

### LESION DESCRIPTION/ RECOGNITION & INTERPRETATION

The ability to recognise an abnormality with the subsequent progression through consideration of the differential diagnosis to ultimate arrival at a diagnosis where possible is one of the cornerstones of veterinary diagnosis.

This is greatly facilitated by an ability to communicate the nature of this abnormality in a sound and scientific manner. Use of standardised terminology further facilitates communication between professionals.

### ELEMENTS OF DESCRIPTION

**(A) EXAMINE AND DEFINE THE SPECIMEN.** Where possible:

- 1) Identify the **species** (this information may already be given to you)
- 2) Identify the **tissue** or organ

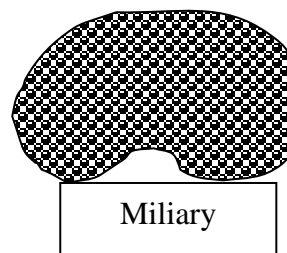
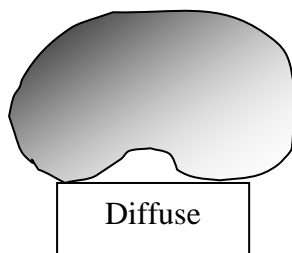
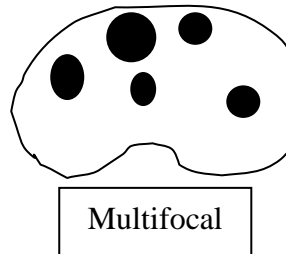
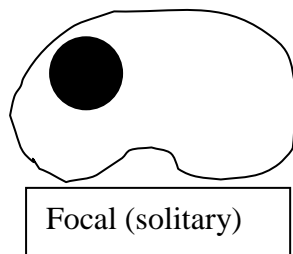
**(B) DESCRIPTION OF THE PATHOLOGICAL CHANGES WHICH ARE PRESENT.** This is the second and crucial component and broadly speaking, can be thought of in terms of '**Where**' is the lesion and '**What**' is the lesion. A useful concept here is to try and imagine that you are giving a verbal or written description to create a 'mental picture' of what you are looking at to someone who is unable to see the specimen.

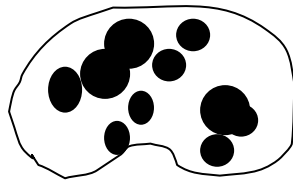
**WHERE is the lesion?:**

- 1) **Focal** (involves part of the tissue or organ)
  - a) **Solitary**
  - b) **Multifocal**
- 2) **Diffuse** (involves whole organ or whole of a specified area)

**Other useful terms**

- 3) **Multifocal to coalescing** a useful term to use when multifocal lesions 'join-up' with each other
- 4) **Segmental**: used for describing a portion of a tubular organ e.g. intestine
- 5) **Miliary**: used for multiple small lesions





Multifocal to coalescing



Segmental

### What Is The Lesion?:

- 1) **Size:** linear measurement in 2 or 3 dimensions. In addition, in certain circumstances it may be useful to indicate how much of the organ or tissue is involved e.g. '90% of the left cranial lung lobe is affected'. Try to develop an ability to estimate size.

In certain circumstances (usually during full post mortem examination), it may also be possible to assess:

- weight (e.g. of an organ or lesion). *[NB. If organs are being weighed, the information is meaningless unless it is given relative to the weight of the animal]*
  - volume (of fluid). Again only relevant if considered in relation to size of animal.
- 2) **Shape** e.g. circular, oval, nodular
    - can also include comments on borders (well/ poorly defined/ demarcated, regular, smooth, ragged)
    - and on surface e.g.

<b>Flat</b>	
<b>Elevated</b>	
<b>Depressed</b>	
<b>Umbilicated</b>	
<b>Pedunculated</b>	
<b>Sessile (broad based attachment)</b>	

- 3) **Colour** – primary colours plus grey, brown, black, white etc.; light/dark; shiny or dull

- 4) **Consistency** – by gentle and *careful* palpation

- type: e.g. soft, firm, hard
- degree e.g. slight, moderate, marked
- cohesion e.g. friable, elastic

More than one term may apply to a single lesion e.g. firm with a soft, friable core

- 5) **Odour** – if appropriate

- 6) **Any special features** e.g. free fluid, blood, exudates etc.

- 7) **Examine the cut surface** if available and note:

- the consistency, the amount of blood flowing from cut surface, any capsule and borders

## (C ) DRAW CONCLUSIONS AND FORMULATE A DIAGNOSIS.

### Context for Veterinary Practice

In addition to being important in terms of adopting a logical approach to the assessment of lesions and their likely relevance, development of this skill is very important when submitting samples to pathology laboratories for diagnosis. Most commonly, this could be in one of two situations:


- 1) When submitting surgical biopsy specimens to a laboratory for diagnostic purposes; especially in instances where it may not be possible to submit the entire lesion. In such cases, diagrammatic representation of the lesion indicating the site the submitted samples originated from is extremely helpful and is likely to improve the quality of the information you gain from the biopsy. Good diagrams often save a lot of description.
- 2) When communicating post mortem findings to a pathologist or other professionals e.g. when requesting second opinion; in court cases etc.


### PATHOGENESIS OF LESION DEVELOPMENT AND DIFFERENTIAL DIAGNOSIS

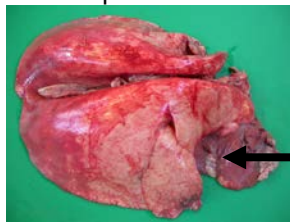
We will now move on to consider how lesions develop and what interpretations it is possible/ reasonable to make based on recognition of particular features. The following are broad generalisations based on the most common associations - as with all things in science interpretation not always 'black and white'; experience is also an important factor.

*(Be aware that some lesions can masquerade as others e.g. tumours vs. granulomas)*

### INTERPRETATION BASED ON LESION DISTRIBUTION


Few broad generalisations can be made here, however it is useful to remember that if lesions are evenly spread throughout an organ, it implies 

In the case of lungs, the typical 'anteroventral' distribution of lesions seen in association with bronchopneumonia is consistent with 



Anteroventral lesion  
distribution

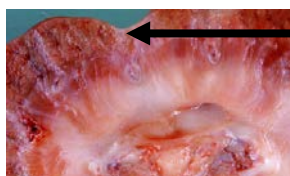
### INTERPRETATION BASED ON LESION SHAPE

**Raised lesions:** Simplistically, a raised lesion implies 



Multifocal raised lesions (sheep  
lungs; *Muellerius capillaris*)

**Depressed lesions:** Implies that something is missing or has been lost. Examples include necrosis (once the active phase of necrosis has resolved, traction on adjacent tissue by scar tissue can create an irregular, disrupted shape). Further examples where something can be missing or lost are: atrophy or e.g. in the lung, collapse (lack of air).



Irregular renal cortex  
due to scar tissue

**Flat lesions:** area neither raised or depressed (but are recognised by colour change). Can imply an acute process in which there has not yet been time for cells/ fluid to accumulate or for cells to be lost.

**Demarcation:** With regard to neoplasms, benign tumours are often well demarcated because they grow by expansion; this may or may not be the case for malignant tumours. With regard to inflammatory lesions, well demarcated inflammatory lesions are often surrounded by fibrosis (therefore chronic) whereas poorly demarcated inflammatory lesions are often more acute.

### INTERPRETATION BASED ON LESION SIZE

This is particularly important if for example you are submitting part of a lesion to a laboratory for histopathology (see lecture 2). Assessing the amount of an organ that is involved can also be important in determining the likely functional significance of a lesion e.g. one small abscess in the liver is likely to be of no significance compared to complete hepatic fibrosis associated with chronic hepatitis affecting the whole organ.



Bovine fascioliasis -  
diffuse



Spleen – solitary chronic abscess  
(incidental)

### INTERPRETATION BASED ON LESION COLOUR

The normal colour of organs and tissues is a net result of the relative amounts of e.g. parenchyma, connective tissue, blood, fat, other pigments. The following is a list of some colour changes which may be seen and their interpretation:

**Red or red/ black:** common colour usually due to increase in amount of blood. This change may indicate haemorrhage or congestion. Significant haemorrhage is usually very dark (haemoglobin depleted of oxygen). Due to the colour contrast, this colour change is most obvious in light coloured tissues (e.g. brain, lung) whereas it is more difficult to see in e.g. spleen. If this colour change is seen in a nodular lesion, consider a haematoma or vascular lesion (or abscess/ inflammation with haemorrhage).

**Black or Brown/ Black:** melanin, exogenous carbon, putrefactive bacteria, haemosiderin. Examples

- If the lesion is a mass, consider a melanin containing neoplasm (melanoma)
- If black colour and not raised, consider non-neoplastic accumulation of melanocytes (melanosis). [*Common in pigmented breeds of sheep – e.g. in tunica intima of aorta*]
- PM change: hydrogen sulphide from GI bacteria can → PM black discolouration (known as pseudomelanosis - see later lecture)
- Haemosiderosis (brown to golden brown). Implies old/chronic congestion or haemorrhage

**Green:** usually bile pigment. Often seen post mortem in areas of liver and intestine adjacent to gall bladder. Also some fungal pathogens e.g. of the respiratory tract have a green → green/black colour.

**Yellow:** e.g. fat, bile pigment (bilirubin), fibrin, cellular exudates, neoplasms. A diffuse yellow colour may be due to icterus (bilirubin) or lipidosis.

**White:** similar to above. e.g. exudates, neoplasia. Also connective tissue (fibrous, cartilage, bone).

**NB** Despite these generalisations, dark colours can obscure changes associated with light colours.

### INTERPRETATION BASED ON LESION CONSISTENCY

**Fluid:** Possibilities for fluid are: oedema (clear), serofibrinous (contains fibrin and implies inflammation), urine (clear plus distinct odour), turbid fluid (indicates cellular elements and may be due to inflammation, lymphatic or neoplastic effusion), blood (dark red).

**NB** recent haemorrhage into body cavities is thick often containing clots cf. serosanguinous fluid (blood tinged oedema) which is often seen at necropsy.

Fluid can also accumulate post mortem.

**Soft:** the normal consistency for many tissues – especially those with little stroma or loosely organised.

**Firm:** normal in fluid-poor, cell-rich tissues. Also many inflammatory or proliferative lesions will feel firm. Addition of scar tissue will increase the firmness of an organ.

**Hard:** implies mineral density e.g. cartilage, bone, mineral salt deposition

### DIFFERENTIAL DIAGNOSIS

At its most basic, in some instances, the diagnosis is straightforward based on the gross appearance of a lesion e.g. trauma associated lesions such as fractured femur or ruptured spleen. With experience, there are also disease entities which have pathognomonic lesions (*definition = characteristic indicative of a particular disease*). Examples which you will become familiar with in the practical classes include *Muellerius capillaris* and *Dictyocaulus filaria* (lungworm) infections in sheep; fascioliasis (liver fluke) in sheep and cattle.

Interpretation becomes more difficult when the morphological appearance of a lesion is not pathognomonic for a particular entity; in such instances it is important to develop the ability to consider what the reasonable options would be and how these different entities might be further differentiated.

For example, a solitary mass in an organ can be either a focus of inflammation (including abscess, granuloma), a neoplasm, a haematoma or a cyst. Examination of the lesion based on the categories discussed previously should allow you to reach a 'most-likely' identity or at least a short list of possibilities. In certain cases however, differentiating between some of these categories may require histology for a definitive diagnosis.

Despite these broad generalisations, always bear in mind that lesions may not be as they seem. e.g. Some tumours may present with ulceration as a dominant feature rather than proliferation. In addition to unpredictable presentations, another point to remember is that often there may be more than one disease process in progress at any given time e.g. the presence of necrosis, inflammation and even abscess formation within the bulk of a tumour is not uncommon.

## APPENDIX

### Suggested cataloguing of material seen in Pathology Practical classes

*The material you will see in these classes is valuable.*

This may be the only time you ever see specific examples of diseases 'in action' and in the case of any material you see which has come from the hospitals (small animal, farm animal, equine), remember that these cases may have been family pets or may have gone through protracted treatment – you are privileged to have access to this material and therefore it is your responsibility to derive optimum benefit from it.

Although not meant to be prescriptive, the following is therefore a suggested mechanism for cataloguing the cases you see in the morbid anatomy classes.


Date		Organ and Species	
Gross Description		Diagram	
(Predicted) Histological Features			
Diagnosis or Differential Diagnosis			
Aetiology (if appropriate)			
Consequences (if appropriate)			

## **POST-MORTEM EXAMINATION – theory and practice.**

**These notes should be studied in conjunction with the videos available on EEVeC in the virtual post-mortem room**

### **INTRODUCTION**

Unlike the medical profession, many veterinary surgeons perform their own post-mortem examinations (PMEs). The relevant RCVS Day One Competency reads:


<b>The new veterinary graduate should be able to: ‘Perform a basic gross post mortem examination, record details, sample tissues, store and transport them’</b>

### **Preparation and Equipment**

#### **1. Clothing**

Protective clothing necessary, especially if a large animal PME. Dedicated overalls, plastic/rubber apron, boots. Disposable or other gloves. Face masks may be needed.

#### **2. Location**

Discrete site; washable and disinfectable; space and ventilation.

#### **3. Personnel**

Is assistance available? Large animal PMEs need assistance. Legal or insurance cases require a colleague for corroborating findings.

#### **4. Documentation**

Consider all information (clinical findings, imaging data, laboratory results). **Confirm that the cadaver is the correct one.**

#### **5. Equipment.** Do not “double-up” surgical instruments.

- Knives – large boning knife; post-mortem scalpels.
- Forceps – rat-toothed and smooth, large and small.
- Scissors – straight, blunt-ended, large and small. Gut scissors?
- Rib cutters – garden loppers or secateurs.
- Saws – hacksaw type, large and small. Oscillating saw?
- Microbiological sampling – swabs, sterile containers. Culture bottles?
- Tissue/cytology sampling – fixative containers, syringes, hypodermic needles, microscope slides Other – string, sutures, tie-on labels, pencil/marker pen, notebook/Dictaphone, camera. Cork or polystyrene boards are useful for pinning out small cadavers/birds.

### **PM Procedure (Prosection)**

**The aim** - standard, thorough examination of organs and tissues of the body systems. A careful and complete examination following a standard protocol frequently reveals unexpected but significant information even when the clinical history does not indicate diseased states.

*The general approach –*

- Check it is the correct animal (source and signalment).

- Dorso-ventral recumbency.
- Assessment of organ systems during and following removal.
- Sample selection as appropriate.
- Interpretation and report.

## THE PROSECTION

- 1. External examination**
  - Any external features; bodyweight and condition.
- 2. Initial dissection**
  - Axillary and inguinal incisions.
  - Join incisions on both sides and reflect skin cranially to mandibular symphysis and caudally to pubis. In males, reflect penis, remove testes.
  - Examine superficial lymph nodes.
- 3. Neck region**
  - Incise inter-mandibular muscles and reflect tongue ventrally.
  - Incise the soft palate and hyoid apparatus. Examine tonsils, R/P lymph nodes and thyroids.
  - Dissect the oesophagus and trachea together down to the thoracic inlet.
- 4. Opening abdomen and thorax**
  - Incise linea alba near xiphisternum. Extend incision to xiphoid cartilage and down to pubis.
  - Incise abdominal wall around costal arch to display viscera *in situ*.
  - Remove ventral sternum by cutting ribs from costal arch to thoracic inlet bilaterally and around diaphragm. Dissect mediastinal and pericardial sac attachments from sternum, to display thoracic viscera *in situ*.
- 5. Removal of thoracic viscera**
  - Sever connective tissue attachments at thoracic inlet.
  - Separate oesophagus and trachea. Transect aorta and common vena cava at diaphragm.
  - Remove heart and lungs leaving oesophagus connected to stomach.
- 6. Removal of abdominal viscera**
  - Locate descending colon; ligate and transect near pelvic inlet.
  - Small animals – cut root of mesentery to SI and remove intestines, stomach, liver and spleen as a “unit”.
  - Large animals – remove spleen and stomach with intestines separately, leaving liver temporarily *in situ*.
  - Remove liver and adrenals.
  - Remove ventral pelvic bones to expose tissues in pelvic canal.
  - Free kidneys, ureters and bladder and remove together with terminal colon/rectum, genitalia. Make encircling incision around anus and vulva to release the “urinary and genital tracts intact. In large species remove reproductive organs separately.
- 7. Musculoskeletal systems**
  - Open major limb joints, plus others as appropriate.
  - Compare muscle masses as appropriate.
  - Examine bones, refer to radiographs. Femoral bone marrow.
- 8. Nervous System**
  - Remove head at atlanto-occipital joint unless lesion is at that site.
  - Access brain via removal of calvarium and meninges. Leave pituitary *in situ*.
  - Access spinal cord by removing dorsal vertebral arches.



## 9. Special senses

- Eyes, ears, nasal passages and sinuses as appropriate.

## 10. Individual organ dissections

- Heart – note cardiac silhouette and weight. Use standard dissection technique (see cardiovascular notes).
- Lungs – open airways and gross sections of parenchyma.
- Gastro-intestinal – microbiology/toxicology samples before dissection. Unravel intestines prior to opening.
- Urinary – open bladder and collect contents for urinalysis as appropriate. Assess kidneys and gross section (longitudinal).
- Genitalia – open uterine horns and vulva. Gross section prostate, testes, ovaries.
- Other lesions/sites – as necessary.

## Species Differences

The basic prosection technique can be applied to all the veterinary species including birds and quadruped reptiles. Tortoises require removal of the plastron. Be prepared to adapt the basic technique to accommodate very large animals or unusual lesions (e.g. large tumours).

## Sampling

- Tissues for histopathology – sensible sizes; fix in 10% buffered formalin – rule of thumb = 10 volumes to 1 volume of tissue; plastic leak-proof containers; record sites and type of tissue – use of diagrams.
- Toxicology samples – tissues, fluids or viscera contents. Freeze (-20°C) in plastic leak-proof containers.
- Microbiology samples – swabs; fluids, viscera contents or tissues in individual leak-proof containers. Label, especially if potentially zoonotic and double-bag. For virology, contact laboratory for advice.
- Parasitology samples – most laboratories carry out parasitic egg counts on faeces.
- Cytological samples – laboratories prefer fluid samples as fresh as possible, rather than smears; otherwise rapidly-dried smears or impressions (bone marrow). Label slides with pencil.
- Photographs – if a legal case.

## Reporting

- Logical record of significant findings – use a check list of organ systems.
- Record significant macroscopic diagnoses, in order of importance at end of report. Histological results may be pending – indicate if so.
- Comments – the opportunity for the pathologist to record an opinion as to the significance or time course of lesions. Important if the pme has a legal involvement.

**“Cosmetic” PM Examination: Not all organ systems can be fully examined.**

It is important that you watch the PME videos available in the Virtual PM room on EEVeC

## POST MORTEM CHANGE AND INCIDENTAL FINDINGS

The ability to interpret and assign a relative significance to lesions which are seen on clinical examination, during surgery or at post mortem examination is extremely important.

We will discuss these issues in the 2 categories of

- post mortem and agonal changes
- incidental findings: changes seen clinically, during surgery or PME which may be misinterpreted

## POST MORTEM CHANGES

It is vital that cadavers awaiting post mortem examination are refrigerated as soon as possible. This is particularly important in animals with a large quantity of abdominal viscera (ruminants, horses) since the core temperature takes many hours to fall after death. The problem is exacerbated in sheep by the insulating effect of the fleece.

Note however that freezing should be avoided if at all possible since the formation of ice crystals damages tissue hampering subsequent histopathological interpretation.

## Summary of Post Mortem Changes and Terminology

FEATURE	NOTES
Algor mortis	Cooling of body after death (can aid in estimating time of death)
Rigor mortis	<i>See below</i>
Postmortem clotting of blood	Blood coagulation in vessels. Care re. differentiating from ante mortem thrombi
Hypostatic congestion	Effect of gravity. Often evident in lungs and kidneys.
Pseudomelanosis	Green/ black discolouration due to conversion of iron to iron sulphide by GI bacteria.
Autolysis	<i>See below</i>
Biliary imbibition	Pigment imbibed → liver and any organs in contact e.g. GI tract
Putrefaction	Can → rupture of organs. Presence/ absence of associated inflammation and haemorrhage should clarify if ante mortem or post mortem rupture. <i>See below</i>
Emphysema	Invasion by gas producing bacteria

## Rigor mortis

Begins approximately 2-4 hours after death. Rigidity of skeletal muscles begins to develop (rigor mortis).

Normal Muscle Physiology:

- *Myosin* (thick) filaments and *actin* (thin) filaments.
- Slide past each other during muscular contraction due to cross-bridges between the two filaments.
- ATP is needed for actin and myosin to release

After death:

- Since ATP is not produced after death, fibres undergo sustained contraction
- Compounded by influx of calcium due to failure of membrane pumps
- May last for a few hours to a few days. Depends on:
  - Glycogen content of the muscle (fuels ATP production in the first hours - less glycogen, faster onset),
  - Temperature (high temperature - faster onset. e.g. malignant hyperthermia animals may develop rigor within 1 hour!),
  - pH of muscle (high pH delays or inhibits it - this is why pyrexia animals sometimes do not develop it or do so late, despite the high temperature).
- Reversed by autolysis

### **Autolysis (Self-digestion)**

This is the process whereby the release of intracellular lytic enzymes (from lysosomes) results in self digestion of tissues. An important point to note here is that the process of autolysis results in changes in the cytoplasm and nucleus of cells which resemble those seen in necrosis. Differentiating antemortem necrosis and post mortem autolysis therefore rests on the fact that in autolysis, there is no associated inflammatory or cellular response. Accurate clinical history is also important additional information.

Not all tissues of the body undergo autolysis at the same rate. The mucosa of the stomach and intestine autolyse very rapidly as does the pancreas (as a result of the digestive enzymes it contains) and renal tubules. Muscle and skin retain structure for much longer. In addition to autolysis, bacterial proliferation after death can result in rapid decomposition of organs.

### **Putrefaction**

Occurs when dead tissue is invaded by anaerobic organisms such as Clostridia. Tissue turns green/ brown due to combination of haemoglobin breakdown and formation of hydrogen sulphide.

### **AGONAL CHANGES**

Agonal changes occur around the time of death/time of irreversible circulatory failure. This often leads to vascular congestion e.g. in the lungs, and may be exacerbated by hypostatic changes causing pooling of blood in dependant sites (resulting in asymmetric congestion – see hypostatic congestion above). Kidneys, liver and pancreas may similarly be affected by vascular congestion. The spleen is particularly susceptible to extreme congestion relating to barbiturate euthanasia (also seen in animals dying under anaesthesia). Another common barbiturate associated change is that of crystal deposition on the endocardium of the heart.

Agonal regurgitation of GI contents can also occur resulting in food material being present in the airways and possibly alveoli.

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### **INCIDENTAL FINDINGS**



This category includes features, which may be noticed either at exploratory surgery or at post mortem examination. Either way it is important to have an appreciation of their relative significance.

#### **Neoplasia or not?**

- **Nodular Hyperplasia.** The definition of hyperplasia was given earlier in the course and should be reviewed. Nodular hyperplasia occurs when hyperplastic nodules of tissue develop in organs (most notably the liver, spleen, adrenal and pancreas of older dogs). The lesions comprise multiple (occasionally single) nodules of well-differentiated cells within the tissue and are important as they may be mistaken for genuine neoplasms.
- **Inflammatory lesions:** in some situations, inflammatory lesions can resemble a neoplastic process as a result of influx of inflammatory cells, combined fibrosis and reactive hyperplasia.

#### **Neoplasia – significant or not?**

The incidence of genuine neoplastic processes increases with age – many of these are benign and can therefore often be considered in the 'incidental' category. Nevertheless, the consequences of a benign lesion very much depend on its location (*example 1*) and whether or not it predisposes the animal to any other condition (*example 2*).

Example 1	Example 2
	

### Other Incidental Age –Related Changes

- Bones, joints, connective tissues all lose flexibility and become more rigid
- 'Wear and tear' to joints
- Splenic siderofibrosis - Yellow, dry encrustations on capsule of the spleen- thought to represent sites of previous local haemorrhage with subsequent deposits of Fe, Ca and fibrosis.

### Other Organ Specific Changes

#### LUNGS

**Anthracosis:** in polluted areas, inhaled carbon particles phagocytosed by alveolar macrophages. Can become visible as multiple small black foci in lung tissue. In addition as the macrophages then travel to the tracheobronchial lymph nodes, the medulla of these nodes can become black.

#### Vasculature

**Arteriosclerosis:** common but usually incidental. Degeneration and proliferation in the arterial wall results in loss of elasticity and hardening. Most often evident around branches of arteries e.g. from abdominal aorta.

(Atherosclerosis): very important in man but not common in animals except e.g. dogs with hypothyroidism and hypercholesterolaemia. Here the artery walls are thickened due to accumulation of cholesterol.

### Drug Associated Changes

#### Barbiturates

##### 1. Local reaction

##### 2. Barbiturate crystals

##### 3. Splenomegaly

This is most prominent in dogs and horses following either barbiturate euthanasia or anaesthesia. The spleen can be massively enlarged making the capsule fragile. The congestion also disrupts the normal histological appearance of the spleen.

#### Steroids

Long term steroid use can lead to 'steroid-induced hepatopathy'. Pathogenesis: glucocorticoids induce the enzyme glycogen synthetase leading to **increased storage of glycogen** within hepatocytes. The hepatocytes in midzonal areas are often preferentially affected and can be up to 10x normal size.