxygen therapy is recommended in the anemic patient.

Oxygen delivery is the product of oxygen content and cardiac output. Animals can tolerate a decrease in oxygen content if they can increase their cardiac output to compensate. Animals tolerate anemia and poor cardiac output poorly.

Oxygen delivery needs to be sufficient to meet the consumption requirements of the patient. Normally oxygen delivery far exceeds oxygen consumption. Oxygen extraction normally ranges between 20 and 30% of the oxygen delivery. Mixed venous oxygen represents the balance between whole body oxygen delivery and oxygen consumption. Mixed or central venous $PO_2$ ranges between 40 and 50 mm Hg in normal dogs. When oxygen delivery is decreased (low cardiac output, anemia, hypoxemia,, vasoconstriction), the tissues continue to "draw" their normal amount of oxygen and so oxygen extraction increases and venous oxygen decreases. Venous $PO_2$ values below 30 mm Hg are usually attributed to excessively low oxygen delivery, but could be caused by high oxygen consumption. Values below 20 mm Hg should be considered life-threatening. When oxygen delivery finally becomes too low to support oxidative phosphorylation, lactic acidosis ensues. Venous $PO_2$ values above 60 mm Hg (while breathing room air) are primarily suggestive of reduced tissue uptake of oxygen (shunting, septic shock, metabolic poisons, hypothermia), but could also be attributed to high oxygen delivery .

Hemoglobin saturation (SO2) and oxygen content for various PO2 values

| $PO_2$ | $SO_2$ | $ContO_2$ | $PO_2$ | $SO_2$ | $ContO_2$ | $PO_2$ | $SO_2$ | $ContO_2$ |
|---|---|---|---|---|---|---|---|---|
| 500 | 99.9 | 21.6 | 82 | 96.1 | 19.6 | 46 | 81.5 | 16.5 |
| 400 | 99.8 | 21.3 | 81 | 95.9 | 19.5 | 45 | 80.4 | 16.3 |
| 300 | 99.7 | 20.9 | 80 | 95.8 | 19.5 | 44 | 79.3 | 16.1 |
| 200 | 99.4 | 20.6 | 79 | 95.7 | 19.5 | 43 | 78.1 | 15.8 |
| 150 | 98.9 | 20.3 | 78 | 95.5 | 19.4 | 42 | 76.8 | 15.6 |
| 125 | 98.4 | 20.2 | 77 | 95.4 | 19.4 | 41 | 75.5 | 15.3 |
| 120 | 98.3 | 20.1 | 76 | 95.2 | 19.4 | 40 | 74.1 | 15.0 |
| 115 | 98.1 | 20.1 | 75 | 95.1 | 19.3 | 39 | 72.7 | 14.7 |
| 110 | 98.0 | 20.0 | 74 | 94.9 | 19.3 | 38 | 71.1 | 14.4 |
| 109 | 97.9 | 20.0 | 73 | 94.7 | 19.3 | 37 | 69.5 | 14.1 |
| 108 | 97.9 | 20.0 | 72 | 94.5 | 19.2 | 36 | 67.9 | 13.8 |
| 107 | 97.8 | 20.0 | 71 | 94.3 | 19.2 | 35 | 66.2 | 13.4 |
| 106 | 97.8 | 20.0 | 70 | 94.1 | 19.1 | 34 | 64.4 | 13.0 |
| 105 | 97.7 | 20.0 | 69 | 93.9 | 19.1 | 33 | 62.6 | 12.7 |
| 104 | 97.7 | 19.9 | 68 | 93.6 | 19.0 | 32 | 60.7 | 12.3 |
| 103 | 97.6 | 19.9 | 67 | 93.4 | 19.0 | 31 | 58.7 | 11.9 |
| 102 | 97.6 | 19.9 | 66 | 93.1 | 18.9 | 30 | 56.7 | 11.5 |
| 101 | 97.5 | 19.9 | 65 | 92.8 | 18.9 | 29 | 54.6 | 11.1 |
| 100 | 97.5 | 19.9 | 64 | 92.5 | 18.8 | 28 | 52.5 | 10.6 |
| 99 | 97.4 | 19.9 | 63 | 92.2 | 18.7 | 27 | 50.4 | 10.2 |
| 98 | 97.4 | 19.9 | 62 | 91.8 | 18.6 | 26 | 48.1 | 9.8 |
| 97 | 97.3 | 19.9 | 61 | 91.5 | 18.6 | 25 | 45.9 | 9.3 |
| 96 | 97.3 | 19.8 | 60 | 91.1 | 18.5 | 24 | 43.6 | 8.8 |
| 95 | 97.2 | 19.8 | 59 | 90.6 | 18.4 | 23 | 41.2 | 8.4 |
| 94 | 97.1 | 19.8 | 58 | 90.2 | 18.3 | 22 | 38.8 | 7.9 |
| 93 | 97.1 | 19.8 | 57 | 89.7 | 18.2 | 21 | 36.3 | 7.4 |
| 92 | 97.0 | 19.8 | 56 | 89.2 | 18.1 | 20 | 33.8 | 6.9 |
| 91 | 96.9 | 19.8 | 55 | 88.6 | 18.0 | 19 | 31.3 | 6.3 |
| 90 | 96.8 | 19.7 | 54 | 88.0 | 17.8 | 18 | 28.7 | 5.8 |
| 89 | 96.7 | 19.7 | 53 | 87.3 | 17.7 | 17 | 26.1 | 5.3 |
| 88 | 96.7 | 19.7 | 52 | 86.7 | 17.6 | 16 | 23.5 | 4.8 |
| 87 | 96.6 | 19.7 | 51 | 85.9 | 17.4 | 15 | 20.9 | 4.3 |
| 86 | 96.5 | 19.6 | 50 | 85.1 | 17.3 | 14 | 18.4 | 3.7 |
| 85 | 96.4 | 19.6 | 49 | 84.3 | 17.1 | 13 | 15.8 | 3.2 |
| 84 | 96.3 | 19.6 | 48 | 83.4 | 16.9 | 12 | 13.3 | 2.7 |
| 83 | 96.2 | 19.6 | 47 | 82.5 | 16.7 | 11 | 10.9 | 2.2 |

$PO_2$ = partial pressure of oxygen (mmHg); $SO_2$ = oxyhemoglobin saturation (%), calculated from $PO_2$ using a mathematical model for human hemoglobin[43]; $ContO_2$ = oxygen content (ml/dl), calculated by the formula: $(1.34 \times Hb \times SO_2) + (0.003 \times PO_2)$.

**Effect of anemia, hypoxemia, and oxygen breathing on $PO_2$, $SO_2$, and $ContentO_2$**

| Condition | $F_IO_2$ | $PO_2$ (mm Hg) | $SO_2$ (%) | Hemoglobin (gm/dl) | $ContentO_2$ (ml/dl) |
|---|---|---|---|---|---|
| Normal | 0.21 | 100 | 98 | 15 | 19.9 |
| Anemia | 0.21 | 100 | 98 | 5 | 6.9 |
| Hypoxemia | 0.21 | 50 | 85 | 15 | 17.3 |
| Hyperoxemia | 1.0 | 500 | 99.9 | 15 | 21.6 |
| Anemia & hyperoxemia | 1.0 | 500 | 99.9 | 5 | 8.2 |
| Anemia & hypoxemia | 0.21 | 50 | 85 | 5 | 5.8 |

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# Laboratory assessment – haematology & biochemistry

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## Introduction

Surgical patients presenting urgently can often be an anaesthetic challenge. Although a pertinent history and physical assessment of all major body systems will provide much of the necessary information, some basic laboratory data will be invaluable, not only in managing life-threatening disease (eg Addisonian crisis, hypocalcaemia), but also to allow effective drug and procedure selection and monitor responses to therapy (transfusion, electrolyte supplementation). Rapid generation of clinically acceptable results is possible with limited equipment.

## What samples should be collected?

Blood and urine samples will yield all of the necessary data. **It is highly desirable that all samples be taken prior to initiating any therapy**; this will then provide a reference value for all subsequent tests. Assessing urine specific gravity after a bolus of intravenous fluid is not very useful. Cystocentesis should be considered if a urinary bladder is palpable. Ideally, blood samples should not be taken through intravenous cannulae unless the cannula has just been placed; even then a portion should be discarded first. If a cannula is not in place then blood may be collected into capillary tubes placed into the hub of a 25G needle inserted into a vein.

The database should include:

Complete blood count (CBC) + peripheral blood smear
Total Plasma Proteins (TPP; referred to as total solids in some texts)
Glucose
BUN/Urea
Creatinine
Urinalysis (specific gravity, pH, protein, glucose, blood, ketones)

Depending on the clinical circumstance, other follow-up tests may also be required:
White blood cell count
Liver function tests

## Interpretation of values

### Packed Cell Volume and Total Plasma Proteins

- **Packed cell volume (PCV)**: Is the percentage of blood made up of RBCs as measured by centrifugation of capillary tubes. This is a reliable test for the evaluation of anaemia (decreased PCV) or erythrocytosis (increased PCV).
  Results are expressed as %.

- **Haematocrit (Hct)**: Is also the percentage of blood made up of RBCs, but is calculated by an automated cell counter from the number of red blood cells per litre and the average volume of each red cell (MCV).
  Results are expressed in %.

These values should be interpreted in conjunction with each other, as their relative changes may be significant. Acute blood loss will not immediately affect PCV & TPP due to the time lag involved in fluid shifts from the interstitial space. PCV may actually increase in dogs following haemorrhage as a result of acute splenic contraction and release of sequestered red cells. Total protein may be a more sensitive guide to blood loss; a trauma patient with a normal PCV but with TPP <60g/l should be investigated for recent haemorrhage. Changes in PCV and TPP will become more apparent with fluid administration.

A low PCV with normal TPP is suggestive of anaemia (increased red cell loss destruction or inadequate production). Jaundiced or red serum is suggestive of haemolytic disease, prompting a slide agglutination test to assist in the diagnosis/exclusion of immune-mediated haemolysis. Hepatic and post-hepatic causes of icterus must also be excluded. The level to which PCV must decline before clinical signs of reduced oxygen-carrying capacity become apparent is highly variable, and depends on the speed of red cell loss as well as the nature (ie haemolysis of haemorrhage). Cats tend to be more tolerant of major red cell loss than dogs.

A high PCV may be breed-related (GSD, sighthounds) or physiologic (altitude). Pathologic causes of a high PCV include relative polycythaemia (secondary to haemoconcentration) with concomitantly elevated TPP, or absolute polycythaemia (myeloproliferative disease, chronic hypoxia, renal neoplasia). Clinical signs related to increased blood viscosity and poor tissue perfusion (neurologic signs, cyanosis, tortuous retinal vessels, coagulation defects) may be evident in the latter cases.

Decreases in total proteins may result from haemorrhage, fluid overload, third-space losses (pyothorax, peritonitis, acute gut losses), protein-losing diseases (intestinal disease, protein-losing nephropathy (PLN)) or liver disease resulting in decreased albumin production. Intestinal protein loss usually results in panhypoproteinaemia (ie low albumin and globulins) whereas globulins in PLN are usually normal and in hepatic disease may be raised. Other database changes that would be suggestive of liver dysfunction include low urea, hypoglycaemia, and coagulation abnormalities. Further testing might also show increases in ammonia & bilirubin and a low cholesterol.

Increased TPP values will occur with haemoconcentration or prior administration of blood (in which case a higher PCV would be expected), blood products, or increases in plasma globulins (chronic antigenic stimulation, chronic inflammatory disease, myeloma, lymphoma).

The following table highlights the more common relative shifts in PCV and total proteins:

| PCV | Total Proteins | Differential Diagnosis |
|---|---|---|
| Increased | Increased | Dehydration |
| | Normal | Splenic contraction<br>Primary or secondary polycythaemia<br>Haemorrhagic gastroenteritis (HGE) |
| Normal | Normal | Normal<br>Dehydration with hypoproteinaemia<br>Anaemia with hypoproteinaemia and dehydration |
| | Decreased | Recent haemorrhage (mild)<br>Liver disease<br>Protein losing nephropathy/enteropathy<br>Third-space protein loss |
| Decreased | Normal | Haemolysis<br>Decreased red cell production<br>Chronic low-grade haemorrhage |
| | Decreased | Haemorrhage (acute) |

**Changes in red cell size**

Macrocytosis - most commonly due to regenerative anaemias in conjunction with a reticulocytosis

Microcytosis – may be due to:

- Iron deficiency – usually in conjunction with a thrombocytosis
- Chronic blood loss
- Portosystemic shunts
- Breed related

**Indicators of regeneration are:**

- Increased MCV
- Anisocytosis
- Polychromasia
- Macrocytosis
- Nucleated RBCs
- Howell-Jolly bodies
- Increased Reticulocyte count

**White Blood Cells**

**Leucocytosis**

An increase in the total circulating white cell count. Usually caused by an increase in the total number of neutrophils – neutrophilia. Leukocytosis can be due to several causes:

- Physiological
- Response to corticosteroids
- Response to inflammation

**Neutrophilia**
Causes – adrenalin, corticosteroid, inflammation, leukaemia

- Physiological
- Inflammation
  - Acute
  - Chronic
- Infectious
    - Localised
    - Systemic
- Non-infectious / Inflammatory
    - Immune mediated disease
    - Surgery
    - Tissue necrosis
    - Haemorrhage / haemolysis
    - Early oestrogen toxicity
    - Acute pancreatitis
- Neoplasia
    - Large necrotic tumour
    - Myeloproliferative disorder

**Degenerative left shift**
Indicates the number of immature neutrophils is greater than or equal to the number of mature cells. Most commonly seen in the early stages of gram-negative sepsis and with inflammation affecting a large surface area e.g. pleura, peritoneum. Mature neutrophils move into tissues and immature cells are released from the bone marrow. Uncontrolled infection or failure of the bone marrow to mount an adequate response can be reflected as a persistent degenerative left shift – a poor prognostic indicator. There can be a left shift or a degenerative left shift without a change in total WBC count and so blood smear examination is essential to recognise this.

**Neutropenia**
- Secondary to increased tissue demand, reduced cell production or ineffective production.

Lack of neutrophils means that with infection the classical signs may not be evident e.g. lack of pus formation.

Eosinophilia
- Hypersensitivity / Immune Mediated reactions (+/- basophilia)
- Parasitism (+/- basophilia)
- Eosinophilic gastroenteritis
- Eosinophilic myositis
- Panosteitis (GSD)
- Eosinophilic leukaemia

- Hypoadrenocorticism (usually sick/stressed animals are eosinopenic – ↑eosin's is a good indicator for hypoadrenocorticism)
- Oestrus in some bitches
- Neoplasia e.g. Mast cell tumour, lymphoma (cat)
- Hypereosinophilic syndrome (cats) – infiltration of organs by mature eosinophils and circulating eosinophilia, particularly affects intestines, liver & spleen.

Mild eosinophilia is normal in most German Shepherd dogs as a breed variation.

**Eosinopenia**

- Stress leukogram – neutrophilia, monocytosis, lymphopenia & eosinopenia
    - Stress, steroid therapy, Cushing's

**Basophilia**

- Functionally similar cell to mast cell
- Pro-inflammatory
- Usually occur with eosinophilia
- Associated with allergic respiratory disease, heartworm, pulmonary eosinophilic disease, rarely with Cushing's, heparin or penicillin administration.
- Basophilic leukaemia is very rare.

**Monocytosis**

- Stress, glucocorticoid therapy or hyperadrenocorticism
- Inflammatory disease especially pyogranulomatous / suppurative / necrotic inflammatory disease
- Immune mediated disease
- Neoplasm's with necrotic centres
- Monocytic / myelomonocytic leukaemia

**Lymphocytosis**

- Physiological
- Age related
- Chronic infections (particularly those which elicit antibody response)
- Hypoadrenocorticism
- Lymphocytic leukaemia

**Lymphopenia**

- Part of stress leukogram (see above)
- Viral disease
- Neoplasia
- Cell loss e.g. lymphangiectasia, repeated drainage of chylothorax
- Septicaemia / endotoxaemia (? Part of stress response)

**Thrombocytopenia**

Aetiology

- Decreased platelet production
- Increased platelet destruction
- Disorders of destruction / sequestration – usually involve liver or spleen e.g. splenic torsion also hypothermia, endotoxaemia
- Increased platelet consumption or use
- Disease associated
- Breed related
- Genetic predisposition to primary IMTP suggested by high disease prevalence in Cocker Spaniels, miniature & toy Poodles & Old English Sheepdogs
- Immune mediated diseases – female predisposition
- Commonly middle aged dogs for primary IMTP

**Thrombopathia**

Platelet function defects not defects in numbers of platelets
Inherited vs acquired

Inherited

- Chediak higashi syndrome of Persian cat – abnormal aggregation
- Thrombasthenia of Otterhound – platelets cannot aggregate or support clot formation
- δ-storage pool disease of American Cocker Spaniel – abnormal platelet aggregation
- Others e.g. Basset Hound, Spitz, Grey collies with cyclic haematopoiesis, DSH, Great Pyrenees

Acquired

- Drugs e.g. Aspirin, acepromazine
- Systemic disease e.g. uraemia, hepatic disease
- Haematologic disease e.g. IMTP, myelo/lymphoproliferative disease, dysproteinaemia [multiple myeloma]

**Thrombocytosis**

Reactive

- Iron deficiency anaemia
- Acute blood loss (days)
- Chronic inflammation
- Cushing's disease and corticosteroids
- Vincristine
- Rebound after thrombocytopenia

Primary

- Myeloproliferative disease

- Megakaryocytic leukaemia
- Essential thrombocythaemia
- Polycythaemia vera (rarely)

**Glucose**

Assessment of blood glucose may provide invaluable information in many emergency patients. Maintenance of glucose homeostasis involves the actions of insulin, glucagon, catecholamines, growth hormone and cortisol; therefore any disease process affecting the secretion or action of these substances is likely to disrupt blood glucose. The brain is the primary organ affected by hypoglycaemia and clinical signs may vary from depression to stupor, coma or seizures (blood glucose should be checked in all seizuring patients as a matter of urgency). In severely ill patients low blood glucose should prompt an aggressive search for a septic focus (especially intra-abdominal causes).
Athough transient hyperglycaemia can sometimes occur with a major CNS insult, hypoglycaemia is more common. Hyperglycaemia that persists through fasting is due to an absolute or relative inadequacy of insulin or insulin resistance. If this becomes severe then osmolar increases occur which may produce neurologic signs. The polyuria that occurs with hyperglycaemia may result in severe dehydration in patients with concurrent vomiting or a reduced fluid intake.

Differential diagnosis of hypoglycaemia (common causes in italics)

*Sepsis*
*Neoplasia*
*Hypoadrenocorticism*
Insulin overdose
*End-stage liver disease*
*Neonatal hypoglycaemia*
*Severe prolonged seizures*
Toy breed & Hunting-dog hypoglycaemia

Differential diagnosis of hyperglycaemia (common causes in italics)

*Stress (cats)*
*Diabetes mellitus*
*Corticosteroid use/hyperadrenocorticism*
*Acute pancreatitis*
Head trauma
*Prior administration of glucose-containing fluids*

## BUN/Urea & Creatinine

Following evaluation of urea, creatinine and other renally-associated parameters can be assessed to determine the nature and chronicity of the azotaemia. Low urea values seen in conjunction with low blood glucose levels are strongly suggestive of liver disease/failure.

Differential diagnosis of increased urea/creatinine:

- Pre-renal
  - Dehydration
  - Cardiac failure
  - Shock
- High-protein diet/GI bleeding
- Primary renal failure
- Post-renal
  - Urethral trauma/obstruction
  - Bladder trauma/obstruction

Differential diagnosis of decreased urea/creatinine:

- Diuresis (iv fluids, diabetes, hyperadrenocorticism)
- Liver failure (cirrhosis, portosystemic shunt)
- Low-protein diet
- Severe malnutrition

## Lactate

Tissue hypoperfusion results in increased lactate production and decreased lactate clearance, and blood lactate levels can be used to assess the severity of hypovolemia.The reference range for plasma lactate concentration in normal dogs (by direct amperometry) is less than 2.5 mmol/L irrespective of sample site. A blood lactate concentration in the range of 3-5 mmol/L constitutes a mild increase, 5-10 mmol/L is a moderate increase, and greater than 10 mmol/L represents a severe elevation. Clinical experience suggests that lactate concentration
accurately reflects the degree of hypovolemia in dogs. Furthermore, plasma lactate concentrations almost invariably fall with successful fluid resuscitation and can be used to guide fluid therapy in the treatment of hypovolaemia. Failure of plasma lactate concentration to normalize following fluidresuscitation suggests ongoing systemic hypoperfusion, or an occult local source of lactate production. Lactate can also be used as a prognostic indicator. In one of the landmark studies in people, as lactate concentration increased from 2.1 to 8.0 mmol/L survival decreased from 90% to 10%.I Experimental evidence and clinical experience suggests that similar results are obtained in canine patients. If plasma lactate concentration fails to fall below 10 mmol/L following appropriate fluid challenge, or if a significant and sustained rise in plasma lactate concentration occurs, the prognosis for survival appears to be grave to hopeless. Point-of-care lactate analysers are currently undergoing evaluation in cats and dogs.

## Electrolytes – Sodium, Potassium, Calcium

### Sodium Disorders

#### Hypernatraemia

Hypernatraemia may be due to gain of sodium in excess of water, loss of water in excess of sodium, or both. Net water loss is by far the most common cause in the emergency setting; fluid losses may be hypotonic (ie both sodium and water are lost but water losses are in excess of sodium losses) or pure water losses. Fluid losses are usually renal (eg osmotic diuresis) or gastrointestinal (eg osmotic diarrhoea) in origin; "third–space" losses such as effusions may also result in hyponatraemia. Reduced intake is most commonly due to vomiting but may also result from lack of access to water. Sodium gain may result from over-administration or (more rarely) from reduced excretion, such as in hyperadrenocorticism.

#### Clinical signs of hypernatraemia

The central nervous system (CNS) is the primary major body system affected by hypernatraemia, as a fluid shift occurs from the brain parenchyma to the intravascular space. This generally occurs with serum sodium concentrations in excess of 170mmol/l, as a gradient across the cell membrane of 30-35mOsmol/l is required. Speed of onset of this gradient appears to dictate severity of clinical signs more than gravity; the author has seen many patients with serum sodium concentrations in excess of 190-200mmol/l with no obvious neurologic disturbances. Early clinical signs usually consist of behavioural changes, lethargy and vomiting, and progress to seizures, stupor and coma. Acute changes in cell volume can result in cell death and irreversible CNS damage.

#### Hyponatraemia

As sodium constitutes the "osmotic skeleton" of extracellular fluid, disorders resulting in low sodium concentrations may reasonably be expected to result in disturbances in plasma osmolarity, particularly when osmolarity is low. Patients in this latter category can be further subclassified as:

*Hypovolaemic* – This is the most common category of patients likely to be encountered in the emergency setting. Fluid is lost via the kidneys (diuretics, hypoadrenocorticism), gut or as "third-space" losses (pleural and peritoneal effusions). Although non-renal losses are often hypotonic in nature, hyponatraemia develops due to impaired renal water excretion (secondary to reduced glomerular filtration), increased vasopressin release and increased thirst.

*Hypervolaemic* – the three main causes in this group are congestive cardiac failure, nephrotic syndrome and severe liver disease. All of these disorders result in activation of the vasopressin and renin-angiotensin axies, resulting in reduced free water excretion.

*Normovolaemic* – inappropriate ADH secretion, administration of hypotonic fluids, and psychogenic polydipsia are all potential causes, as a new "steady state" of mild overexpansion of extracellular fluid volume exists

Hyponatraemic patients with increased plasma osmolarity usually have increased levels of a solute other than sodium in the plasma. This is most commonly glucose (in diabetes) or mannitol (if administered). Total body water will invariably reduce in these patients as osmotic diuresis ensues.
Normal plasma osmolarity with hyponatraemia most commonly occurs in the presence of significant hyperlipidaemia (usually grossly visible) or absolute hyperproteinaemia. Reduction of the aqueous phase of the sample often results in erroneous measurement. This scenario is not common but should be remembered.

**Clinical signs of hyponatraemia**
As might be expected, acute decreases are more clinically obvious and serious than chronic decreases. In humans, decline faster than 0.5mmol/l/hr may be fatal. Influx of water into the CNS parenchyma produces the majority of the clinical signs in acute cases. More chronic development allows for loss of potassium and organic osmotically active compounds from CNS cells, resulting in a more normal brain volume. Common clinical signs in hyponatraemic patients may relate to the underlying disease, but commonly include weakness, ataxia and seizures.

**Potassium Disorders**

**Hyperkalaemia**
Hyperkalaemia invariably results from reduced renal excretion or extracellular shift of intracellular potassium (~98% of total body potassium is intracellular). Increased intake as a cause of hyperkalaemia in animals with a normal urinary tract is rare due to the efficiency of renal excretion.

Differential diagnosis of hyperkalaemia:

- Reduced renal excretion of potassium
  - Acute renal failure
  - Urinary tract obstruction or disruption
  - Hypoadrenocorticism
  - Severe gastrointestinal disease
- Extracellular shift:
  - Metabolic acidosis
  - Massive tissue injury (crush, snakebite, haemolysis, reperfusion)
- Miscellaneous causes
  - Oversupplementation of intravenous fluids
  - Drugs (suxamethonium, ACEI, beta-blockers)
  - *Trichuris vulpis* infestation
  - Body cavity effusions

Clinical signs associated with hyperkalaemia are most commonly associated with cardiac conduction disturbances and reduced membrane excitability. Atrial standstill and bradycardia are the most consistently reported electrocardiographic changes, although a predictable sequence of events is postulated, starting with narrow peaked T waves and shortening of the QT interval. This progresses to widening of the QRS and loss of the P

wave. The ultimate progression is to ventricular fibrillation. The astute clinician should be aware that individual variation can and will occur.

**Hypokalaemia**

Hypokalaemia is arguably one of the most frequently detected CSED in small animals. Potential causes are too numerous to list and usually result from reduced intake, gastrointestinal losses (vomiting, diarrhoea), renal losses, or intracellular shifts secondary to drug or fluid administration or acid~base disturbances.

**Clinical signs of hypokalaemia**

The effects of hypokalaemia are most commonly seen as abnormalities of cardiac and skeletal muscle and the kidneys. Hypokalaemia causes skeletal muscle weakness, which may be manifested as generalised weakness, cervical ventroflexion, a stiff gait, and a plantigrade stance. This syndrome of muscular abnormalities may be referred to as hypokalaemic polymyopathy and is more common in cats than in dogs. These abnormalities result from an increase in the cell resting membrane potential (i.e., the threshold becomes more negative). Increased creatinine kinase levels and rhabdomyolysis occur due to impaired potassium regulation of skeletal muscle blood flow during muscle contraction and exercise, resulting in ischaemia and cramps. Severe hypokalaemia can result in fatal respiratory paralysis. Cardiac conduction abnormalities may occur as a result of prolongation of the action potential, and low potassium levels predispose to atrial and ventricular tachyarrhythmias, atrioventricular dissociation, and ventricular fibrillation. Common electrocardiographic changes seen with hypokalaemia include ST-segment depression and increased amplitude or inversion of the T wave. With increasing potassium depletion, increased P-wave amplitude, prolongation of the PR interval, and widening of the QRS complex may occur. Hypokalaemia also predisposes to digitalis-induced cardiac arrhythmias.

Renal dysfunction may be seen with hypokalaemia, producing symptoms of polyuria and polydipsia. These symptoms are largely caused by primary stimulation of thirst and reduced urinary concentrating ability. Hypokalaemia impairs collecting tubule responsiveness to antidiuretic hormone and may also disrupt sodium transport in the loop of Henle. Severe potassium depletion also promotes renal ammoniagenesis, which may induce signs of hepatic encephalopathy in patients with concurrent liver disease. Other common clinical manifestations of hypokalaemia include ileus, nausea, vomiting and constipation.

**Calcium disorders**

**Hypercalcaemia**

Hypercalcaemia is relatively uncommon in dogs and cats. Common causes of hypercalcaemia which may be seen in the emergency setting include:

Neoplasia (probably the most common differential in dogs)
hypoadrenocorticism
acute or chronic renal failure
hypervitaminosis D secondary to cholecalciferol intoxication.
Idiopathic hypercalcaemia in cats

Granulomatous disease (cats)

Other less common causes include primary hyperparathyroidism, nonmalignant skeletal lesions (eg. osteomyelitis) and laboratory error.

**Clinical signs of hypercalcaemia**

The severity of clinical signs depend on the magnitude of hypercalcaemia and its speed of onset. Clinical signs are most severe when hypercalcaemia develops rapidly, and as the magnitude of hypercalcaemia increases. Simultaneous disturbances in other electrolytes and acid-base balance (and organ dysfunction secondary to hypercalcaemia) will contribute to the clinical signs. Anorexia, vomiting, and constipation can result from hypercalcaemia by reducing excitability of gastrointestinal smooth muscle as well as from direct effects on the central nervous system. Decreased excitability of skeletal muscle contributes to generalized weakness. Clinically important effects of hypercalcaemia on the heart are not commonly detected, but prolongation of the PR interval and shortening of the QT interval can be detected on the electrocardiogram. Polydipsia and polyuria are common clinical signs in hypercalcaemic patients, as is azotaemia resulting from prerenal and renal factors. Dehydration is another frequent finding and probably represents increased fluid losses secondary to vomiting and polyuria.

**Hypocalcaemia**

Hypocalcaemia is a common abnormality noted in sick animals and occurs more frequently than hypercalcaemia. Clinical studies suggest a prevalence of between13% and 25% in critically ill dogs. Hypoalbuminaemia is one of the most common causes of hypocalcemia. Such hypocalcaemia is usually mild and few if any clinical signs are observed. It is assumed that ionised Ca levels are normal and that total serum Ca levels are low due to a reduction in the protein-bound fraction of serum calcium. Renal failure is the second most common disorder associated with hypocalcaemia, probably due to decreased calcitriol synthesis by the diseased kidney. Animals with chronic renal failure are usually asymptomatic.

Eclampsia, pancreatitis and ethylene glycol poisoning are less common causes of hypocalcaemia, but are more likely to be encountered in the emergency setting and also result in clinical signs. Eclampsia typically occurs between 1-3 weeks postpartum in small bitches and is usually attributed to extreme diversion of Ca into milk production during lactation. Acute pancreatitis can result in deposition of Ca salts via saponification in tissues surrounding the inflamed pancreas. Also, metabolites of ethylene glycol can chelate calcium and become deposited in soft tissues, resulting in hypocalcaemia and loss of organ function. Hypoparathyroidism should also be considered in feline patients that have recently undergone bilateral thyroidectomy or removal of parathyroid gland adenomata. In many cases, the parathyroid glands are only transiently suppressed m their ability to secrete parathyroid hormone.

**Clinical signs of hypocalcaemia**

Most clinical signs seen in association with low calcium concentrations can be explained by an increase in neuromuscular excitability. These can include behavioural changes, muscle tremors, seizures, muscle cramping, and stiffness. Signs tend to be intermittent even when low total calcium concentrations are persistent, and some animals show surprisingly fewsigns despite severe depletion. This maybe a consequence of more chronic development of hypocalcaemia. Acute development of hypocalcaemia usually is associated with more severe signs. Co-existing acid~base or other electrolyte disturbances may alter the nature of the clinical signs observed. Cardiovascular signs of hypocalcaemia may include hypotension, hypocontractility, tachycardias and prolongation of the QT interval.

**Urinalysis**

**NB a urine output of less than 0.5ml/kg/hr constitutes oliguria.

Specific gravity (SG) is used to assess the capacity of the renal tubules to concentrate or dilute glomerular filtrate, and should be interpreted in relation to the patient's hydration status, urine output and urea/creatinine values.

The normal ranges for dogs (1.015 – 1.045) and cats (1.035 – 1.060) can be wide. Low SG occurs in most diseases that display polyuria, due to osmotic diuresis (eg diabetes mellitus, post-obstructional diuresis) or a secondary nephrogenic diabetes insipidus (eg pyometra). High specific gravity invariably occurs as a result of dehydration or severe hypovolaemia. Patients with urine SG < 1.030 and clinical signs of dehydration have strong evidence of renal dysfunction.

Normal urine pH should be 5-7.5 in dogs and cats. Acidic urine may occur due to:

- Meat diets
- Metabolic or respiratory acidosis
- Paradoxical aciduria with metabolic alkalosis
- Urinary acidifiers (methionine, Vitamin C)

Alkaline urine may result from the following:

- Post-prandial alkaline tide
- Cereal diets
- Stale urine
- Metabolic alkalosis
- Distal renal tubular acidosis

Protein content in urine should be related to the presence or absence of haematuria or UTI. Protein-creatinine ratios can be used to quantify the urinary protein loss (normal value <1). Very alkaline urine can result in dipstick positive tests for protein.

Glucose in urine may occur in hyperglycaemic patients as a result of stress-induced exceeding of the renal threshold (cats), administration of glucose-containing fluids, or

diabetes mellitus. If the patient's blood glucose is normal, then some form of proximal tubular defect is likely.

Ketones (beta-hydroxybutyrate, acetoacetate, acetone) in urine are indicative of diabetic ketosis/ketoacidosis. ß-OHB is not usually detected on dipsticks but is metabolised to acetoacetate and acetone, thus erroneously often giving the impression of worsening ketosis despite therapy.

**Assessment of Haemostasis and Coagulation**

| **Primary haemostasis** | **Secondary haemostasis** |
|---|---|
| Petechiae common | Petechiae rare |
| Haematomas rare | Haematomas common |
| Bleeding usually at multiple sites | Bleeding often localized |
| Bleeding usually includes epithelial surfaces | Bleeding into muscles or joints is common |
| Bleeding from cut/venepuncture is prolonged | Delayed bleeding after venepuncture – stops then restarts |

**Specific tests to assess haemostasis**

Tests of Primary Haemostasis

- Buccal mucosal bleeding time
    - Time taken for bleeding to stop from a standardised superficial incision
        - Tests vascular response, platelet numbers & function
    - Reflect upper lip (hold in place with gauze bandage)
    - Make an incision in non-vascular part of mucosa with spring loaded cutting device (Simplate, Technicon) or no 11 scalpel blade
        - Standard width and depth
    - Filter paper is used to absorb blood (do not touch incision, dab below)
    - Time is recorded for bleeding to stop
    - 1.7-4.2 minutes – normal dogs (cats 1-3.2 minutes with sedation)
    - Prolonged times with vWD, thrombocytopenia & qualitative platelet defects
- Platelet number & morphology
- Platelet function – clot retraction is a crude method of assessing platelet function when normal platelet numbers exist
- Platelet aggregation tests
- vWF antigen by electroimmunoassay or ELISA.

**Tests of secondary haemostasis**

- Activated clotting time (ACT)
    - Time taken for whole blood to clot in the presence of a substance (diatomaceous earth) that initiates contact activation of coagulation
    - Pre warm ACT tube to $37^0$C, minimise tissue factor collection when taking blood sample – discard first 0.5 ml blood

- Add 2ml blood to ACT tube – start timing as soon as blood enters tube
- Mix sample by inversion and place in $37^0$C water bath.
- Tilt sample every 10 seconds till first clot is observed
- 60-110 seconds - normal dogs
- Prolongation suggests abnormalities of intrinsic & common clotting pathways. Also prolonged in severe thrombocytopenia & hypofibrinogenaemia

- **Whole blood clotting time**
  - Use glass tubes instead of ACT tubes – add 1ml to each of 2 glass tubes and tilt every 30 seconds till blood has coagulated.
  - Normally 6-7 minutes. Prolonged for same reasons as ACT
- **Clotting tests**
  - Collect sample into sodium citrate anticoagulant, separate plasma and freeze if not to be assayed immediately at $-20^0$C
  - Minimize trauma during collection as trauma can lead to contamination with tissue thromboplastin and activation of coagulation cascade and spurious results.
  - **Prothrombin time**
    - Coagulation disorders of extrinsic (Factor VII) & common pathways (Factors II, V & X and fibrinogen) identified
    - Prolongation relative to laboratory reference indicates a significant deficiency of factors (< 30% normal levels)
    - Prolonged in acquired Vitamin K deficiency, Liver disease, specific factor deficiencies, DIC
  - **Activated partial thromboplastin time**
    - Identifies abnormalities of intrinsic (Factors VIII, IX, XI, XII) & common pathways and is not affected by thrombocytopenia or platelet dysfunction
    - Variation with different reagents – need to establish laboratory ranges in each laboratory
    - Prolongation relative to laboratory reference indicates a significant deficiency of factors (< 30% normal levels)
    - Not useful to detect carriers of haemophilia with 40-60% or higher levels of normal Factor VIII or IX activity

**Tests of Fibrinolysis**

- **Fibrin degradation products (FDP's)**
  - End product of fibrinolysis
  - Special kits e.g. ThromboWellco test, Wellcome diagnostics, Dartford, Kent, UK
    - Can also detect fibrinogen and so false elevations if not all fibrinogen is removed from the sample before testing
  - Healthy dogs FDP levels < 10 μg/ml
  - FDP > 40 μg/ml indicates increased fibrinolysis
  - DIC is main cause of increased fibrinolysis

  - FDP's increased with coagulopathy secondary to Vitamin K antagonism, hepatic disease & thrombotic disease
- Thrombin clot time (TCT)
  - Time taken for citrated plasma to clot when exogenous thrombin & calcium are added
  - Assesses only ability of thrombin to convert fibrinogen to fibrin
  - TCT is prolonged with hypofibrinogenaemia or dysfibrinogenaemia or via inhibition of thrombin e.g. by heparin, FDP's, abnormal serum proteins
- **Fibrinogen**
  - Fibrinogen concentration ranges from 1.5-3.0 g/l in normal dogs
  - Hypofibrinogenaemia with increased consumption in DIC or from advanced liver disease, ? congenital abnormality
- **Antithrombin III (ATIII)**
  - Circulating natural anticoagulant, main inhibitor of thrombin
  - ATIII decreases in hypercoagulable diseases e.g. DIC
  - Several assays available
  - DIC –> ATIII decreased compared to normal pooled plasma
  - ATIII decreases in hepatic disease due to reduced synthesis
  - ATIII decreases in protein losing conditions (PLE, PLN) due to similarity in size to albumin i.e. is lost with albumin
  - Specific factor assays – Contact specialist laboratory

**Summary of haemostatic abnormalities**

| | BMBT | OSPT | APTT | Platelets | Fibrinogen | FDP |
|---|---|---|---|---|---|---|
| Thrombocytopenia | ↑ | N | N | ↓ | N | N |
| von Willebrands | ↑ | N | N / ↑ | N | N | N |
| Haemophilias | N | N | ↑ | N | N | N |
| Warfarin toxicity | N | ↑ | ↑ | N / ↓ | N / ↓ | N / ↑ |
| DIC | ↑ | ↑ | ↑ | ↓ | ↓ | ↑ |

**Peri-operative Cardiac Arrythmias.**

R. Eddie Clutton

Peri-operative electrocardiographic abnormalities are important for several reasons:

- They are common in companion animals.
- Certain forms reduce cardiac output and cause hypotension. Minor arrhythmias may be haemodynamically significant when myocardial disease is present.
- Many benign arrhythmias degenerate into lethal rhythms such as ventricular fibrillation (VF), asystole, or electromechanical dissociation (EMD). Anaesthetists must be able to differentiate malign arrhythmias from benign patterns (and artefacts) understand their cause, and so be able to respond rapidly.
- Some pre-operative arrhythmias result from cardiac disease and if untreated deteriorate critically under anaesthesia. In these cases, therapy is aimed at controlling the underlying cause, not the arrhythmia itself.
- While many anaesthetics are arrythmogenic, others, e.g. acepromazine, are not. Anaesthesia, even when produced with arrhythmogens can suppress rhythm disturbances if these arise from pain, severe anxiety or excessive myocardial work.
- Spontaneously arising intra-operative arrhythmias indicate a deterioration in the myocardial *milieux interieux* as a result of poor anaesthetic management, i.e. deranged blood - gas, pH, temperature and electrolyte values.
- Electrocardiography can assist in determining the 'level of anaesthesia through changes in heart rate and rhythm in response to surgery. Tachyarrythmias often occur when anaesthesia is inadequate, making the ECG particularly useful when neuromuscular blocking agents are used. Bradycardia and bradyarrythmias often result from anaesthetic overdose,

**Pre-operative bradycardia and bradyarrythmias**

Some benign bradyarrythmias, e.g. sinus arrest, wandering pacemakers, are linked to athletic fitness and are inconsequential. However, slow heart rates which do not rise on stimulation or physical exertion warrant investigation because most anaesthetics will cause further slowing. See **table 1.**

**Table 1.** Causes of pre-operative bradyarrythmias and suggested treatment.

| Cause | Example | Response |
|---|---|---|
| Cardiac disease (congenital, ischaemic, traumatic, idiopathic) | canine sick-sinus syndrome<br>3rd degree AV block<br>sino-atrial standstill | Provide chronotropic challenge*<br>Consider transvenous pacing. |
| Hyperkalaemia | Addison's disease<br>renal failure | Treat with $HCO_3^-$, $Ca^{2+}$, insulin - glucose, or peritoneal dialysis |
| Hypothyroidism | | Achieve euthyroidism |
| Polycythaemia | multiple causes | Attempt isovolaemic haemodilution |

**Table 1 continued.** Causes of pre-operative bradyarrythmias and suggested treatment.

| | | |
|---|---|---|
| Hypertension | idiopathic | Give vasodilators |
| | fluid over-transfusion | Provide diuretics |
| | blood, colloid over-transfusion | Phlebotomy |
| Toxaemia | Uraemia | Restore renal function, peritoneal dialysis |
| Idiopathic | | Provide chronotropic challenge* |
| Drugs | Digoxin | Suspend medication, modify dose and provide chronotropic challenge* |

* If the need for surgery is pressing and the aetiology remains undetermined, the heart's ability to increase rate is tested first with atropine (40 $\mu g\ kg^{-1}$ IV) and if that fails, isoprenaline given by infusion (10 - 50 $ng\ kg^{-1}min^{-1}$). A positive response with either indicate the appropriate therapy for managing intra-operative bradyarrythmias. Animals responding to atropine may be treated pre-operatively with oral antimuscarinic drugs like propranthelline. If neither treatment increase heart rate then three options remain: find a means of artificial pacing, postpone surgery or conduct at the subject's peril.

**Pre-operative tachycardia and tachyarrythmias**

The cause of elevated preoperative heart rates or tachyarrythmias (**table 2**) should be investigated because during surgery, even benign rhythms may deteriorate.

**Table 2** Causes of pre-operative tachyarrythmias and suggested treatment.

| Cause | Example | Response |
|---|---|---|
| Anxiety | | Give acepromazine, or other anxiolytic drugs |
| Pain | | Provide analgesia |
| Pyrexia, heat stress | | Investigate cause, give NSAIDs if appropriate |
| Cardiac disease | Congestive failure | Treat |
| | Fixed low output diseases | Treat |
| Hypotension | Hypovolaemia | Restore circulating volume |
| Anaemia | | Examine [Hb] and treat if inadequate |
| Hyperthyroidism | | Render euthyroid |
| Phaeochromocytoma | | $\beta_1$ and $\alpha_1$ antagonists prior to surgery |

Occasional APDs need not be treated unless they arise more commonly than 20 per minute or if they periodically entrain a ventricular response. Vagal manoeuvres, e.g. carotid sinus massage, or ocular pressure are said to occasionally convert atrial tachycardia to sinus rhythm, but the effects is to brief to be of any value and so digoxin alone, or with propranolol is required. Although $\beta_1$agonists are often used for the treatment of pre-operative tachyarrythmias the beneficial vagomimetic and mood-elevating effects of morphine should not be forgotten.

**Intra-operative bradycardia and bradyarrythmias**

The heart rate may slow during anaesthesia due to any of the causes listed in **table 3** and result in a significant hypotension.

**Table 3** Causes of pre-operative bradyarrythmias and suggested treatment.

| Cause | Response |
|---|---|
| Inadequately treated / recurring pre-operative causes | See table bradycardia |
| Anaesthetics | Depends on causative drug* |
| Anaesthetic overdose | Reduce delivered concentration of inhaled drug and ventilate lungs* |
| Hypothermia | End surgery and re-warm; consider gastric or colonic lavage. |
| Hypertension | Consider vasodilators and, or $\beta_1$antagonists |
| Vagal stimulation | Check surgery † |
| Hyperkalaemia II | Hyperventilate, with oxygen, give $HCO_3^-$ (1 mmol $kg^{-1}$ over 10 mins) |
| Severe acidosis | Hyperventilate lungs with $O_2$ and give $HCO_3^-$ (as above) |
| Severe hypoglycaemia ¶ | 50% dextrose |
| Severe hypoxia¶ | Ventilate lungs with 100% $O_2$ |
| Terminal myocardial hypoxia | Initiate CCPR |

*'Lightening' the level of anaesthesia or beginning surgery, it has not already begun, is the simplest treatment for drug-induced bradycardia. Anti-muscarinic drugs reverse heart rate depression caused by high-dose opioids, but their use with $\alpha_2$ - agonist is controversial.

† If bradycardia, bradyarrythmias or AV conduction blocks arise during a specific surgical manipulation, surgery must be suspended and the level of anaesthesia assessed. 'Light' animals should be 'deepened' and surgery continued more gently. If the problem persists then atropine (20 - 40 μg $kg^{-1}$) glycopyrrolate (5 - 10 μg $kg^{-1}$) or isoprenaline (10 - 50 ng $kg^{-1}min^{-1}$) should be given.

**Intra-operative tachycardia**

The heart rate is often raised in the three or so minutes after induction when the electrocardiogram is first attached. However, this is usually short-lived in animals which were anxious before induction and, or induced with intravenous barbiturates. It may remain high in animals given pre-anaesthetic antimuscarinic drugs. Importantly, a persisting tachycardia may indicate undetected hypovolaemia or another physiological derangement (see table 4). Intra-operative tachycardia may be hazardous because it reduces cardiac output and threatens myocardial $O_2$ balance. If sustained for long periods, it may suddenly deteriorate without premonitory signs to a more dangerous arrythmia.

**Table 4.** Causes of intra-operative tachycardia and suggested treatment

| Cause | Response |
|---|---|
| Inadequate anaesthesia or analgesia | Increase inspired concentration, low dose of IV anaesthetic, alfentanil or fentanyl (see table **5**) |
| Hypercapnia | PPV |
| Hypoxaemia | Provide 100% oxygen and ventilate lungs |
| Hypotension, shock' ,septicaemia | Rapid IV fluids |
| Hyperthyroidism | see table C |
| Drugs | Discontinue $\beta_1$ –agonists, if given. See table c. |
| Hyperthermia | Consider abdominal lavage with ice-cold fluids |
| Hypokalaemia | Give KCl; 0.05 mmol $kg^{-1}$over 1 min, repeat if necessary |
| 'idiopathic' tachycardia | See table **5** |

Diagnosis is assisted by noting rate of onset. Inadequate anaesthesia is often clearly associated with surgical manipulations although some procedures, e.g. aural resection, may be intensely stimulating even at deep' levels of anaesthesia. In contrast, slowly worsening blood gas derangements and hypovolaemia cause an insidious tachycardia.

Three 'reflex' responses can be made whenever tachycardia, or tachyarrythmias arise during surgery. First, the 'level of unconsciousness' is evaluated; many surgical; manipulations will cause tachyarrythmias, less commonly bradyarrythmias, in the inadequately anaesthetised animal. If there is a link between surgery and rhythm disturbance, the level of anaesthesia should be deepened and surgery continued with gentler manipulation. If this is unsuccessful a short-acting vagomimetic opioid drug like alfentanil or morphine may be given. Second, several lung inflations with oxygen-enriched gas will usually suppress arrythmias resulting from hypercapnia and, or hypoxaemia. Third, the rapid infusion of fluids often slows elevated heart rates due to hypovolaemia or hypotension, though the response may be slow and slight. If the cause remains unidentified a vagal manoeuvre (ocular pressure or carotid sinus massage) may be attempted. Although it rarely works, it provides time to prepare a pharmacological response.

**Table 5. Drugs used to control intra-operative tachycardia.**

| | Drug | Dose |
|---|---|---|
| 1) | Morphine | 0.25 - 0.5 mg $kg^{-1}$ |
| | *or* Alfentanil* | 1 - 5 $\mu g\ kg^{-1}$ repeated every 5 mins or to effect |
| | *or* Fentanyl† | 1 - 2.5 $\mu g\ kg^{-1}$ repeated every 20 mins or so. |
| 2) | Propranolol | 50 $\mu g\ kg^{-1}$ every 2 min to effect (see wherever) |
| | *or* Esmolol | |
| 3) | Digoxin | |
| 4) | Procainamide | |
| 5) | Neostigmine | |

*Alfentanil (Rapifen; Janssen Pharmaceuticals) † Fentanyl (Sublimaze; Janssen Pharmaceuticals)

**Intra-operative Ventricular Arrythmias**

Tachyarrythmias may deteriorate into more dangerous rhythms. For example, untreated pleomorphic PVDs in time 'coalesce' and become ventricular tachycardia. This in turn leads to ventricular fibrillation and death. Therefore, tachyarrythmias must be treated aggressively using a) the three 'reflex' responses described above, and b) an IV bolus of lignocaine (1 - 4 mg $kg^{-1}$). The beneficial effects may be short-lived (30 - 40 seconds) and although repeated injections (same dose) can be given it is preferable to infuse a lignocaine / saline mixture at a rate of 25 - 75 $\mu g\ kg^{-1}\ min^{-1}$ (Dogs) **lower (cats)**.[1]. If lignocaine is ineffective. intravenous procainamide may be used (1.0 to 2.0 mg $kg^{-1}$). If ectopic beats return procainamide can be infused at 10 - 40 $\mu g\ kg^{-1}\ min^{-1}$. If PVDs persist and deteriorate into ventricular tachycardia then cardioversion with an externally synchronised DC non-phasic defibrillator in conjunction with lignocaine should be attempted.

Note: the appearance of intra-operative PVDs in digitalised animals may point to relative digoxin overdose caused by hypokalaemia. The latter commonly follows respiratory alkalosis, i.e. excessive ventilation. In minor cases a reduction in alveolar ventilation may effect a cure, otherwise potassium chloride (up to 0.05 mmol $kg^{-1}$) should be given IV over one minute. A repeated dose may be necessary.

* Add 12.5 ml 2% lignocaine solution (without adrenaline) to 500 ml saline. Providing the administration set produces 20 drops ml $kg^{-1}$ 1 drop per (animal's body weight in kg) per minute is equivalent to 25 $\mu g\ kg^{-1}\ min^{-1}$

**Table C.Responses to intra-operative tachyarrhythmia**

| Arrhythmia | Response |
|---|---|
| Sinus tachycardia | see table 5 |
| PVDs | lignocaine IV bolus, the infusion, or procainamide |
| Ventricular tachycardia | lignocaine IV bolus, the infusion, or procainamide |
| Ventricular fibrillation | CCPR |
| Electro-mechanical dissociation | CCPR* |

* Attempts to correct EMD with previously recommended $Ca^{2+}$ salts, adrenaline or high-dose steroids * are invariably futile.

# THORACIC IMAGING

## The anaesthetist's perspective

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### Radiography

The basic requirement for good thoracic radiography is the correct positioning of the animal and ensuring that sufficient views are taken without compromising the animal. Often animals requiring thoracic radiography are distressed, may be in pain and difficult to handle. Administration of a mild sedative may improve the animal's disposition and make radiography easier and help to ensure that the radiographs are of diagnostic quality. This obviously presents a challenge as the choice of chemical restraint is critical.

Thoracic studies should be taken at inspiration so that there is good contrast between the air-filled lung and the other thoracic structures. For the lateral view the animals legs should be drawn cranially clear of the thorax. The sternum should be elevated with a foam pad so that the spine and sternum are level on the same plane. Centreing should be at the caudal tip of the scapula. Usually dorso-ventral studies are taken in preference to ventro-dorsal views. Dyspnoeic animals should not be placed in dorsal recumbency as the breathing may become severely compromised. The dorso-ventral view is preferred for cardiac examination as the heart sits on the sternum and has a more natural conformation. It is important that the DV view is positioned carefully and that there is no rotation. The limbs should be drawn cranially and the elbows adducted away from the sternum.

When the animal lies on its side the dependant lung is relatively compressed and the uppermost lung is well inflated. If there is a lesion in the dependant lung it may not be visualised because of the lack of contrast. In order to identify as many abnormalities as possible two opposing recumbent lateral studies should be taken. This is particularly important when screening for metastatic lung disease. Another instance where two views may be advantageous is when an animal is demonstrating pulmonary signs and when pulmonary changes are not visible on initial studies.

**Anatomical variants**

There is considerable variation in the conformation of the thorax between breeds of dogs. The deep chested dogs tend to have the appearance of having an upright heart. The cardiac silhouette in shallow chested dogs occupies a large amount of the thoracic cavity. The Bassett and the Corgi have an anatomical indentation at the costo-chondral articulations. On the dorsoventral view this indentation gives the appearance of a double margin and may be mistaken for pathology.

Fat dogs often have a large amount of fat in the pericardial sac which may elevate the cardiac silhouette from the sternum. In addition the mediastinum which normally lies within the width of the vertebral column on the dorso-ventral view may be widened by the mediastinal fat and simulate mediastinal masses. A sternal fat pad may be interposed between the cardiac silhouette and the sternum. This displaces the heart away from the sternum and this displacement should not be mistaken for an abnormality.

Inspiratory studies in LLR often results in the cardiac silhouette becoming separated from the sternum. This should not be mistaken for a pneumothorax.

The thymus may be seen as a soft tissue triangular shape in the left cranial thorax in young animals and it should not be mistaken for a lung mass.
An anatomical variant associated with chondrodystrophic breeds is a widened mediastinum with deviation of the trachea to the right.
It is clear that a good knowledge of anatomical features and radiographic artefacts are required in order to extract as much information as possible from the thoracic studies.

**Thoracic Abnormalities**

The pleural cavity is a potential space which is not usually identified unless it contains fluid, air or a mass.

**Pneumothorax**

Pneumothorax is a common complication of road traffic accidents. The cardiac silhouette is kept in a central position by the inflated lungs. Lung collapse means the heart displaces into the dependant hemithorax when the animal is placed on its side. The separation of the heart from the sternum is a cardinal sign but not an exclusive indication of pneumothorax.

- The lung margins may be identified separated from the costo-vertebral and rib margins.
- Retracted lung margins are usually well defined
- The lungs have an increased opacity
- Lung markings are absent in the affected pleural space

Overexposed radiographs will often prevent a subtle pneumothorax being seen.

On the dorso-ventral view it is important that skin folds are not mistaken for lung margins, this can be difficult in deep chested dogs.

Tension pneumothorax is a life-threatening condition. This occurs when air enters the pleural cavity and is unable to escape. It may be unilateral in which case the cardiac silhouette is grossly displaced to one side (mediastinal shift). If the condition is bilateral the diaphragm is pushed caudally and its normal rounded contour becomes flattened. The lungs are seen as relatively opaque structures in the perihilar region. Occasionally so called 'tenting' of the costo-diaphragmatic margins may be seen. This occurs when the diaphragm is displaced so far caudally that the muscular insertions become reversed and are identified.

**Pleural fluid**

If the volume of fluid is small, right and left lateral studies may be useful.

On the dorso-ventral view the cranial lung lobes may be completely collapsed if there is a large volume of fluid present. The first place to identify fluid is in the costo-diaphragmatic recess.

The identification of small volumes of fluid often requires a ventro-dorsal study. Causes of fluid in the pleural cavity are chyle, blood, neoplasia and transudates associated with systemic disorders such as cardiac or hepatic disease. Differentiation of these fluids is not possible using radiography alone. There are many causes of the thoracic fluid and thoracocentesis is advisable to establish cytological details and for bacteriological or fungal culture and sensitivity.

- The lungs become retracted from the rib margins
- The lungs are seen as a scalloped undulating lung border.

- The fluid dissects along the pleural fissures which are clearly outlined.
- The cardiac silhouette and diaphragmatic outline are obscured by the fluid.
- The trachea may be displaced dorsally.
- Fluid will be seen in the diaphragmatico-lumbar recess. It should be remembered that in the cat this recess does not extend to the vertebral margins.

Thoracic drainage by means of diuresis may be rewarding and further radiographs taken 48 hours later. If the fluid volume has not reduced sufficiently at this stage aggressive drainage using a thoracostomy drain is advisable as pleural masses may be identified.

Ultrasonographic examination may be useful to examine the pleural cavity while fluid is present and pleural masses or a diaphragmatic hernia may be seen. Aspiration of the fluid under ultrasound guidance is often advantageous.

**Lung lobe torsion.**

This is an unusual condition and may be associated with pleural fluid. It is unclear whether the lung initially twists around the bronchus because it is floating in fluid or whether the torsed lung produces the fluid. If the lung becomes torsed passive congestion and transudation ensues and pleural fluid builds up. Identification of the affected lung in the early stages is possible as the fluid tends to collect around a single lobe which is radio-opaque due to consolidation.

- The lung margin is rounded and the bronchus may be identified travelling in an abnormal direction.

- The right middle lobe and cranial portion of the left cranial lobe are the lobes that are more commonly affected.
- Following thoracocentesis the lung lobe fails to expand and its abnormal contour and bronchial architecture may be identified.

**Pleural thickening**

This may be seen in older dogs as an ageing phenomenon due to fibrosis. It may also be seen as a sequel to pleural infection or haemorrhage.

The fissures are seen as thin radio-opaque lines along the lung margins traveling from the lung periphery diagonally towards the mediastinum.

**Pleural masses**

These are rare and include mesotheliomas, pleural fluid is the usual radiological feature. Ultrasound guided fine needle aspiration is possible.

**Mediastinum**

The mediastinum is usually the width of the thoracic vertebra on the DV view. In some breeds it may be up to twice the width of the vertebral bodies.

**Pneumomediastinum**

The normal air filled trachea is no longer clearly seen due to the presence of free air in the mediastinum. Free air outlines the cranial mediastinal structures such as the brachiocephalic trunk and cranial vena cava. The intrathoracic aorta may be well defined and air may dissect caudally into the abdominal cavity. The causes may include trauma to the thorax or neck and occasionally it is identified in cases of paraquat poisoning.

**Mediastinal fluid**

Mediastinal fluid may be present for a variety of reasons including oesophageal rupture, trauma and heart failure.

Fluid in the mediastinum outlines the fissures from the mediastinum laterally in a reverse direction to the pattern seen with pleural fissures. This pattern has been likened to the branches of a christmas tree. On a dorso-ventral view the fluid pools in the sternal region and the cardiac silhouette may be poorly defined.

**Mediastinitis**

Infection of the mediastinum may occur as a result of an oesophageal perforation. This is usually due to a foreign body but may be seen in some traumatic cases. Fluid may be seen in the fissures extending from the mediastinum laterally. The mediastinum may be widened. If the infection is encapsulated a mediastinal mass be identified.

**Mediastinal masses**

Cranial mediastinum:-enlargement of the mediastinum is identified by widening of the mediastinum in lateral and dorso-ventral directions. The trachea may be deviated dorsally or ventrally depending on the origin of the mass. The causes may include thymic or oesophageal abnormalities, lymphadenopathy, neoplasia, haemorrhage or infection. Lateral deviation may also be noted. Oesophageal displacement may also be identified. The cardiac silhouette is displaced caudally. It may not be clearly outlined if the cranial mass extends caudally and contacts the cardiac border. One way of identifying where the heart is lying, is to identify the tracheal bifurcation which occurs just dorsal to the heart base. Ultrasonography is helpful in

delineating the size and echogenic characteristics of the mass but an FNA or biopsy (under u/s guidance) is required for a cytological diagnosis.

**Mediastinal shift**

Displacement of the mediastinum is usually associated with lung or pleural pathology. Causes include-pneumothorax, lung lobe collapse or removal, and diaphragmatic hernias. Rotated studies can simulate a mediastinal shift.

**Trachea**

The trachea lies within the cranial mediastinum and is identified as an air filled linear tube coursing from the thoracic inlet towards the heart base. It is surrounded by a soft tissue area which comprises the cranial mediastinum and all the structures contained within it. The trachea usually diverges from the vertebral column forming an acute angle. In some breeds the trachea may parallel the spine e.g. barrel chested dogs such as the corgi. Mineralisation of the trachea is usually an incidental finding.

**Tracheal perforation**

This may occur following a traumatic injury or direct penetration such as a dog bite or laceration. Pharyngeal perforation is also a common sequelae to stick injuries.

- Air is seen dissecting through the soft tissue fascial planes
- The lumen is narrowed and
- There may be disruption of the tracheal rings
- Tracheal stricture may occur as a sequelae.

**Tracheal/main stem bronchus avulsion**

The normal tracheal shadow is disrupted within the mediastinum.

**Diaphragm.**

The two crura of the diaphragm should be seen. On the right lateral study they are parallel to each other. On the left lateral recumbent view they intersect in the mid-thoracic region. On the dorso-ventral view the diaphragmatic outline is a smooth curve. On the ventrodorsal view sometimes the two crura are seen travelling laterally from the vertebral origin and the tendinous portion is superimposed centrally. This pattern may look like the leaves of a shamrock.

**Diaphragmatic Rupture/Hernia.**

Disruption of the diaphragm may occur as an acquired or congenital problem.

**Acquired diaphragmatic rupture.**

If the traumatic injury was in the recent past then the disruption to the diaphragmatic outline may be identified. Often the injury may have occured many months previously and the presence of intra-thoracic fluid makes diagnosis slightly more difficult. The fluid arises from the presence of the liver within the thorax. The liver becomes congested as the diaphragm constricts the tissue. Passive transudation occurs and fluid builds up in the pleural cavity. It is the fluid compressing the lungs which accounts for the exacerbating clinical signs. A diaphragmatic rupture should always be

considered in the differential diagnosis when pleural fluid is identified, even without the definite history of trauma.

Abdominal contents may be seen in the thoracic cavity. Often the abdominal radiograph holds the key to the diagnosis. Most diaphragmatic ruptures involve cranial displacement of the liver. The liver may not be seen in the thorax if it is surrounded by fluid. However if the liver is displaced cranially then the gastric silhouette will also be displaced cranially. This means that careful analysis of the abdominal cavity is advisable. If necessary as little as 2-3mls of barium may be given, even to the most distressed dog to outline the position of the stomach.

**Congenital diaphragmatic hernia**/peritoneopericardial hernia.

This condition is seen in cats but is rare in dogs. Young adult cats often have a history of being normal or slightly unwell with a variety of symptoms. The cardiac silhouette is enlarged but well defined. Abdominal contents such as the intestines may be seen within the pericardial sac. If the liver lies within the pericardial sac then the silhouette is enlarged and has an homogenous opacity. However the cardiac silhouette will be seen to be continuous with the diaphragmatic outline. The hepatic silhouette will not be clearly outlined in the abdomen and the stomach will not lie in a normal position.

**Thoracic wall**

**Vertebrae**

Congenital anomalies such as hemivertebrae or kyphosis may cause distortion of the dorsal aspect of the thorax. Fractures of the vertebrae may be seen and callus formation observed.

**Ribs**

**Fractures** are common as a result of trauma. They are often difficult to find and careful examination of the rib margins is required. Soft tissue swelling and the presence of a localised fluid opacity may indicate the site of the lesion. Subcutaneous air may be present at the site of a fracture.

**Neoplasia** of the ribs results in the so called 'iceberg effect' when the majority of the lesion lies within the thorax. The lungs are displaced away from the thoracic wall and fluid may be seen in the pleural cavity.

**Sternum**

Fracture:- Sternal fractures may be seen following trauma.

**Pectus excavatum.**

This is often seen in cats and sometimes in dogs.

There is dorsal deviation of the sternum at various points along the length of the sternum. In severe cases the caudal half of the sternum may curve dorsally towards the vertebral column. Depending on the degree of the displacement there may be displacement of the cardiac silhouette dorsally and to one side of the thorax.

**Osteomyelitis**

Infection may result from direct penetration from a foreign body or as a complication of a sternal fracture with sequestration. Variable amounts of periosteal reaction and bone erosion may be seen. There may be a soft tissue swelling externally and a fluid or soft tissue opacity in the overlying intrathoracic region. The cardiac silhouette and lung may be displaced.

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