

A photograph of a gross pathology specimen, likely a liver, showing a reddish-brown color and a smooth, glistening surface. The specimen is placed on a blue, textured background. The text is overlaid on the image in yellow boxes.

Gross Pathology

Description and Interpretation

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These notes are intended to supplement the fresh specimens shown in gross pathology laboratories for VETM 3450, but do not replace the need to attend these wet labs. Developing competence in the skill of pathologic description and interpretation needs practice, which can only be achieved by palpation and observation of real specimens.

Students are responsible for material in Part 1 of these notes. Parts 2 and 3 are not required reading, but should be helpful in learning, reinforcing and integrating the material.

Acknowledgements: This work is based partly on material from previous gross pathology laboratory instructors, and we wish to acknowledge the contributions of these individuals: Drs. Brian Wilcock, Andrew Brooks, John S. Lumsden and Geoff Wood.

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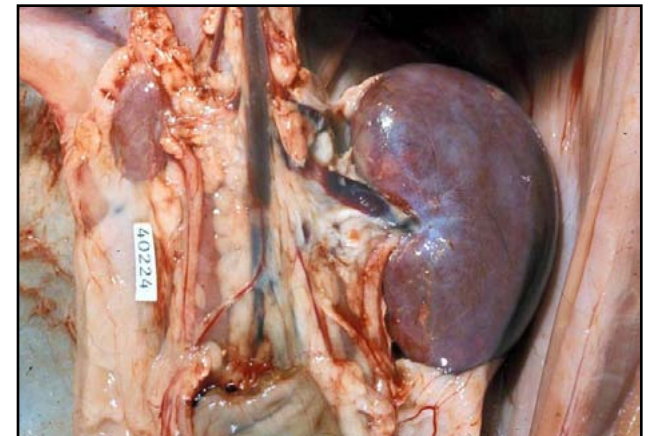
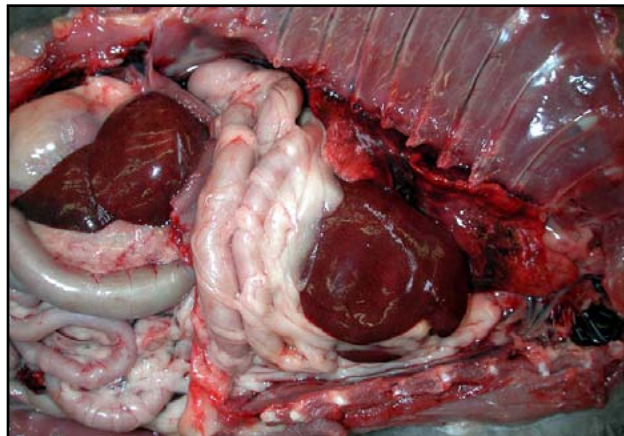
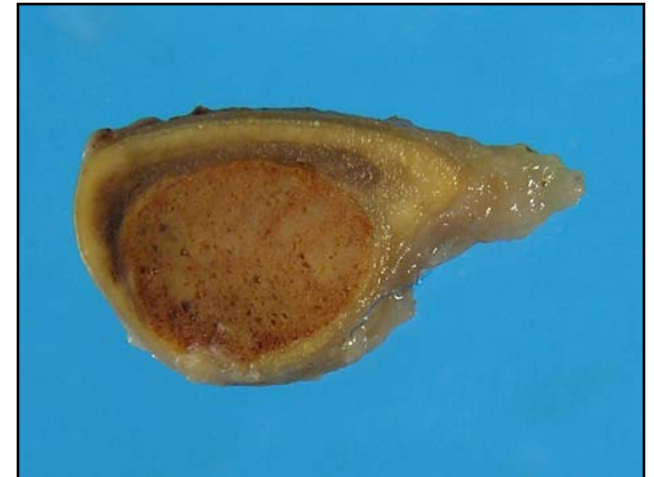
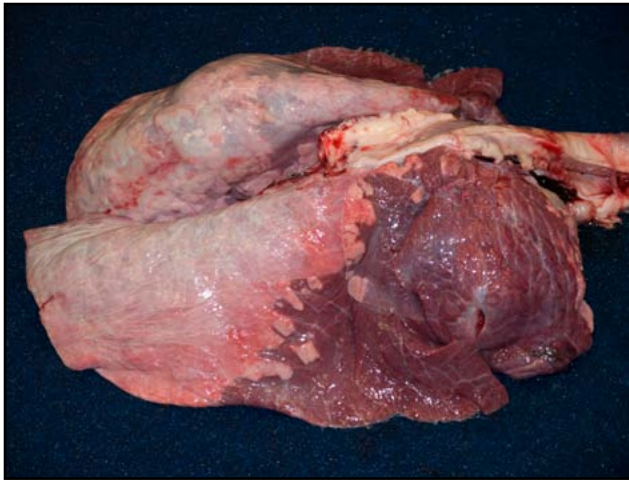
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Part I. Introduction to Pathologic Description and Interpretation



The discipline of pathology is the study of the changes occurring in tissues during disease, including both the structural or morphologic changes (lesions) and molecular abnormalities. Pathology also addresses the causes and mechanisms by which these abnormalities develop, and their functional consequences. Veterinarians need to understand the tissue changes that occur in disease for at least two reasons: on a conceptual level to understand how a cause of disease results in clinical signs, and on a practical level by observing and interpreting lesions to determine the cause of a disease and to explain clinical observations.

Experienced veterinarians often glance at a lesion and immediately recognize the likely cause and expected clinical signs. However, at an earlier stage of learning—or for cases with atypical or unusual lesions—it is useful to develop these skills in sequence. It should be obvious that these methods are not unique to pathology, since exactly the same investigative approach is used in other medical disciplines. The general approach to evaluating tissue specimens is similar to other aspects of clinical investigation, and is summarized as follows.

Investigating gross lesions

1. Observe the tissues
2. Identify the abnormalities
3. Describe the abnormalities:
Objective, factual, concise, organized
4. Make an interpretive summary:
Likely pathologic process, morphologic diagnosis
5. Full case interpretation:
Etiology, pathogenesis, clinical correlates

1. Observe the specimen

Observation is at the heart of pathology, as it is for many other disciplines, observation is a skill that can be developed with practice and aided by knowledge.

2. Identify abnormalities

A lesion is any observable change in a tissue, if it is considered to be abnormal. The ability to detect and describe lesions is a critical first step in investigating how the lesion happened, its functional significance,

and its cause. Determining that a lesion even exists is not always easy, and requires that we are able to appreciate that the tissue in question differs from normal. It is a mistake to rush to the description; instead, take time to observe the tissue and consider how it differs from normal.

There are two pitfalls in this regard. Firstly, a knowledge of anatomy is required; we must be able to identify not only the tissue we are looking at, but also the orientation within the animal (which is the cranial aspect of the tissue?), as well

**Consider three questions
when identifying abnormalities in tissues:**

- 1. Is it a normal anatomic structure?**
- 2. Is it a post mortem artefact?**
- 3. Is it different from the normal tissue?**

as anatomic structures within the tissue such as cortex, medulla, blood vessels, etc. Secondly, we must be able to recognize artefacts that commonly develop after death, so we are not distracted by them and thereby overlook the important lesions.

Postmortem changes develop after the death of the animal, and other changes may occur at the time of death. It is important that you are familiar with these changes, and able to differentiate them from significant antemortem tissue lesions. The most pervasive of these changes is autolysis, or “self-digestion” of tissues. Autolysis develops at different rates in different tissues, being slow in muscle, and

rapid in intestine. These changes occur particularly quickly in warm weather, and in animals such as sheep and swine where the insulating effect of fleece or fat prevents dissipation of body heat after death. Autolyzed tissues have diffuse or localized areas of softening and pallor, which can be distinguished from antemortem necrosis by the lack of grossly or histologically visible zone of reaction or inflammation in surrounding tissues. Rigor mortis is the post-mortem contraction of striated, smooth and cardiac muscle. Rigor results in stiffening of the carcass during the 1-6 hours after death, and then dissipates over the following 24-48 hours although there is much variation depending on the

energy reserves of the animal and ambient temperatures. Hypostatic congestion involves the gravitational pooling of blood after death, depending on the position of the body. This often results in reddening of one lung, while the other remains of normal colour. Blood pools in the viscera as blood vessels dilate after death, making the intestines, lungs and sometimes other tissues appear red-purple and congested. This pooling of blood may be segmental in the intestines, while other areas may be blanched due to the pressure of adjacent viscera.

Imbibition of blood or bile pigments results in discoloration of tissues. Hemoglobin imbibition is commonly seen in carcasses subjected to freezing and thawing, and in autolysed fetuses, with breakdown of red blood cells and diffusion of hemoglobin causing red staining of tissues and of fluids in body cavities. The green-black discoloration of bile imbibition is particularly evident in areas of liver and loops of bowel which are in contact with the gall bladder after death. Gas production by saprophytic bacteria can result in the rapid development of emphysema in the liver, and tympany of the gastrointestinal tract. This may even lead to postmortem rupture or displacement of the viscera, and

prolapse of the rectum. Putrefaction can induce green-black discoloration of tissue (pseudomelanosis) due to the breakdown of blood and formation of iron sulfides. Post mortem trauma (abrasions, fractures) can be differentiated from that sustained during life by the absence of hemorrhage or an inflammatory reaction. Similarly, post mortem scavenging lacks evidence of adjacent tissue hemorrhage, and often involves damage to the eyes, rectum and external genitalia.

3. Describe the abnormalities

Description is the basis for scientific communication, by which we are able to share information about lesions or diseases. Description is the basis of the pathology report, which forms the legal record and archive of the findings. Description allows the veterinarian to discuss cases with colleagues, and is an essential element when submitting tissues to diagnostic laboratory. Equally important, the act of description focuses and refines our observational skills, and thus enhances our ability to examine tissues in a detailed manner.

In gross (macroscopic) pathology, lesions are described in terms of their distribution, extent, size, colour, shape, contour,

Descriptions should be well-organized, so that the reader develops a clear mental picture of the lesion without needing to re-read previous sections. The best organizational structure will vary depending on the specimen, but the following is a guideline:

- If multiple types of lesions are present, describe one at a time
- Descriptions generally start with an overview of the location and distribution of the lesion, then move on to the fine details of the lesion's size, shape, colour, texture, etc.
- We usually describe the most important abnormalities first, and describe artefactual changes last, if at all. Arguably, this may introduce subjectivity to an otherwise objective factual description, but such reports are more easily understood by the reader.

texture and strength. It is appropriate to emphasize that description must be objective and factual, not judgemental or interpretive. In short, we are simply acting as recorders of what is present without prematurely deciding on the pathologic process, cause, or significance. There will be many instances where this idea of a completely objective description will be challenged, because it is often difficult or inefficient to be completely objective in describing a lesion. However, at least initially, it is best to describe lesions objectively, because these statements of fact cannot be proven wrong. In theory, an observant lay person should be as effective in writing a

pathologic description as an experienced pathologist. In contrast, your interpretation of what is happening in the tissue is a subjective process that requires insight, knowledge and experience. It is in developing this interpretation that your knowledge of veterinary pathology and of veterinary medicine is put to use. Even if this interpretation is incorrect, your objective description of the facts should be correct.

Descriptions should be concise. The skill of concise description improves with practice, and involves describing the essential features of a lesion without wasting ink on unnecessary words that don't add useful in-

formation or meaning. This skill will serve you well, and allow you to write records, reports, and laboratory submissions quickly but with the necessary details included.

The language we use for descriptions should be considered. In general, pathologic specimens can be adequately described using common English words. However, you will find that pathologic descriptive terms make the task much easier, because a

long series of English words can be captured with a single pathologic term. We don't use these terms to be exclusive or to flaunt our knowledge; instead, the language of pathology helps us describe lesions concisely and accurately. Nonetheless, it is worth recognizing that most pathologic terms summarize your interpretation of the lesions and should therefore not be included in the description (eg. hemorrhagic, congested, atelectatic, atrophied), whereas relatively few pathologic terms are purely descriptive and objective (eg. ecchymotic, petechial, indurated, friable, inspissated, sessile, papillary).



For these laboratories, descriptions should be narrative (sentences) rather than point form, using present tense, and avoiding acronyms or short forms. Point form and acronyms are effective for making personal notes, but their meaning is usually less apparent to others than to ourselves. Bulleted points in sentence form are acceptable. Do not describe the normal features or normal appearance of the tissue unless there is a specific reason to do so.

A description should define and clearly communicate the significant morphologic abnormalities in a tissue. This verbal or written “picture” should be clear enough to allow the audience to visualize the lesion, and formulate a morphological diagnosis without having actually seen the specimen.

Descriptions should generally include elements of location, distribution, size, extent, shape, contour, colour, texture and strength, but not all of these features are relevant for every lesion.

Location & distribution

Size & extent

Shape & contour

Colour

Texture & strength

The kidney contains a single localized lesion that extends from mid-medulla to the capsular surface, is 1 x 2 cm diameter, affects 5% of the renal tissue, and is triangular, well-demarcated, flat, white-tan with a red margin, and is softer than but retains the strength of the adjacent tissue.

Close your eyes.

Can you picture the lesion, from the description?

i. Location

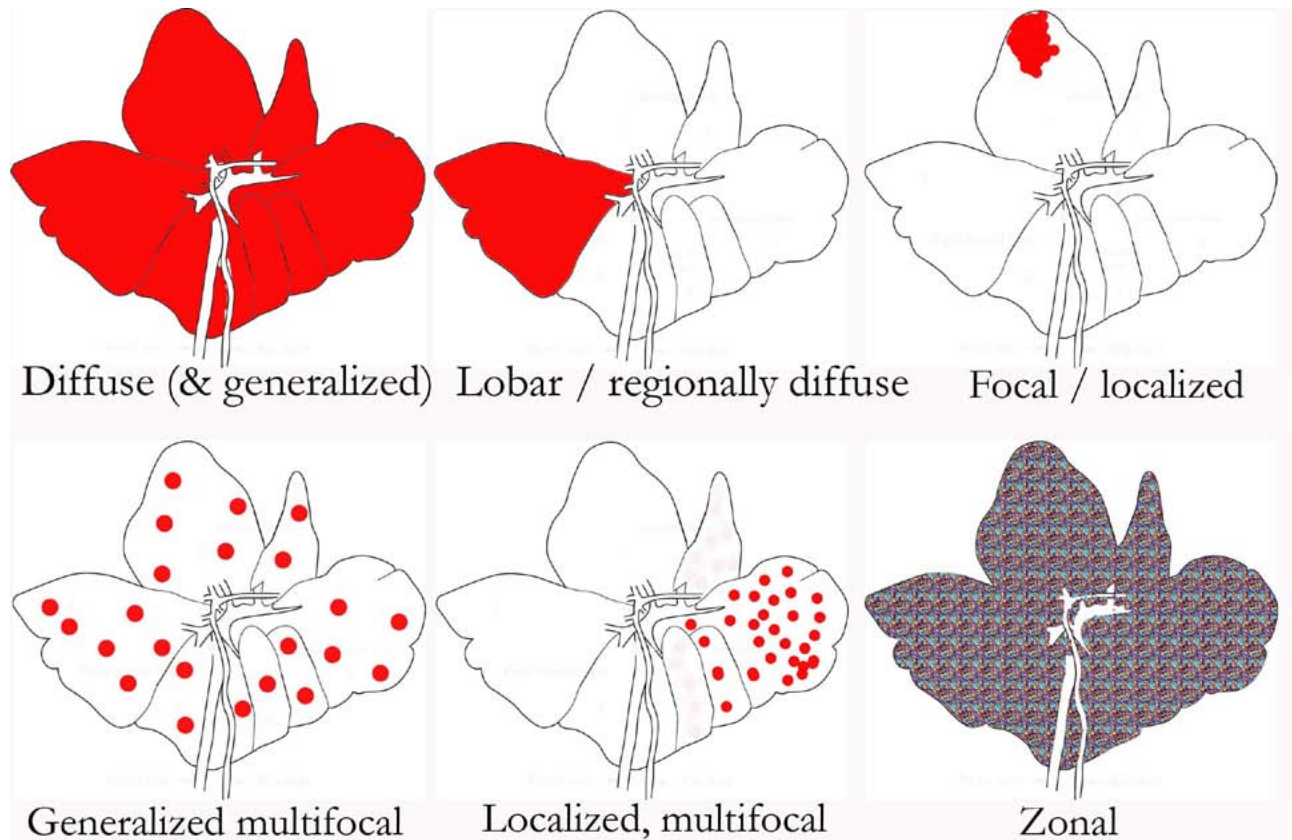
State the name of the affected

organ or tissue. Wherever possible, precisely specify the location of the lesion; for example, in the renal cortex, pleura, white matter of the parietal lobe of the cerebrum, periphery of the right middle lung lobe, or septal cusp of the left atrio-ventricular valve. Describe the lesion's proximity to anatomic structures, such as the bronchi, or a tendency to track along mesenteric blood vessels, or that part of the liver adjacent to the stomach. Lesions in the digestive tract may have a mucosal or serosal location, or are transmural if they involve all layers. Any abnormalities of position should be noted.

Descriptors of location include cranial, rostral and caudal; ventral and dorsal; left and right; lateral and medial; axial and abaxial; central and peripheral; and superficial and deep. Although you can use plain English words for your descriptions, medical terms allow us to describe lesions more concisely and more precisely.

ii. Distribution

The distribution of the lesion is often the single most useful observation, for it gives valuable insight into the nature of the injurious event. Terms to describe the distribution of lesions are common English words,



As seen from a distance, lesions may be localized to one lobe of the liver, or generalized throughout all lobes.

In terms of the “close-up” distribution of lesions within the affected part of the tissues, the lesion is said to be:

- diffuse if the entire liver or all of the affected lobe is red and hard
- focal or multifocal if the lesions comprise discrete spots or nodules
- zonal if the affected areas follow a specific anatomical structure within the tissue (eg. lesions affecting the peri-acinar zone of liver).

but some carry some precise meaning that may not be generally appreciated.

Wherever possible, the anatomic basis for a lesion's location and distribution should be described, as this is sure to be a clue to pathogenesis. In paired or symmetrical organs (kidney, brain, etc.), lesions may be unilateral or bilateral, and symmetrical or asymmetrical. In lung and liver, a lobar/lobular distribution affects some lobes/lobules entirely but spares others. A segmental lesion in a tubular organ (eg., intestine) suggests a localized insult or a vascular basis for the disease. Radially arranged lesions in the renal cortex suggest they are based on the arcuate blood supply or the agent has arrived via the collecting ducts. Other terms to describe distribution of lesions include coalescing, patchy, mottled and dappled.

iii. Size & extent

The size of lesions, unlike colour or distribution, does not usually give much insight into the cause, but is critical for determining the impact of the lesion on overall tissue function. Size is best described as a metric measurement. Avoid the common tendency to describe lesions in terms of relationship to other familiar objects, be-

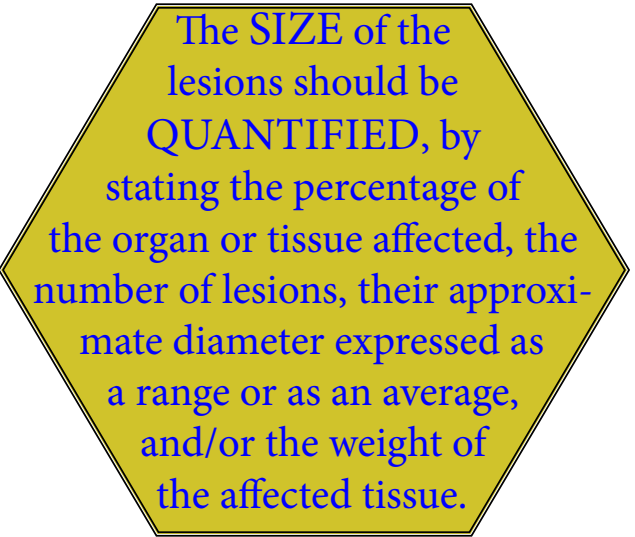
cause these objects are not of uniform size ("the size of a grape") and your audience may not be familiar with them ("sliothar-sized"). When lesions range in size, describe the minimum and maximum sizes; for example, "1-3 cm diameter nodules". Uniformity in size can be mentioned.

Describe the number of lesions present, but recognize that the exact number usually carries little value. "About 30" or "innumerable" is generally sufficient.

The extent of a lesion can be expressed as the percent of the organ (or part of the organ) that is affected. Describing the extent of a lesion may predict its functional significance. One can provide a magnificent description of a lesion, yet fail to mention that it involves only one percent of the organ and is thus unlikely to have any functional significance.

iv. Shape, demarcation & contour

The shape of lesions is usually described in simple geometric terms: spherical, rectangular, diamond-shaped, triangular, ovoid, elliptical, etc. Shape may give a clue as to the pathogenesis; for example, triangular or other sharply angular lesions are often infarcts, or have a basis in the lobular



anatomy of the organ.

Focal or multifocal lesions are described as well-demarcated or poorly demarcated. Well-demarcated lesions might represent a different tissue or a different lobule within a tissue, a distinct structure such as a parasitic cyst, or a tumour that is expansile but non-infiltrative.

Surface contour can be particularly useful in thinking about underlying processes. In general, raised areas imply that something has been added to the tissue, such as fluid (edema or blood), cells (inflammation, hyperplasia, neoplasia) or gas (due to bacterial growth in tissues, or release from the damaged lung). Depression of the surface below that of the adjacent tissue is usually

the result of fibrosis, or loss of tissue such as from atrophy or necrosis. Other lesions of necrosis have a flat surface contour, which may represent no change in tissue volume, or simply a change that cannot be detected grossly. Other words to describe the surface contour include bulging, nodular (a raised mass), exophytic (abruptly protruding), sessile (attached to the organ by a broad base), pedunculated (attached by a narrow stalk), papillated (shaggy), villous (finger-like), rugose, corrugated, fissured, serrated, soft, pitted, cavitated, or sunken.

v. Colour

The colour of a lesion is relatively easy to describe; red, white, yellow, black and tan are the most commonly encountered colours in both healthy and diseased tissue. The use of the suffix “-ish” should be avoided: if a lesion is not quite white, saying “white-ish” does not give us any guidance as to the direction in which this lesion deviates from the ideal of white. Saying yellow-white, for example, would better communicate the actual colour. More refined indicators of colour are encouraged: amber, bronze, ochre, olive, coffee, and ecru are more precise than “tan”, although it should be recognized that these names may be interpreted differently by others,

and a visit to any paint store will prove that this approach can be overdone.

The perception of colour is also influenced by how closely one examines the change. A liver that looks orange on casual inspection might, in a manner analogous to colours on a television screen, actually be identified as having alternating zones of red and tan if one were to inspect it more closely. Noting that liver has alternating tan and red zonal colour change is sure to be more informative, when attempting to deduce a cause, than the more casual observation of an orange liver. Since colour change often is not uniform, some useful adjectives include words like variegated, mottled, marbled, and coalescing.

Colour potentially carries information about the nature of the pathologic process. In general, red is the colour imparted to tissue by increased amounts of hemoglobin such as in areas of hemorrhage (focal), congestion or hyperemia (diffuse). White can indicate the presence of inflammation, necrosis, fibrosis, or mineral deposits. Yellow may also indicate inflammation (pus), bilirubin (icterus), lipid (fatty liver), or fibrin (on the surface of an organ). Green colouration may be seen with bile pigment, eosinophilic inflammation, necrosis

(gangrene), some algal or fungal infections, hemosiderin (hemoglobin breakdown products), or putrefaction. Black colouration may indicate the presence of melanin, blood (hypoxic blood or hemoglobin breakdown pigment), necrosis, trematode pigment, exogenous carbon or hydrogen sulfide (from the gastrointestinal tract).

vi. Texture & strength

Descriptors of consistency include those that estimate the firmness of a tissue, and those that estimate the strength. While there is a tendency to equate firm with strong, and soft with weak, this is clearly not true. Fishing net is soft and pliable, yet is very strong. A soda cracker is firm, yet crumbles easily.

These descriptive words must be used in context of the adjacent normal tissue, since a lesion described as firm when found in a liver would likely be perceived as soft if it were to occur in bone. In general, increasing degrees of tissue hardness can be described as soft, rubbery, firm, and hard; although the meaning of these terms will differ from one person to the next.

Other adjectives describing tissue texture may be useful. Turgid is a type of firmness

that is due to an internal fluid pressure. Abscesses, for example, may feel turgid yet collapse readily upon excision. Gritty texture suggests mineralization. Caseous lesions—resembling cheese—are crumbly and somewhat dry. Elastic material on a surface is usually fibrin. Spongy lesions usually contain multiple pockets filled with fluid or air. The texture of fluids may be described as watery, viscid, mucoid, gelatinous, creamy, clotted, or inspissated.

The strength of tissue must always be judged by the cut surface, as it is obscured by the fibrous capsule. Tissue strength may be described as friable if it is weak and crumbles easily when palpated, or tough if it has greater than normal strength. Loss of strength or friability is one of the most reliable and consistent indicators of necrosis. Toughness of tissue usually results from formation of fibrous scar tissue. A liver with hepatic lipidosis feels quite firm, yet crumbles readily once you apply some digital pressure to the cut surface; it is thus “firm but friable”.

4. Make an Interpretive Summary: Morphologic Diagnosis

Components of the morphologic diagnosis

- Organ or tissue affected
- Severity: mild, moderate, severe
- Time: acute, subacute, chronic
- Distribution: diffuse, multifocal...
- Pathologic process: inflammation, neoplasia, necrosis...
+ a modifier: fibrinous, granulomatous, necrotizing, proliferative

The morphologic diagnosis is both a summary of the lesions and an interpretation of the likely disease processes. It is not an objective statement of fact, and instead summarizes your opinion or guess on the lesion's severity, duration, distribution, pathologic process, and organ affected.

Why do we use morphologic diagnoses? This interpretive summary is the first step in progressing from an immense variety of possible lesions or observations, to a shorter list of likely diagnoses. Morphologic diagnoses are a tool for problem solving, by helping to summarize the findings, and

generate and narrow the list of differential diagnoses. This helps us consider causes of the disease, selection of appropriate laboratory tests, and appropriate therapy.

Morphologic diagnoses include the following components:

- **Severity:** a subjective estimate of the expected significance of the lesion. Mild, moderate, or severe. These terms are subjective, but communicate what you think is the importance of the lesion.
- **Duration:** acute, subacute, and chronic. These are not definite indicators of time, but “acute” usually implies a duration of less than 4–6 days, while chronic lesions have probably been developing for more than two weeks and often also involve regeneration or repair (fibrosis). Peracute lesions develop in minutes or hours, usually without time to develop observable lesions except hemorrhage or edema.
- **Distribution of lesions:** multifocal, diffuse, localized, generalized ...
- **Pathologic process** causing the lesion: inflammation, necrosis, edema, hemorrhage, atrophy, hyperplasia, neoplasia, fibrosis. For inflammatory lesions, indicate the type of exudate where possible: fibrinous, suppurative, granulomatous, etc.
- **Organ or tissue,** or component of the

tissue affected: hepatic, cutaneous, renal glomerular, ...

A few special rules apply:

a) Inflammatory lesions usually include the description of the organ and an indicator of inflammation (“-itis”) in a single word: hepatitis, dermatitis, glomerulonephritis. Pneumonia is inflammation of the lung. Communication is the key: keep it simple if the Latin translation is too complex to permit useful communication—inflammation of the lacrimal gland is more effective than sialodacryoadenitis, if your audience doesn’t know this word!

b) For neoplasms, the morphologic diagnosis is typically only 2-3 words, the organ or tissue involved (as an adjective or prefix), the type of neoplasm (adenoma, carcinoma, sarcoma etc.), sometimes the distribution, and “metastatic” or “invasive” if relevant. Examples include dermal sebaceous adenoma, invasive hepatic carcinoma, multifocal splenic hemangiosarcoma.

c) Sometimes, the severity or duration of the lesion cannot be determined or is irrelevant. In those circumstances, these components may be omitted. Two examples:

Examples of morphologic diagnoses:

- Mild chronic multifocal generalized suppurative pneumonia
- Severe cranioventral fibrinous and suppurative bronchopneumonia
- Acute multifocal hepatic necrosis
- Diffuse adrenocortical atrophy
- Generalized multifocal renal hemorrhages
- Locally extensive soft tissue hemorrhage of the distal right foreleg
- Small intestinal adenocarcinoma
- Osteosarcoma, right proximal femur

- The severity and duration are not given for neoplasms, because even a small tumour could be fatal if it metastasized or effaced a vital structure, and the duration cannot be determined.
- If there is no evidence of fibrosis, it is often impossible to determine if an inflammatory lesion is acute or chronic. In these circumstances, omit the duration. In other cases, including this information is critical to accurately communicate the disease process (eg.

severe acute fibrinous and suppurative bronchopneumonia).

5. Full Case Interpretation: Etiology, Pathogenesis, Clinical Correlates

This step involves integration of the above observations and interpretations with additional case information (signalment, clinical signs, number of animals affected, response to treatment, etc.), using your knowledge gained from this and other courses, your experience, and your insights, in order to infer the likely cause, pathogenesis, functional importance, and clinical sequelae.

It is a mistake to jump directly from observation to etiology. We encourage a step-wise approach to case interpretation, starting with finding the thing that is abnormal, then objectively valuating this lesion, describing it objectively, then thinking about the pathologic process (eg. neoplasia, inflammation, necrosis, fibrosis, mineralization, hemorrhage, ...) and other components of the morphologic diagnosis. Subsequently, you should start to think about other disease processes occurring in the body that may have incited these

changes in the tissues (eg. disseminated intravascular coagulation or vascular damage or thrombocytopenia as underlying disease conditions that may have caused the petechial hemorrhages), and eventually the etiology or name of the disease condition.

It doesn't always work this way: experienced practitioners or pathologists may look at a puppy with enteritis or a cat with an intestinal tumour and immediately say "That looks like parvoviral enteritis" or "That lesion is intestinal lymphoma". They can do this either because they are going through this same step-wise process quickly in their minds, or because they have seen the lesions before and are recognizing the pattern. In the latter case, it would be a mistake to stop at that point. After immediate recognition of a tentative diagnosis, we should always revisit the specimen and go through the process of description, interpretation, then consideration of various diseases or etiologies. When we leap to a diagnosis, we commonly overlook other possibilities and ignore clues that point to a different conclusion. Work through the case in a logical manner, eliminate some diagnoses based on the character and spectrum of the lesions as well as other case information, to reliably arrive at the cor-

rect diagnosis.

Interpretation of the changes can occur on several conceptual levels, as follows. It is important to understand these terms—disease name, etiology, pathologic process, pathogenesis—because they guide our understanding of how disease develops.

On a more practical level, these are always the subject of exam questions, and giving an etiology when asked for a pathologic process is disappointing.

Name of the disease: the name that we commonly call the disease, such as Johne's disease, strangles, or diabetes mellitus.

Etiology: the cause of the disease. The etiology is often an external agent, and exposure of the animal to this agent triggered development of disease. Other etiologies can be intrinsic, such as genetic mutations. Examples of etiologies are *Mycobacterium paratuberculosis*, *Feline herpesvirus-1*, lead poisoning, acetaminophen toxicity, vitamin D deficiency, exposure to heat or cold, trauma, and mutation in the gene *MYBPC3*.

Pathologic process: the change that is occurring within the tissue, that is caused

by the etiology and results in the observed lesion. Think of the pathologic process as the animal's response to the etiology. Examples of pathologic processes are granulomatous inflammation, neoplasia, necrosis, fibrosis, mineralization, hypoplasia, and hemorrhage.

Pathogenesis: The pathogenesis is an explanation of how the disease developed. The pathogenesis begins with the inciting cause or initial incident, and then describes the sequence of events that leads to the observed lesions or clinical signs. For example:

- The pathogenesis of subcutaneous edema in Johne's disease is as follows: *Mycobacterium paratuberculosis* infection in the intestine... infiltration and activation of macrophages... secretion of inflammatory mediators by macrophages... leaky blood vessels in the intestinal mucosal... protein loss into intestine... low blood protein levels... reduced oncotic pressure in blood... peripheral edema. In this example, *Mycobacterium paratuberculosis* is the etiology, Johne's disease is the name of the disease, and granulomatous inflammation is one pathologic process.

- Multifocal pulmonary abscessation in a feedlot steer might have the following

pathogenesis: carbohydrate feeding... rumen bacteria metabolize carbohydrate... rumen acidosis... overgrowth of *Fusobacterium*... inflammation of the rumen... entry of *Fusobacterium* into the portal bloodstream... hepatic abscessation... erosion into vena cava... embolic showering of bacteria/debris into lung. In this example, excessive carbohydrate ingestion is one etiology, “grain overload” is the name of the disease, and inflammation is one pathologic process.

- Dogs with ACTH-secreting pituitary adenomas develop enlarged bronze-coloured livers, with the following pathogenesis: ACTH-secreting pituitary tumour... increased cortisol production by adrenal cortex (hyperadrenocorticism)... cortisol induces glycogen accumulation in hepatocytes... liver enlargement and bronze colour. In this example, the etiology of the pituitary tumour is not known, hyperadrenocorticism or Addison’s disease is the name of the disease, and glycogen accumulation is one pathologic process.

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3. Describe the abnormalities:
Objective, factual, concise, organized
4. Make an interpretive summary:
Likely pathologic process, morphologic diagnosis
5. Full case interpretation:
Etiology, pathogenesis, clinical correlates

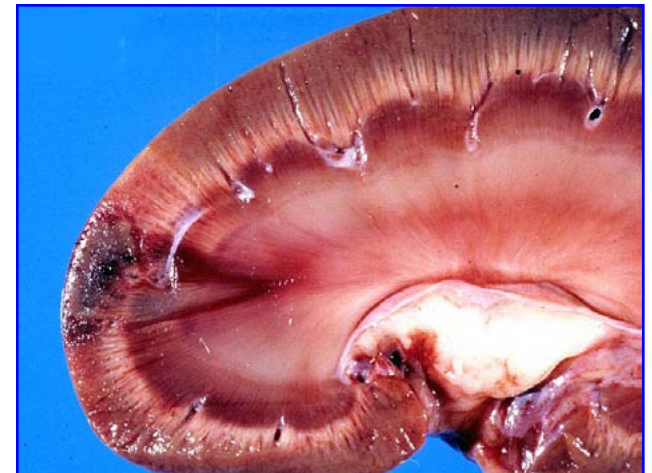
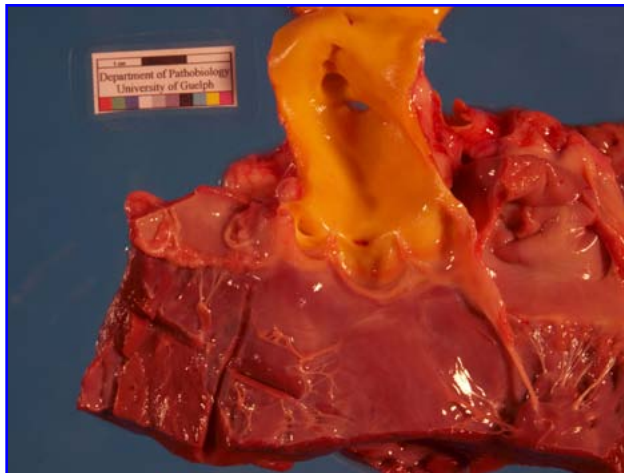
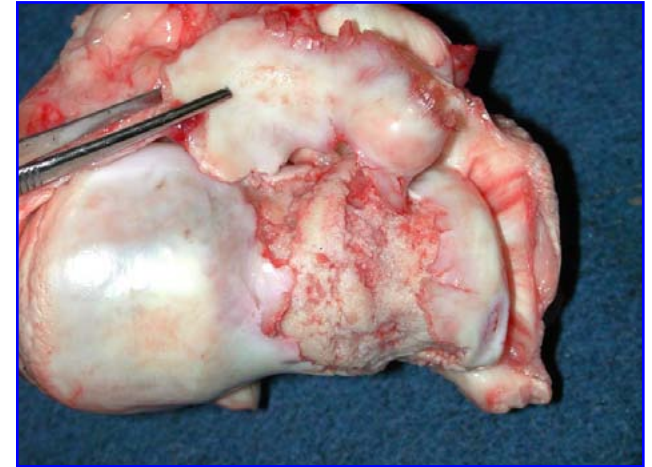
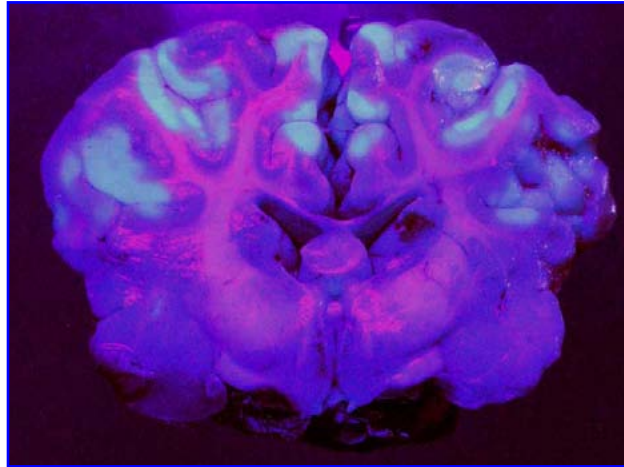
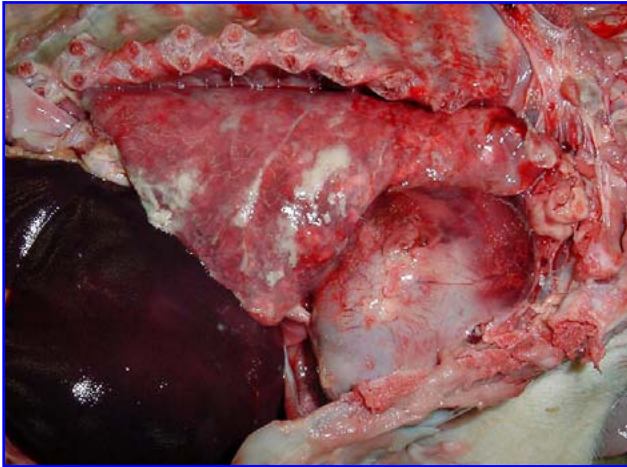
Descriptive terms

1. Location & distribution
2. Size & extent
3. Shape, demarcation & contour
4. Colour
5. Texture & strength

*Can you picture the lesion,
from its description?*

Part II. Recognition of artefacts

Part III. Use and Interpretation of Descriptive Terms

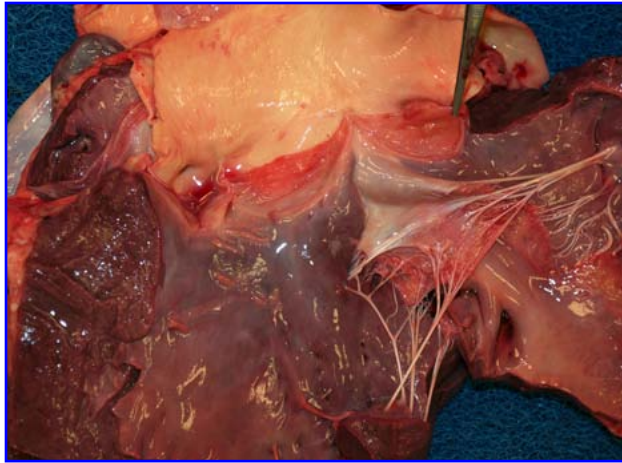


Location of lesions

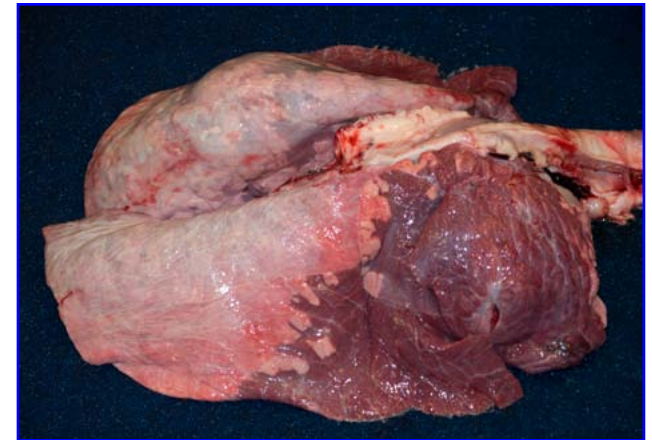
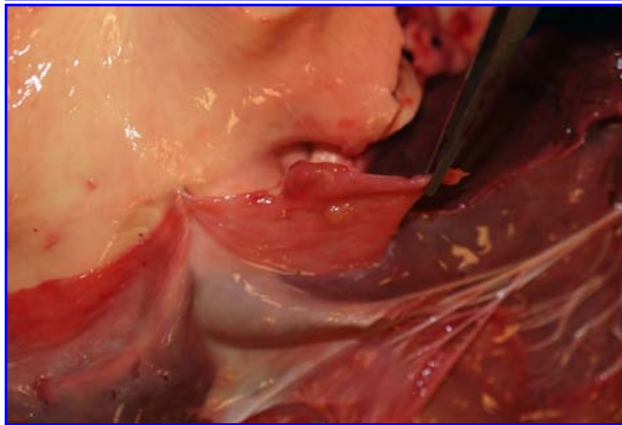
- State the organ or tissue affected, in every description.
- Be as precise as possible in describing the location of lesions, as this provides important clues to the cause, pathogenesis, and significance of lesions

Below: Horse, esophagus. The thickening of the esophagus is due to expansion of the muscular layer. Esophageal muscular hypertrophy is of unknown cause and usually of no significance, but it is important to distinguish this from mucosal lesions.

Bottom: Cow, small intestine. Intestinal mucosal thickening, typical of Johne's disease (paratuberculosis).



Horse, heart. Aortic valve dysplasia. The important feature of the nodular lesions is that they affect the valve, and specifying which valve is affected is key to understanding the clinical manifestations of this lesion.



Location is key to interpreting lung lesions.

Above: Dairy calf. The cranioventral distribution indicates bronchopneumonia, and implies that the cause is a bacterial pathogen entering via the airways.

Below: Puppy with parvoviral enteritis and secondary sepsis. The location of the dark red-black areas at the periphery of the lung is key to identifying them as infarcts, which have developed secondary to sepsis.

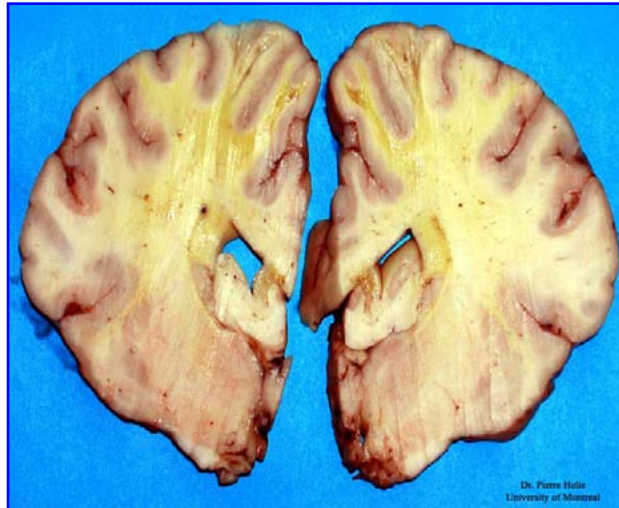
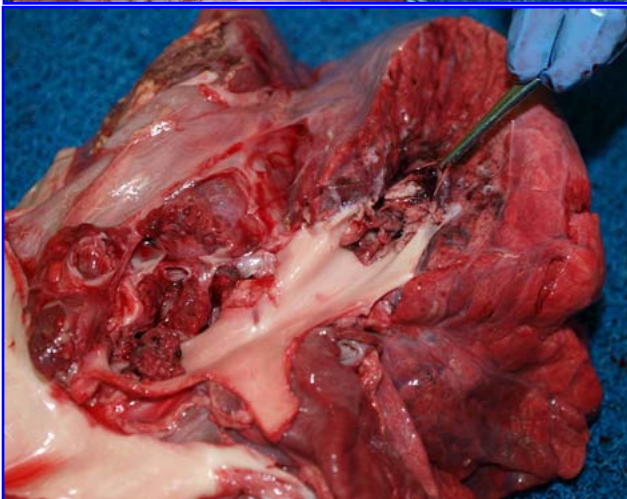
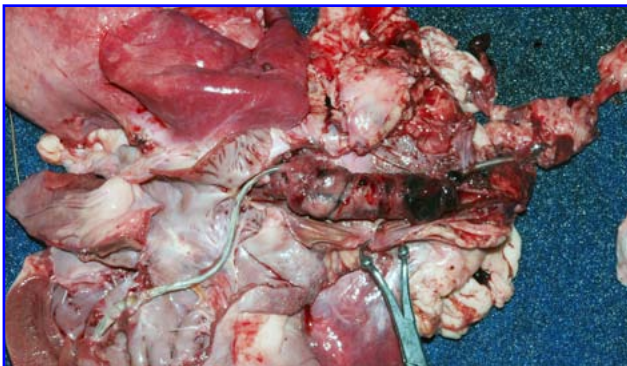


Location of lesions

Recognizing that these cylindrical structures fill the lumen of blood vessels is critical to identifying them as thrombi.

Below: Dog, cranial vena caval thrombosis around a pacemaker lead.

Bottom: Calf, pulmonary embolism from endocarditis of the right atrioventricular valve.



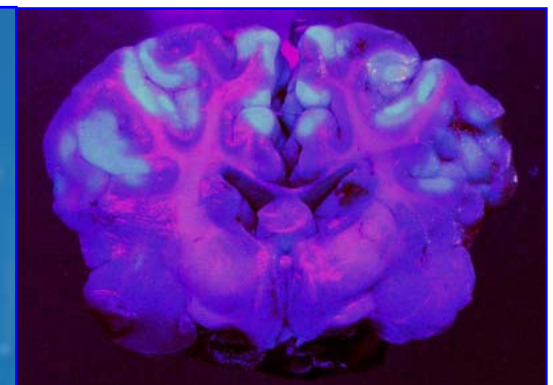
Location begets the diagnosis of two cerebral lesions.

Left: Horse. Location of the yellow lesions in white matter identifies the lesion as leukoencephalomalacia, caused by ingestion of moldy corn.

Below: Calf. Location of the yellow (and autofluorescent, at bottom right) lesions in the cerebral cortical grey matter identifies them as polioencephalomalacia, in this case due to sulfur toxicity.



Above. Dog, sectioned limbs. The location of the dark discoloration specifically involves the distal extremities of all four limbs. This consistent location suggests ischemia as the mechanism of tissue damage, and was probably the result of sepsis in this case.

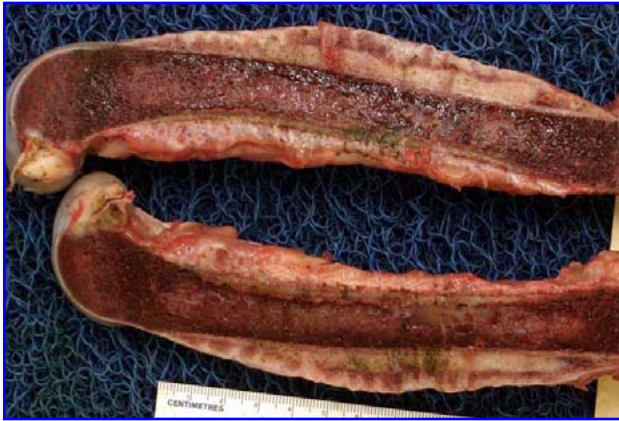
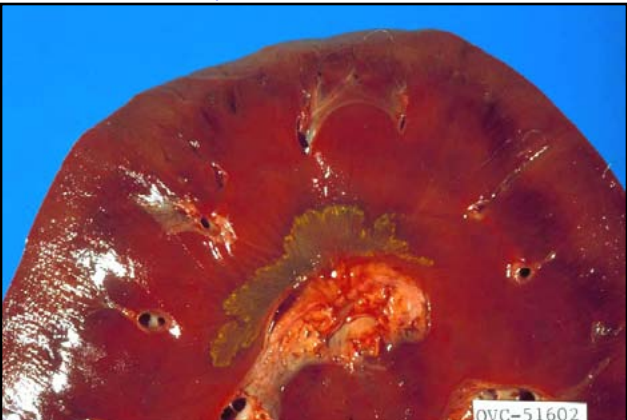


Location of lesions



Above: Dog. The cortical location of the triangular lesion identifies it as a renal infarct, due to obstruction of the arcuate artery by a thrombus.

Below: Horse. The location of the olive green lesion in the deep medulla is characteristic of ischemic damage due to NSAID toxicity.



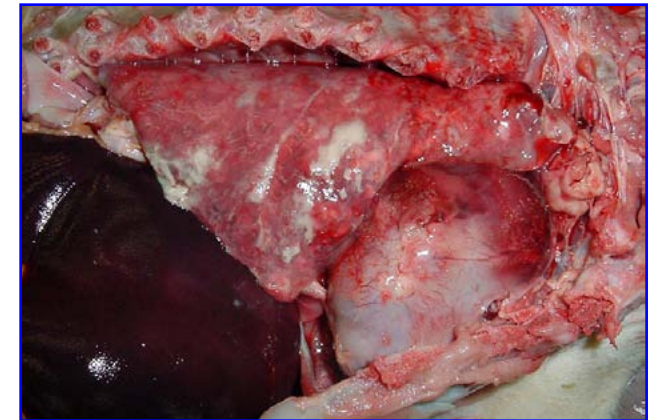
Two lesions of periosteal new bone formation.

Above: Dog. The lesions affect the entire diaphysis but spare the epiphyses, typical of hypertrophic osteopathy secondary to a lung mass.

Below: Cow, affecting the epiphysis adjacent to the joint, due to chronic arthritis.



Below: Pig, thoracic cavity. The lesion does not affect the lung tissue *per se*, but only the pleura. Fibrinous pleuritis has different implications than fibrinous pneumonia, and specific location is the key distinguishing feature.



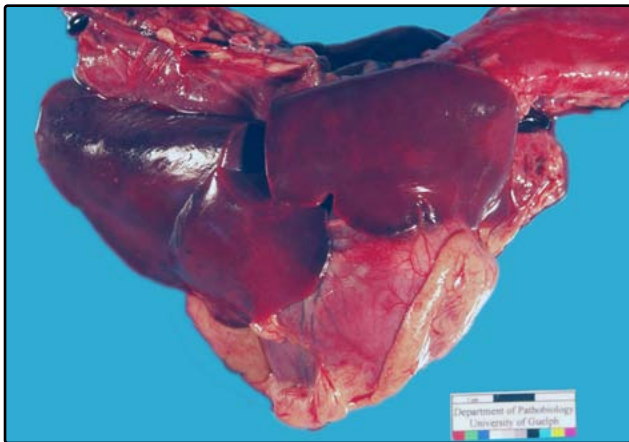
Below: Dog. Clue #1: the dilated spaces represent renal pelvis. Clue #2: the lesion affects only one of the kidneys. These features can only be explained by unilateral ureteral obstruction, in this case due to transitional cell carcinoma.



Distribution: diffuse lesions

- **Diffuse:** the affected tissue is affected uniformly, like a gas diffusing through the air. The colour on a solidly painted wall has a diffuse distribution. Multifocal or zonal are opposites of diffuse.
- Unless otherwise specified, diffuse lesions are assumed to be generalized (present in all areas of the organ or tissue). “Localized diffuse” lesions are simply termed focal or localized.
- Extensive patchy lesions may blur the distinction between diffuse and multifocal: diffuse lesions may differ in intensity across a tissue.
- Diffuse lesions imply a uniform exposure and response of the tissue to the disease-causing stimulus.

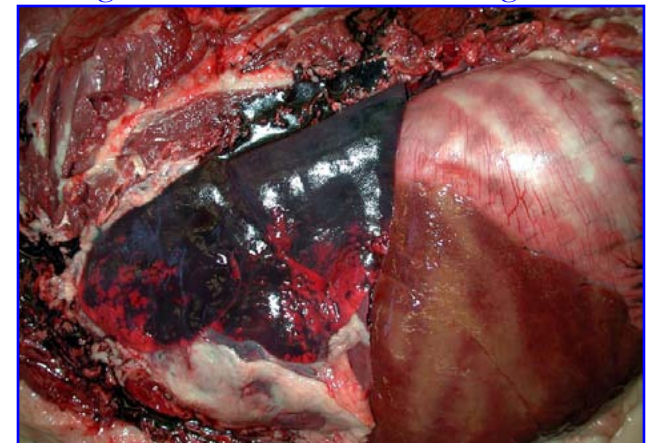
Dog. The lung is diffusely red, and firmer than normal with a liver-like texture. This distribution typifies interstitial lung injury.



Dog. The spleen is uniformly enlarged. Diffuse splenomegaly due to infiltration of leukemic cells.

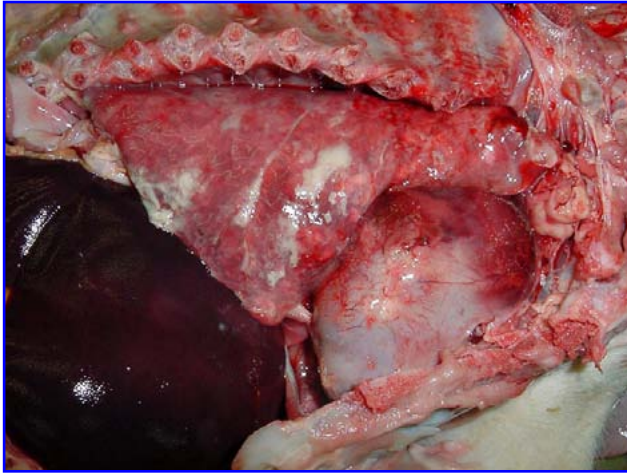


Dog. The lung is diffusely purple, except for a few remaining red areas along the ventral aspect. This represents congestion, and the distribution reflects death occurring in dorsal recumbency during anesthesia. Although the entire lung is not affected, we interpret that the disease process (pulmonary congestion) is occurring throughout most or all of the lung.

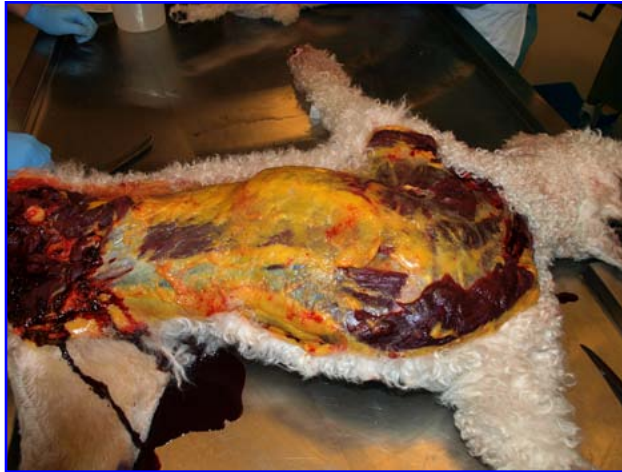
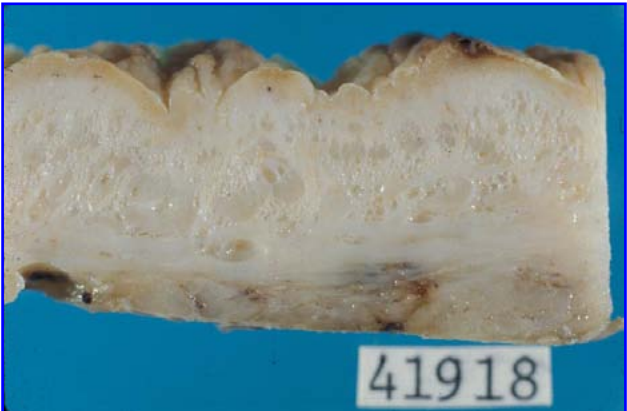


Distribution: diffuse lesions

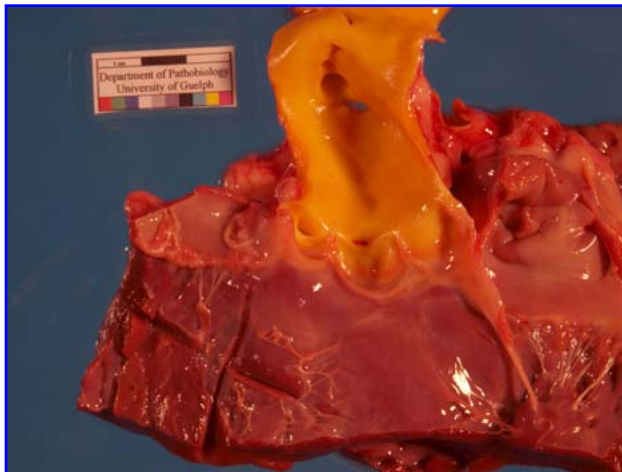
Below: Pig lung. Diffuse fibrinous pleuritis. Fibrin does not cover the entire pleural surface, but we interpret that the disease process (inflammation) affects the entire pleura.



Below: Dog, emphysematous cystitis (urinary bladder). Diffuse thickening and emphysema.



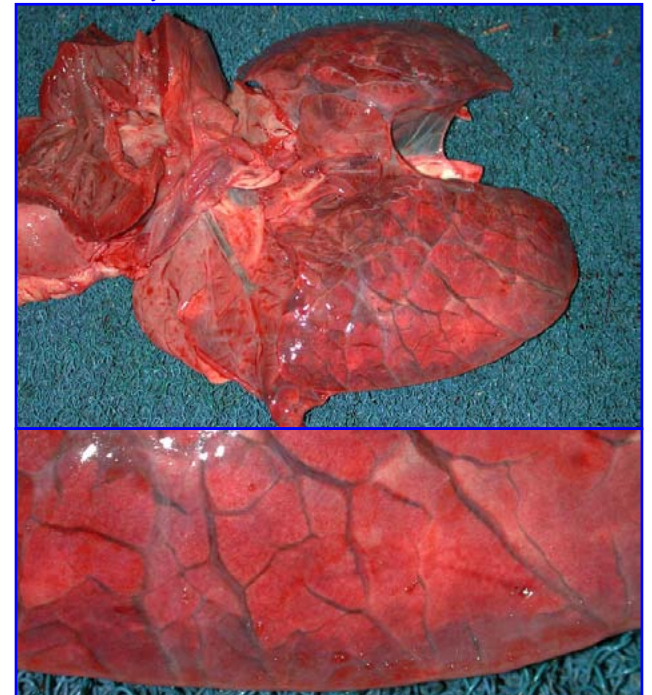
Above and below: Dog. The body is diffusely icteric. This does not mean that all parts of the body are yellow, as the hair remains white, and the muscles and heart are still red. But all parts that are capable of yellow discoloration by bile pigments have turned yellow.



Cow. Diffuse thickening of small intestinal mucosa. Johne's disease (paratuberculosis).



Below: Pig lung. Interlobular septa are diffusely expanded by clear fluid (edema). The lesions affects the interlobular septa uniformly, although the lung tissue itself is not visibly affected.



Distribution: generalized random multifocal

- Generalized: lesions are present in all parts of the organ or tissue. Generalized is the opposite of localized.
- Multifocal lesions are usually randomly distributed: lesions have a haphazard distribution, not based on specific anatomic structures. In contrast, zonal lesions may appear multifocal but with a regular distribution.
- Multifocal lesions vary in shape and surface contour. Many pathologic processes cause generalized multifocal lesions: inflammation, necrosis, neoplasia, hemorrhage, and mineralization.
- This pattern generally implies that the stimulus arrived in the tissue by a blood-borne route.



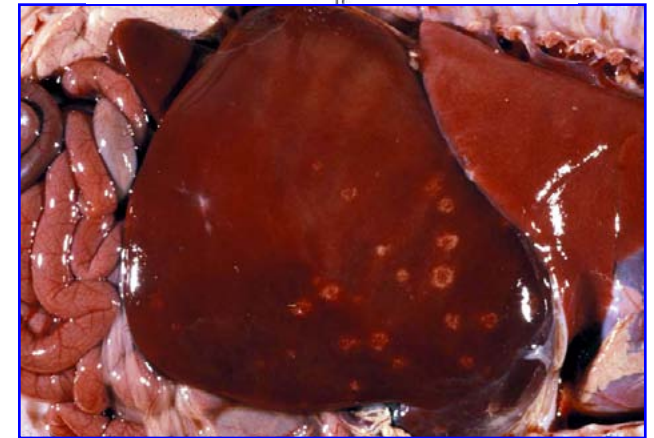
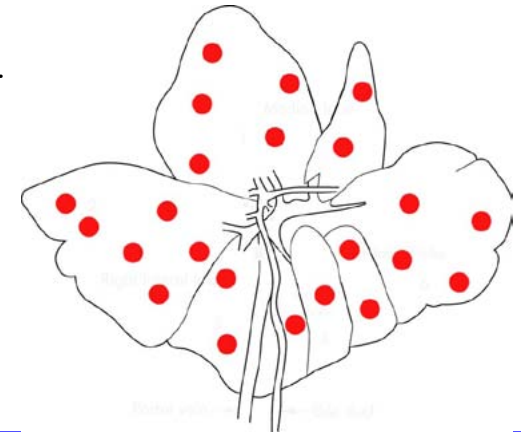
Aborted piglet. The multifocal lesions have a generalized distribution over the entire body surface, and are caused by hematogenous spread of swine pox virus. The white rims of the lesion represent epidermal hyperplasia induced by the virus, while the red depressed centres are erosions that develop after the infected epithelial cells undergo necrosis.



Feedlot steer, esophagus. Tiny multifocal erosions on the mucosal surface represent necrosis caused by hematogenous spread of bovine viral diarrhea virus.



Dog. The umbilicated nodules are carcinoma that metastasized to the liver through the blood.



Aborted lamb. Multifocal raised "target-shaped" lesions in the liver, representing inflammation and necrosis caused by hematogenous spread of *Campylobacter fetus* subspecies fetus.

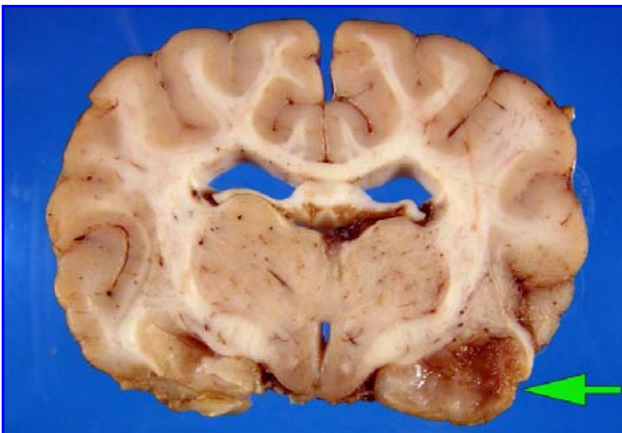
Distribution: focal

Focal: a single lesion, that may be well- or poorly-demarcated. This pattern implies a single point exposure to a damaging stimulus (eg. focal trauma), a rare event occurring once (eg. development of a benign neoplasm), or a lesion filling a single anatomic structure.

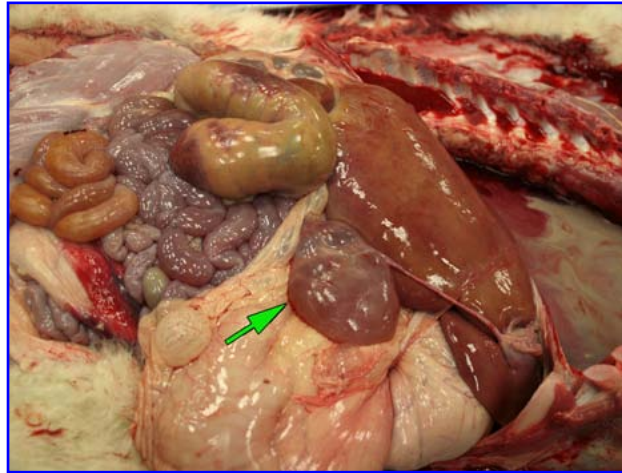
Dog, adrenal gland. A focal adrenocortical adenoma expands the cortex. Neoplastic transformation is a rare event, and the single mass arises from this clone of transformed cells.



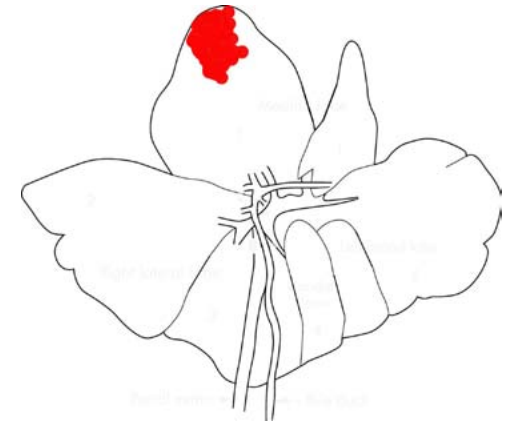
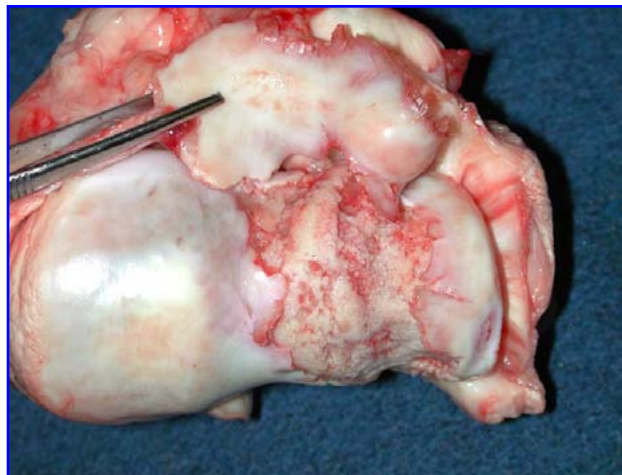
Dog, brain. A focal neoplasm (malignant astrocytoma) in the pyriform lobe (lower right).



Dairy calf. A focal cystic lesion arises from the liver.



Horse. Osteochondritis dissecans, causing focal detachment of the cartilage.



Sheep. Focal osteomyelitis of maxilla, due to bacterial infection penetrating from the oral cavity. The chronic inflammation incites new bone formation in the area of the infection.

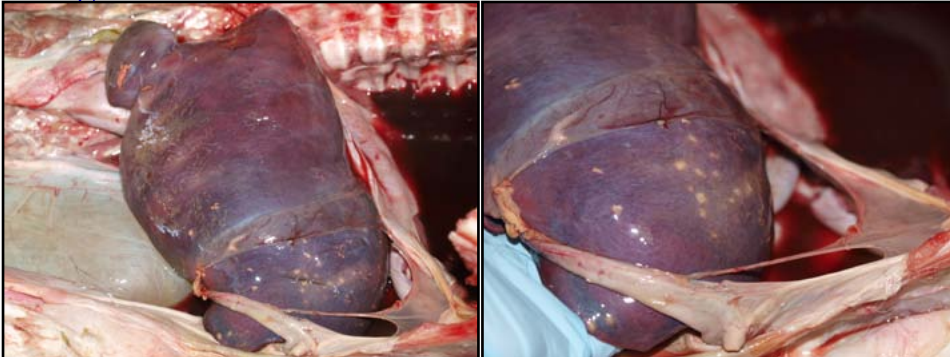


Distribution: localized

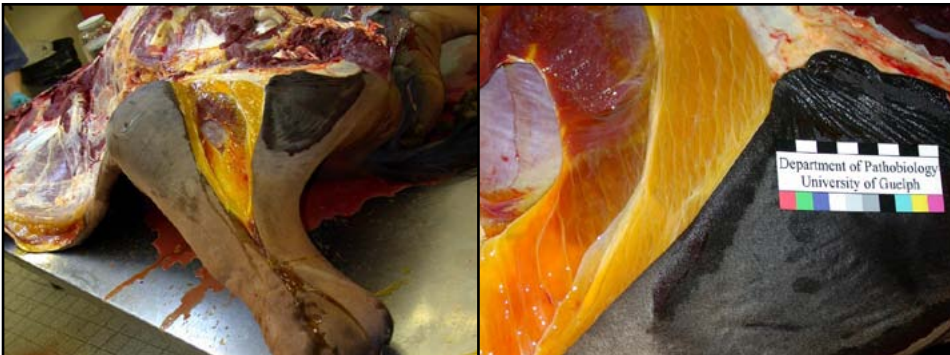
Localized: involving one part or area of a tissue: the opposite of generalized.

Single lesions are usually termed focal. In contrast, the term localized is used to describe larger lesions that don't affect the entire tissue, and have no recognizable anatomic basis. Wine spilled on a carpet makes a focal lesion; a cracked water pipe floods a localized area of the basement.

Neonatal calf, liver. Showering of bacteria from the umbilicus caused a **localized** area of multifocal suppurative inflammation. The lesions are **localized** rather than **generalized**, because the blood in portal veins draining the umbilicus selectively perfuses the right side of the liver.



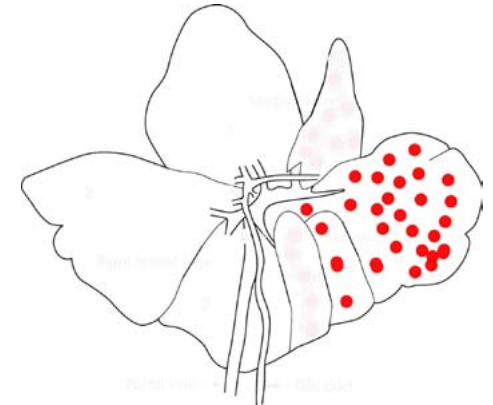
Horse. Bacterial infection of the right hind limb has induced inflammation, resulting in edema of the affected area. The edema is not generalized throughout the body, but instead is localized to the area of the infection.



Right. Horse, thorax. Overlying the ribs near the vertebrae, the parietal pleura contains a localized area of petechial hemorrhage. The horse had endotoxemia due to bacterial colitis, and also had localized areas of petechiation in other tissues.



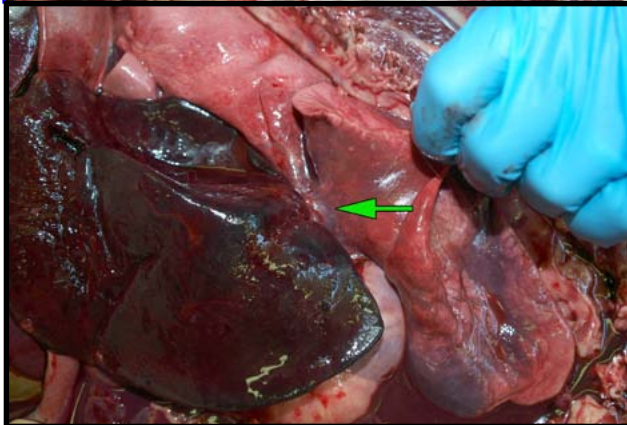
Cow. Hemorrhage in the soft tissues, due to luxation of the hip joint. Either **focal** or **localized** is appropriate to describe the distribution of this lesion.



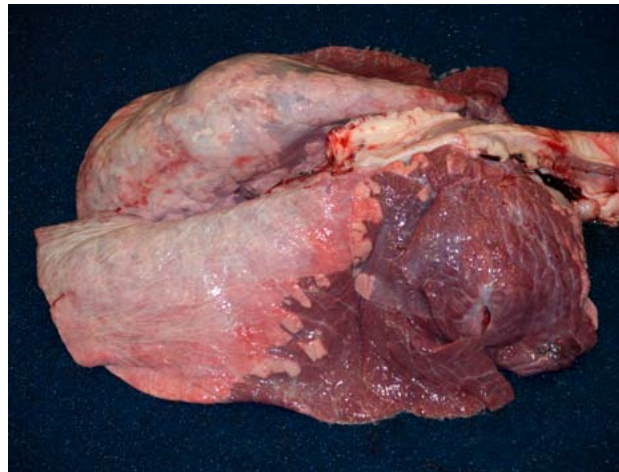
Distributions corresponding to specific anatomic structures

Identifying the anatomic basis for the location or distribution of a lesion often provides an important clue to understanding how a lesion developed.

Dog, thoracic cavity. The right middle lung lobe has undergone venous infarction as a result of torsion. The site of torsion is visible in the lower photo. This is an example of a **lobar** pattern of lesions, in which an entire **lobe** is purple and swollen.



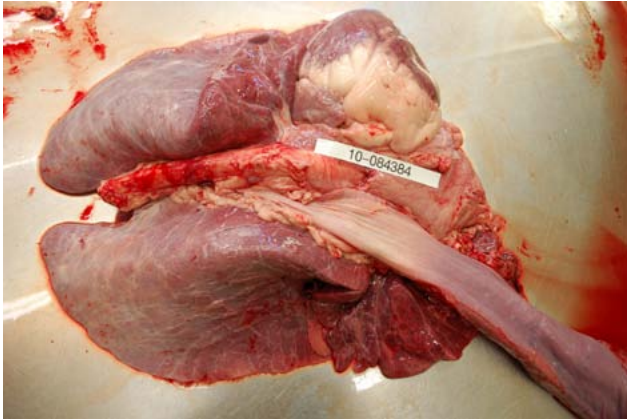
Dairy calf, lung. These cranioventral lesions of bronchopneumonia have a **lobular distribution**. At the junction of normal and abnormal, it is apparent that some **lobules** are entirely affected (purple-tan) while others are normal (salmon pink). This reflects slow spread of infection via the airways, with enough time to involve some lobules entirely but spare others.



Feedlot calf. This lung has a cranioventral distribution of lesions, which contain the focal cavities shown. The nature of these cavities was obscure, until further dissection revealed they form tracts leading to large bronchi. It thus became obvious that the cavities represent bronchiectasis: permanent dilation of bronchi as a result of chronic inflammation.

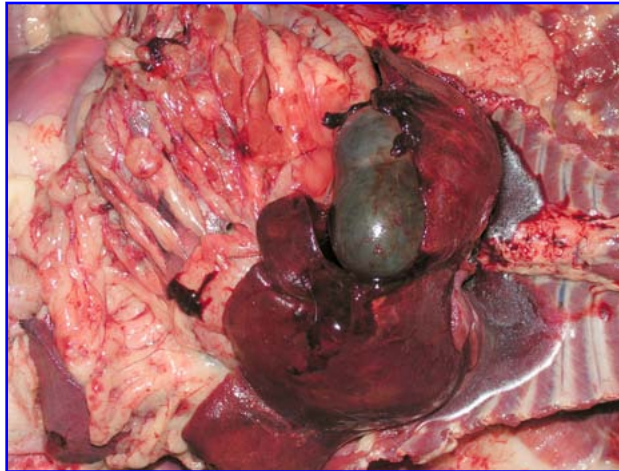


Distributions corresponding to specific anatomic structures

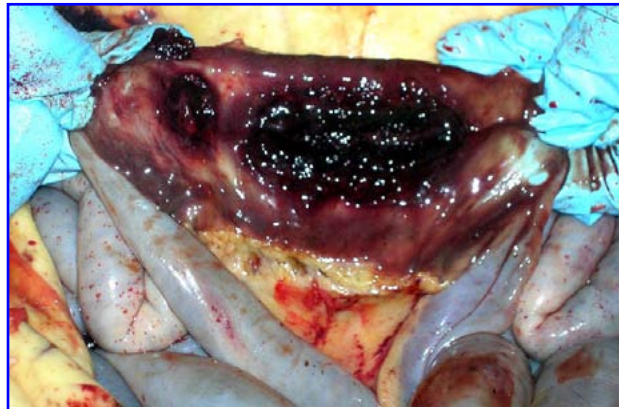


Calf, pluck. The cranial part of the esophagus is red due to congestion, while the caudal part is blanched due to exclusion of the blood. This is a “bloat line”. In bloated calves, distention of the rumen forces blood from the abdominal and thoracic cavities and obstructs venous drainage from the head and neck, leading to the changes observed.

The key observation is that the line of demarcation (the “bloat line”) corresponds to a specific anatomic structure, the thoracic inlet.

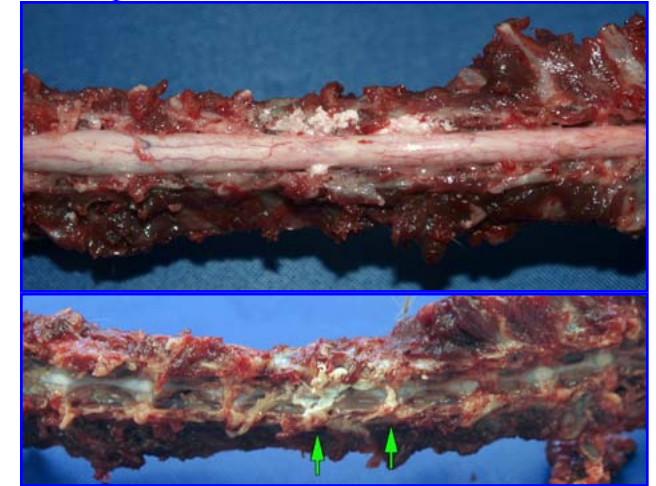


The green mass is focal and well demarcated because it is a single anatomic structure, the gall bladder. Dog, gall bladder mucocoele.



Feedlot calf, small intestine. The cause of these well-demarcated, depressed, hemorrhagic lesions was unknown, until it was recognized that the location and shape correspond to Peyer's patches. The lesion is characteristic of bovine viral diarrhea virus infection, which causes necrosis of the lymphoid tissue forming the Peyer's patches.

Dog, spinal column with dorsal laminectomy. Recognizing the white friable material as extruded disk material is easy, once it is identified that the location corresponds to the intervertebral disks.



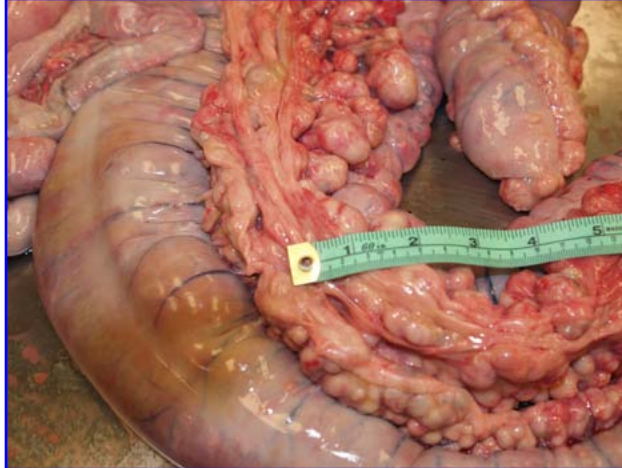
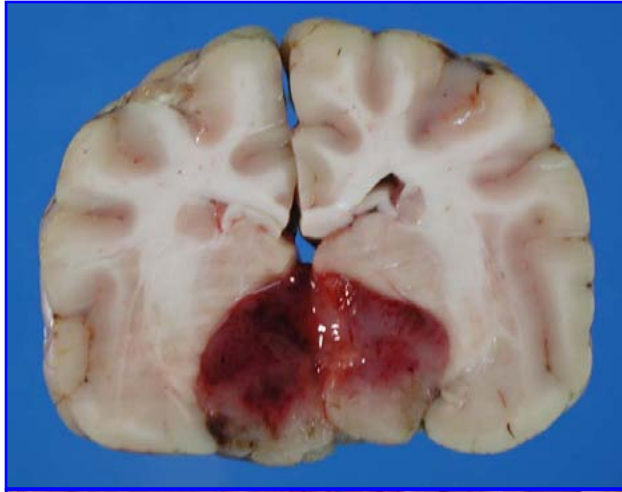
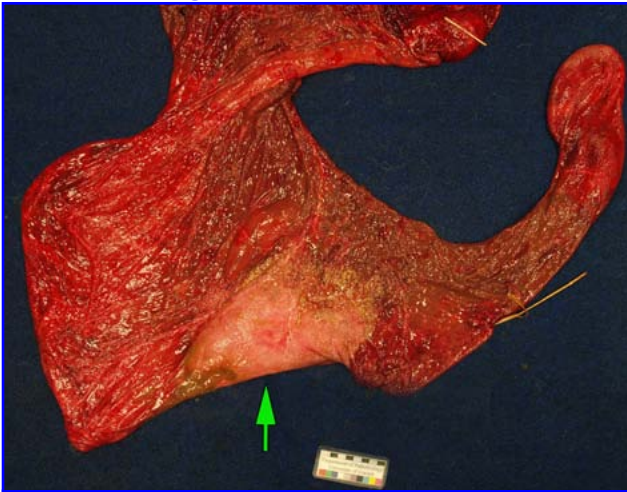
Cat, small intestine. The white lesions are pyogranulomas, in a linear arrangement that corresponds to lymphatic vessels in the mesentery. Lymphangitis, FIP.



Distributions corresponding to specific anatomic structures

The diagnosis in each of these cases requires identification of the specific anatomic structure affected by the lesion:

- Top left: Equine, *Nocardia* placentitis. The pale area of necrosis and inflammation affects the area adjacent to cervix, suggesting that the pathogen entered the uterus via the cervix and vagina.
- Bottom left: Aborted goat kid, goitre due to iodine deficiency. Bilateral masses in the expected location of the thyroid glands.
- Top centre: Dog, pituitary adenoma. The location of the tumour corresponds to pituitary gland, and expands into hypothalamus.
- Bottom centre: Foal, colonic lymphadenitis caused by *Rhodococcus equi*. Recognizing the mesocolonic masses as lymph nodes is key to the diagnosis.



Caribou, distal limb. The pus-filled tracts correspond to the joints and tendon sheaths, and this explains the distribution of the lesion in the distal limb.



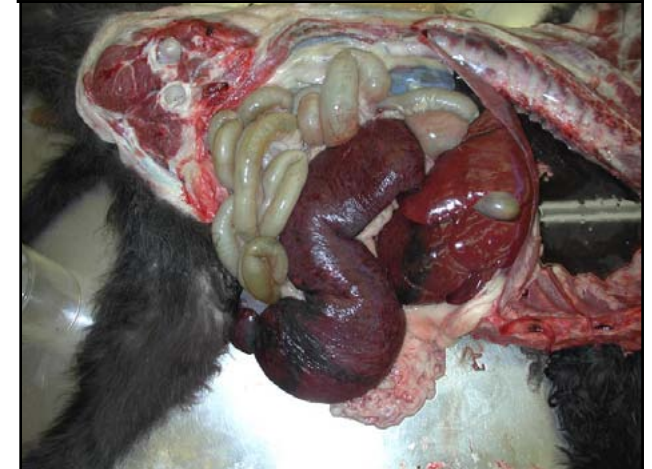
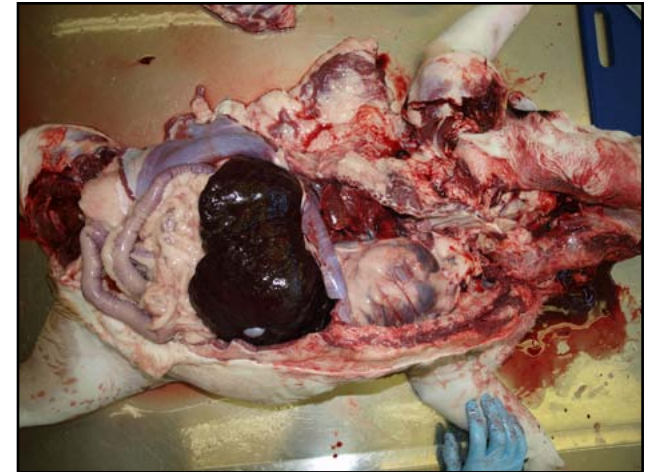
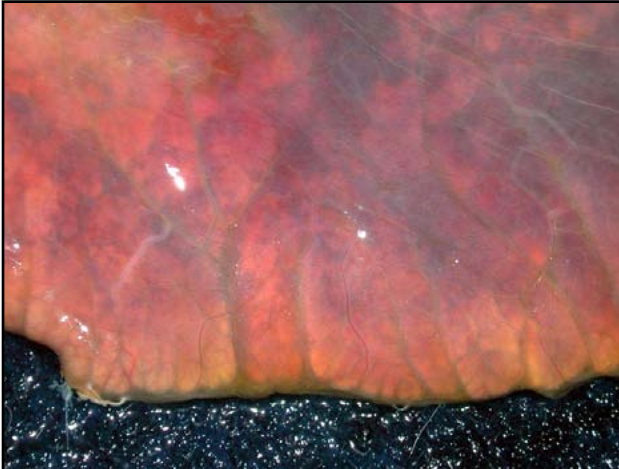
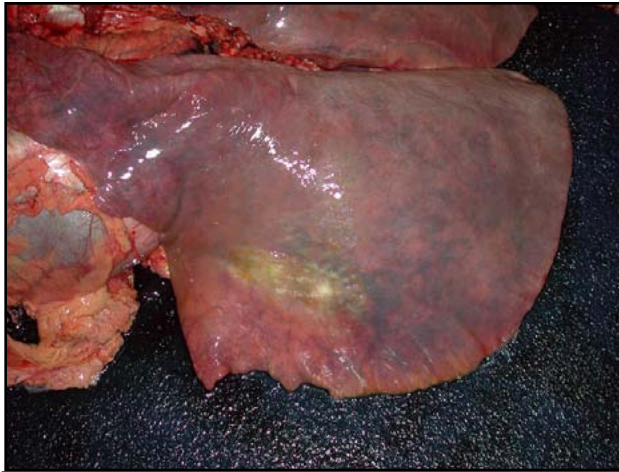
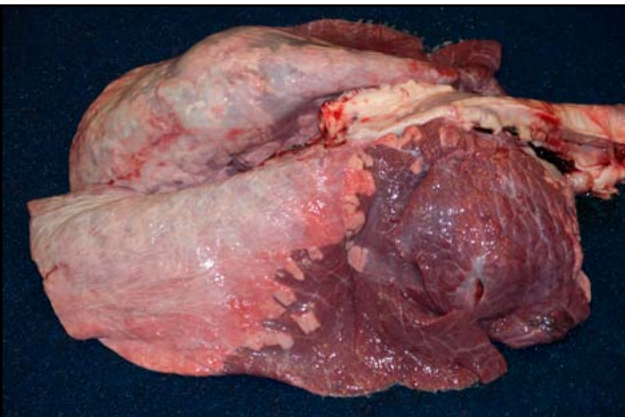
Size & extent

Measures of the size and extent of lesions might include the absolute dimensions (for example, in centimeters), the weight of the affected organ or lesion, or the percentage of the tissue that is affected by the lesion. Each of these measurements provides information of different value, which will be more or less useful depending on the context.



Weight of the affected tissue, compared to normal, is highly effective for communicating the size of these lesions. Middle: Horse, post-surgical pulmonary edema. Below right: Dog, enlarged congested liver due to right heart failure. Bottom right: Dog, splenomegaly due to leukemia.

These two lesions are nearly identical except for the percentage of lung tissue affected. In organs where size or mass is relevant to function—lung, liver, kidney, intestine, endocrine glands—it is essential to state the percentage of tissue affected.



Size and extent



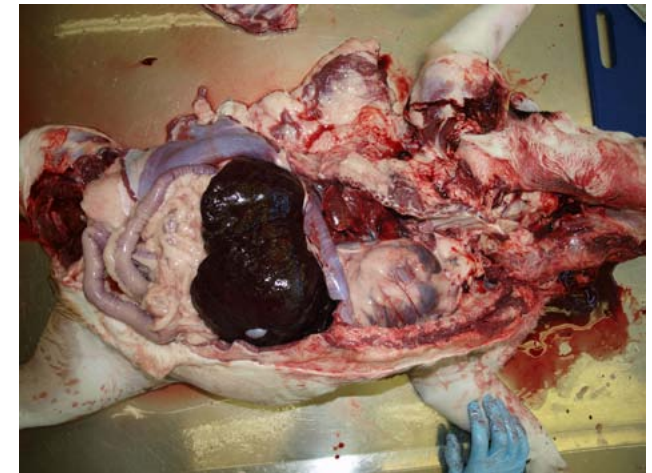
Describing the size of a tissue in relation to a structural reference can be useful.

Above: The size of the adrenal cortex might be described as 2-3 mm thick, but in this case it would be more useful to describe the ratio of cortex: medulla as 1:3 compared to normal of 1:1. Dog, adrenocortical atrophy with hypoadrenocorticism.

Recognizing abnormalities requires some knowledge of the size of normal organs, which is mainly gained by experience. Alternatively, stating the absolute measurement provides a record of the findings, which can later be compared to species- and age-matched controls or to published reference values.

Below: Bulldog, tracheal hypoplasia. The trachea has outer and inner diameters of 5 and 10 mm respectively, with overlapping of the ends of the tracheal cartilage. The laryngeal lumen is narrowed to 5 mm with 2-fold thickening of the mucosa.

Note that these findings represent the dimensions in death, but muscular contraction may have held these structures open in life.



Above: The liver is enlarged by about 50%. Dog, hepatic congestion and hepatomegaly due to right heart failure.

Below: The spleen is diffusely enlarged two-fold. Dog, leukemia.

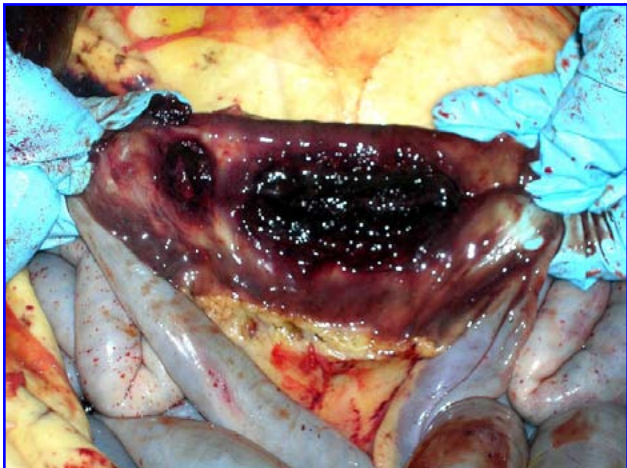


Shape

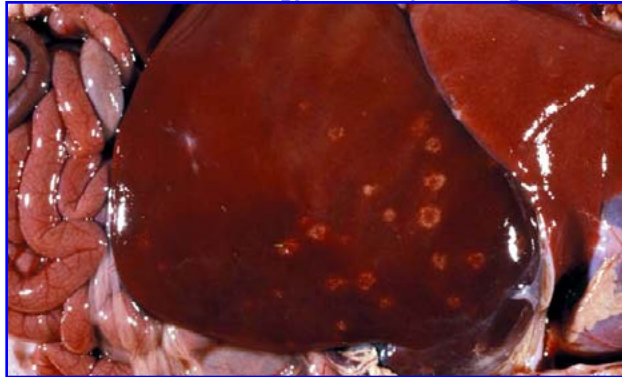
Umbilicate lesions—nodules with depressed centres—are characteristic of carcinomas. Dog, liver, cholangiolar carcinoma.



A lesion's shape may be dictated by the anatomic structure it arises in. Calf, small intestine, Peyer's patch necrosis and hemorrhage due to BVDV infection.



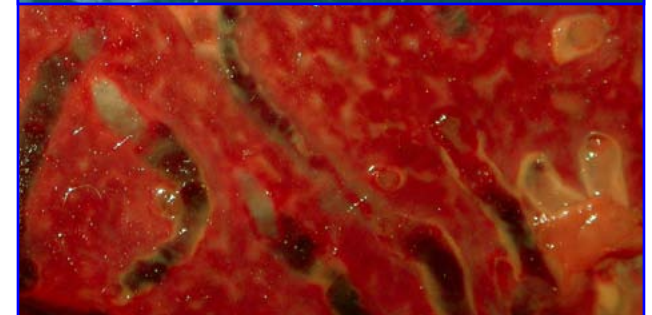
Target lesions usually represent 2 distinct pathologic processes: in this case, a collapsed red centre of necrosis, and a white rim of leukocyte infiltration. Aborted lamb, *Campylobacter fetus* ssp. fetus.



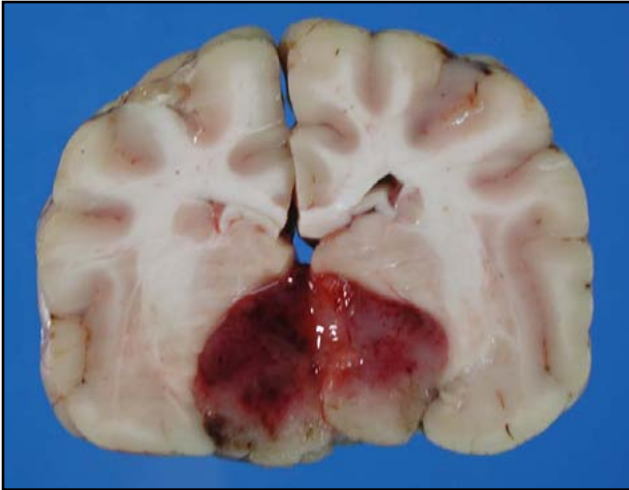
Triangular lesions usually have a vascular basis: this renal infarct resulted from thrombosis of an arcuate artery, and subsequent ischemic damage.



Serpiginous lesions may be inflammation following the course of vessels, but these tracks in the liver of a lamb are necrosis and hemorrhage due to migration of larval cestodes: *Cysticercus tenuicollis*.



Demarcation



Lesions may be well demarcated because:

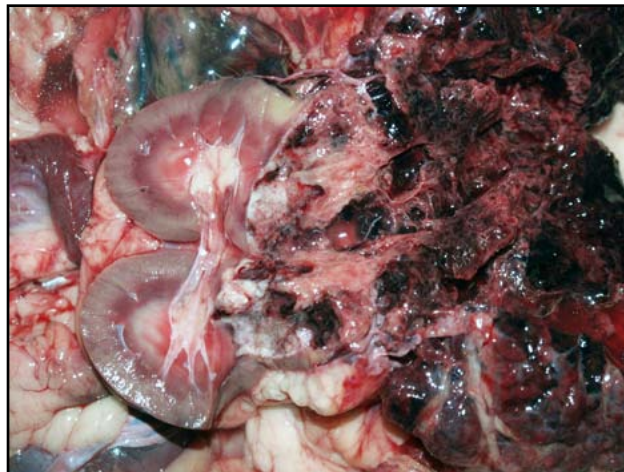
- they grow by expansion, not infiltration (above, dog, pituitary adenoma)
- are contained within a capsule or anatomic structure
- of the manner in which they arise (below, horse, intestinal venous infarction due to strangulating lipoma).



Poorly demarcated white foci represent expanding areas of inflammation. Pig, kidney, *Leptospira pomona*.



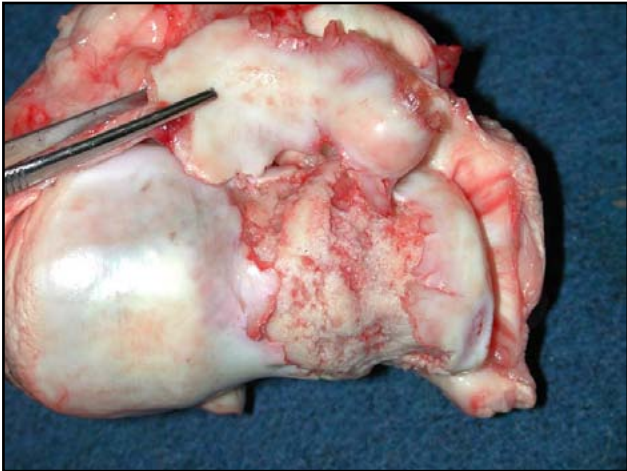
Demarcation may not provide definitive indication of malignancy. Both tumours below are malignant: the renal adenocarcinoma on the left aggressively infiltrates the peri-renal tissue, while the well-demarcated mast cell tumour on the right is a metastasis to kidney.



Surface contour: depression

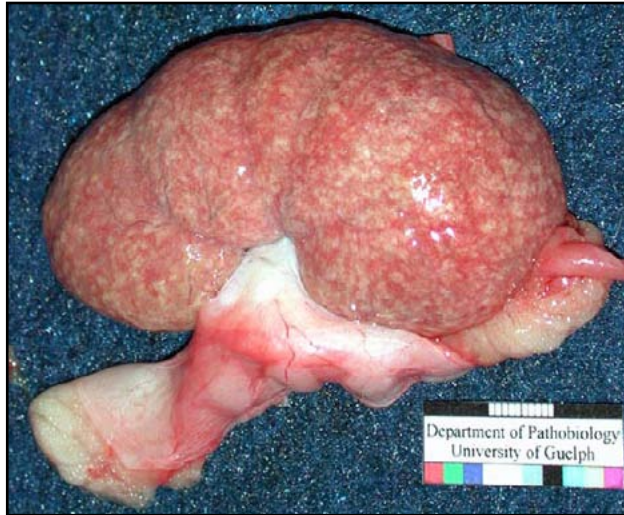
A depressed surface contour results from loss of tissue, shrinkage due to atrophy, or contraction of maturing fibrous tissue.

A raised surface contour represents something added to the tissue: inflammatory, neoplastic or hyperplastic cells; edema, blood or air.



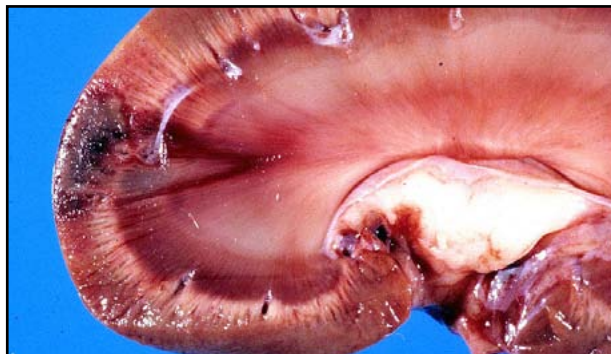
Above: Depressed contour of the joint surface from loss of cartilage tissue. Horse, osteochondritis dissecans.

Below: Sunken eyes in a dehydrated neonatal calf with diarrhea, because the retro-orbital tissue is shrunk from dehydration.

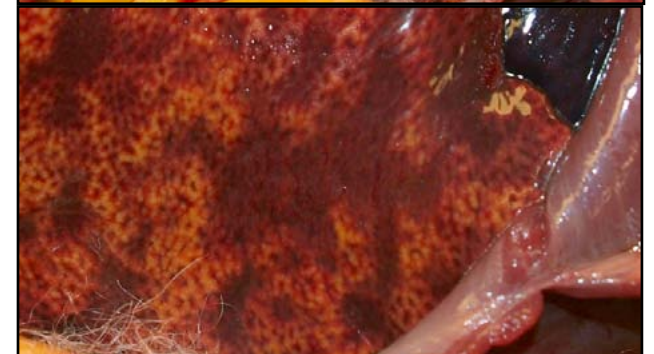


Above: Fibrosis causes depression of the white areas, compared to the more normal pink tissue. Dog, chronic renal disease.

Below: Necrotic tissue is usually flat, as in this case, but may be slightly raised due to cell swelling, or slightly depressed due to cell necrosis. Dog, renal infarct.



Dog with acute hepatic necrosis due to adverse drug reaction. Which is more normal: the raised variegated areas, or the uniformly depressed ones? Since the lesion is known to be acute, the collapsed and diffusely plum-brown areas must be necrosis, not fibrosis. The raised areas have a zonal pattern of orange surviving tissue and plum-brown necrotic tissue.



Surface contour: elevation



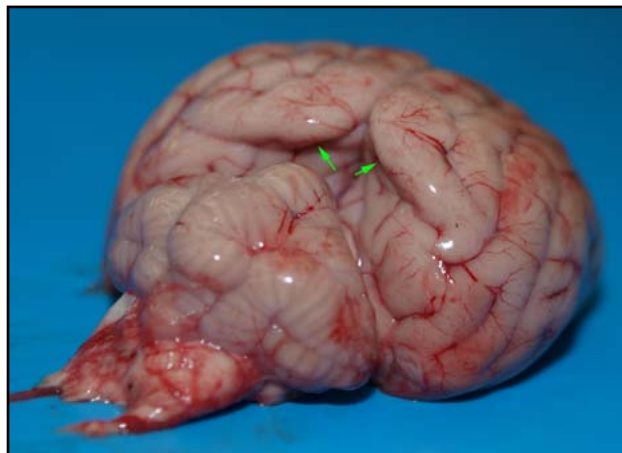
Raised surface contours may result from the presence of inflammatory cells, neoplastic cells, or hyperplastic cells.
Top left: Kidney, pyogranulomatous inflammation caused by feline infectious peritonitis.

Middle left: Renal neoplasm, metastatic mast cell tumour, cat.

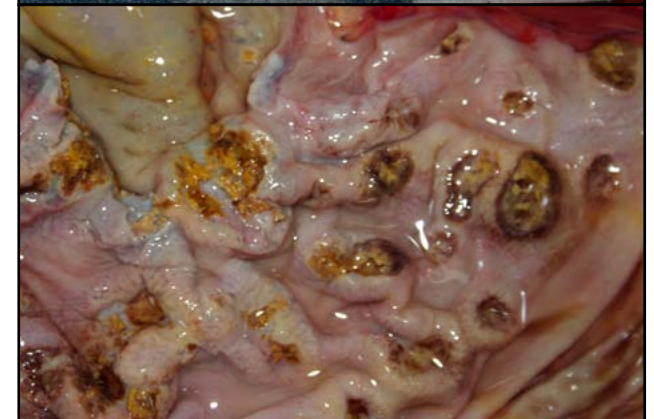
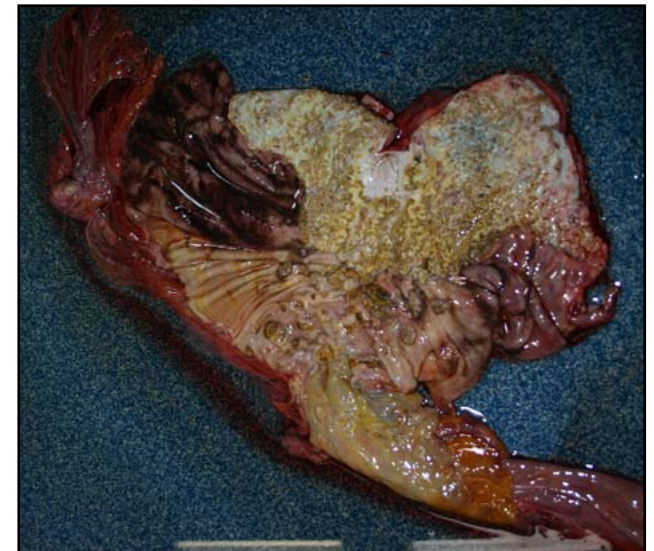
Bottom left: Virus-induced epithelial hyperplasia, with central depressed foci of necrosis. Esophagus, bovine papular stomatitis caused by parapox virus.



Below: Brain, dog with granulomatous meningoencephalitis. Edema causes brain swelling, which results in herniation of the occipital cortex (arrows) and coning of the cerebellum.



Below: Lesions can be both raised and depressed! Nodular expansion of the gastric mucosa due to eosinophil infiltration, with ulceration forming craters in the centre of the nodules. Horse, “multisystemic eosinophilic epitheliotropic disease”.



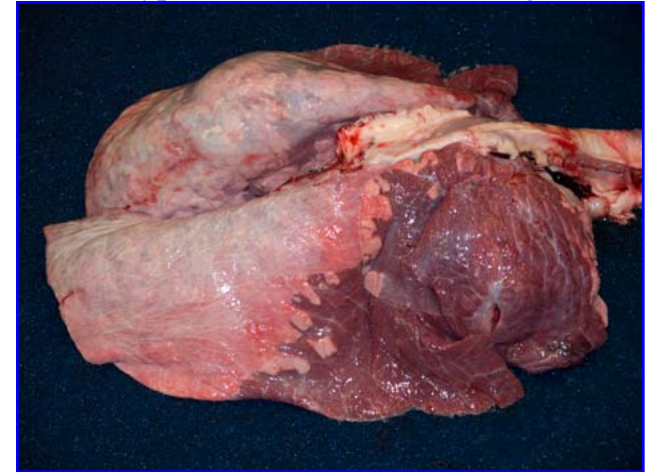
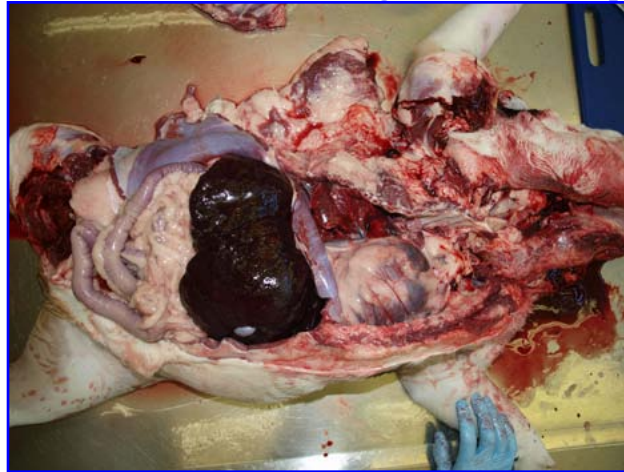
Colour: red

Mechanisms for accumulation of blood in tissues include hemorrhage, congestion, and hyperemia.

Left: Coalescing hemorrhages on the intestinal serosa, due to both thrombocytopenia and vascular damage. Dog, disseminated intravascular coagulation due to immune hemolytic anemia.

Centre: Diffuse congestion of the liver due to right heart failure. Dog, anomaly causing obstruction of pulmonary blood flow.

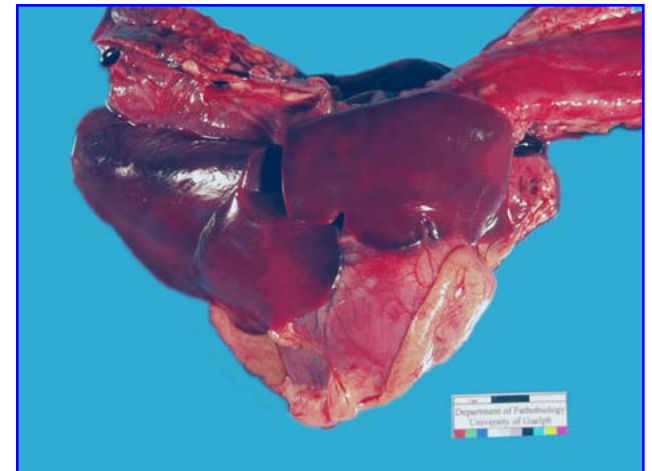
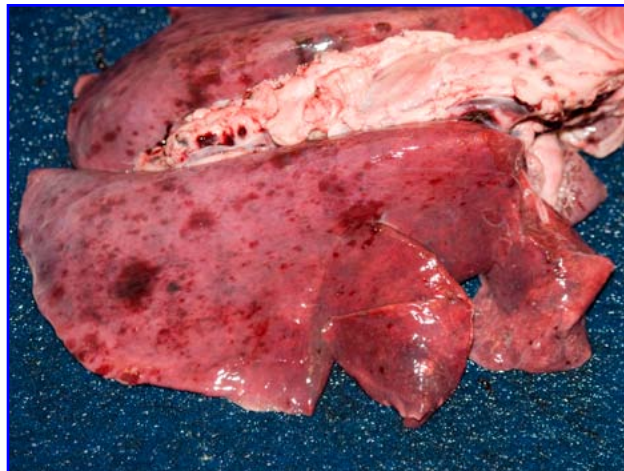
Right: Hyperemia due to inflammation, in the cranioventral area of lung. Calf, bronchopneumonia typical of *Mannheimia haemolytica*.



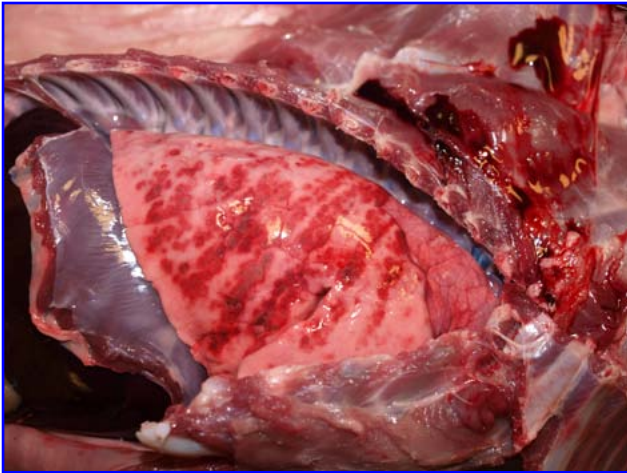
Hemorrhages are usually multifocal or patchy in distribution, whereas congestion or hyperemia are diffuse within the affected area. Extensive hemorrhage may, however, cause diffuse reddening. It is not possible to objectively distinguish congestion from hyperemia by examination of non-living tissues.

Below left: Multifocal red lesions indicative of hemorrhages. Lamb, lung, vascular injury from *Pasteurella trehalosi* septicemia.

Left bottom: Diffuse reddening due to congestion or hyperemia. Dog, lung, diffuse alveolar damage.



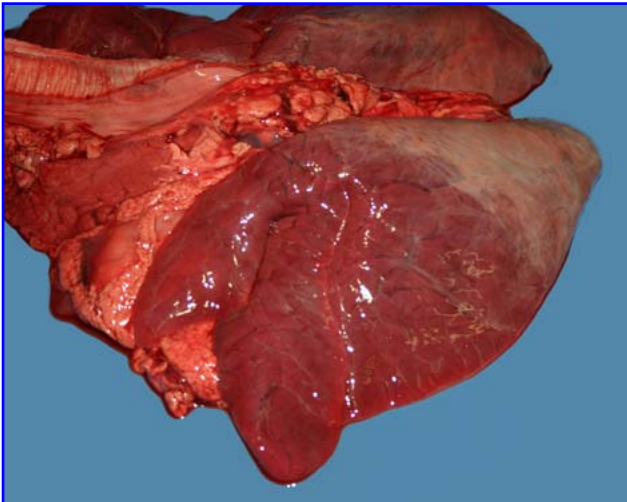
Colour: red



Two additional reasons for redness.

Above: Aspiration of blood into the lung from the upper respiratory tract, at the time of euthanasia. Pig.

Below: Fetal atelectasis: the absence of air intensifies the redness of the lung.



Patterns of hemorrhage into infarcts.

Above: In lung, spleen, liver and adrenal gland, arterial infarcts appear red because of hemorrhage into tissue spaces or sinusoids. Dog, lung, sepsis secondary to parvoviral enteritis.

Below: Arterial infarcts in most tissues appear pale, but may be delineated by a rim of hemorrhage. Dog, kidney.



Above: Blood fills the thoracic cavity, compressing the lungs. Note the pallor of the oral mucosa. Dog, Hemothorax due to anticoagulant rodenticide toxicity.

Below: Ecchymotic hemorrhages from vascular damage. Horse, endotoxemia secondary to bacterial colitis.



Colour: tan and white

Causes of pale, tan or white lesions:

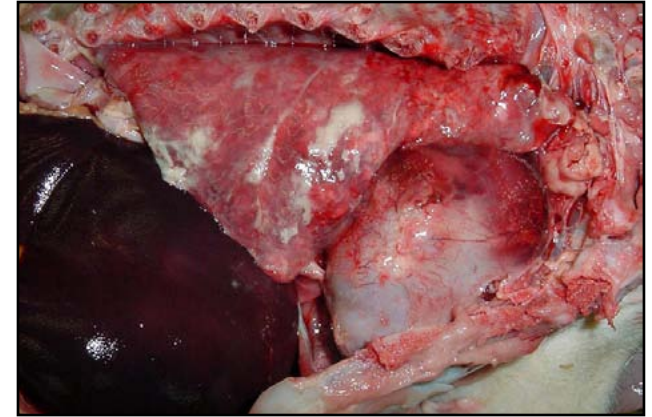
- Addition to of white cells: leukocytes (inflammation), neoplastic cells, epithelium. Lesions may be raised (or flat).
- Addition of white substances: fibrin, fibrous tissue, mineral, lipid, glycogen, urate, nucleus pulposus
- Cell swelling: tissue necrosis. Lesions may be depressed (or flat), and friable (or of normal strength).
- Removal of blood or other pigments: anemia, hypovolemia



Above: Kidney, septic infarct. Hemorrhage and necrosis, with white rim of leukocytes
Below: Pale nodules in liver, which are metastatic sarcoma. Dog.



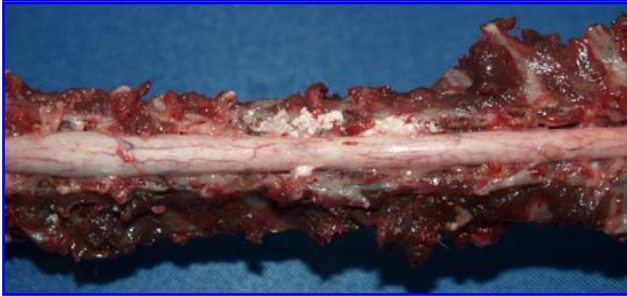
Above: Tan nodules of pyogranulomatous inflammation. Cat, kidney, FIP.
Below: Raised white rims of virus-induced epidermal hyperplasia. Piglet, pox virus.



Above: White fibrin on pleura. Pig, *Haemophilus parasuis* infection.
Below: Hepatic pallor due to fibrosis and leukocytes. Dog, cholelithiasis & cholangiohepatitis.



Colour: tan and white



Top: Mineralized intercostal muscle. Dog, chronic renal failure.

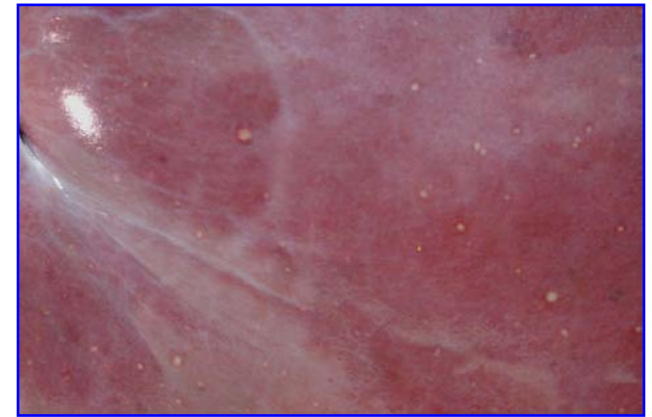
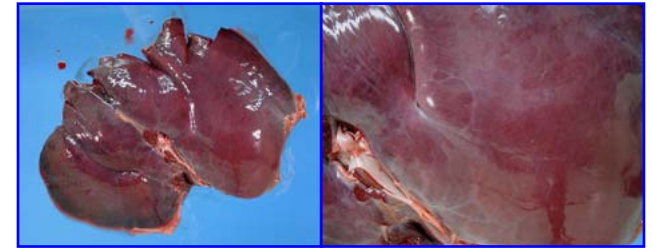
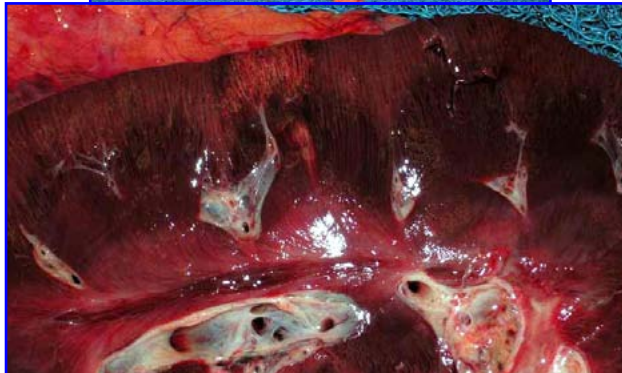
Above: Extrusion of nucleus pulposus.

Below: Hepatic lipidosis. Cat.



Above: Urate crystals cover the epicardium. Falcon, visceral gout.

Below: Focus of pallor with a red margin: necrosis due to infarction, in the renal cortex. Horse, sepsis from bacterial colitis.



Above: Kidney, septic infarct. Hemorrhage and necrosis, with white rim of leukocytes

Below: Pale nodules in liver, which are metastatic sarcoma. Dog.



Colour: yellow

Yellow discoloration of tissues usually represents bilirubin. This is most prominent in jaundice (icterus) as a result of hepatic disease, biliary obstruction, or hemolysis. Below left are examples of mild and severe jaundice, in dogs with liver disease.



The subcutaneous and adipose tissues of horses are often slightly yellow, reflecting the normally higher levels of bilirubin in this species.

Edematous tissues and fibrin may be yellow, as is evident in the area of localized edema in the horse leg below.



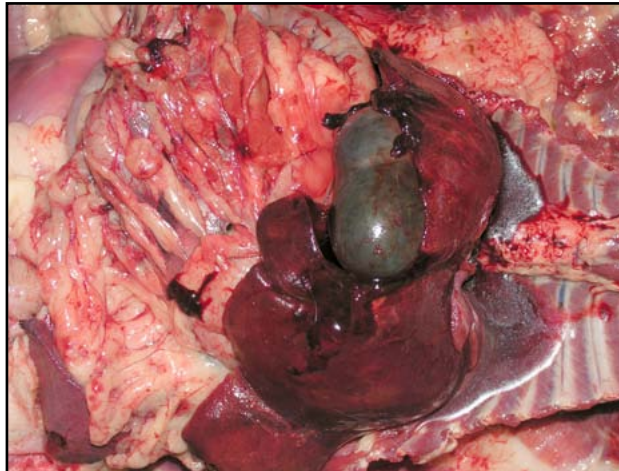
Colour: yellow & green

Green colour in fresh tissues may represent bile, or sometimes eosinophils. Green or yellow discoloration is common near the gall bladder in autolysed carcasses. Putrefaction causes green discoloration of tissues after death.

Below: Dog, acute myeloid leukemia.
Masses in lymph nodes and other tissues are obviously green, as a result of heme pigment in myeloperoxidase, an enzyme in the granules of the neoplastic myeloid cells.

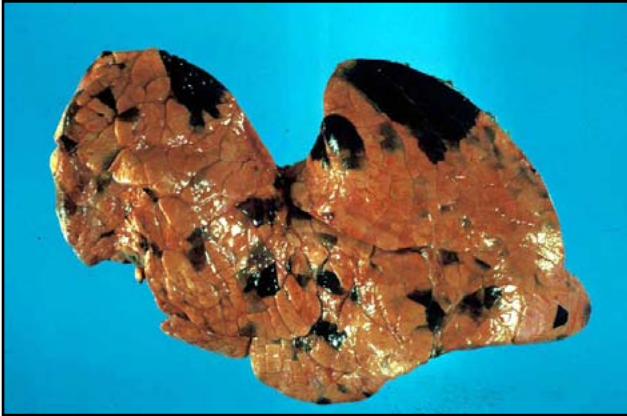
Click on the image to scroll through the photos.

Below: Dog, gall bladder mucocele.
Distention of the gall bladder with inspissated bile causes green coloration.
Bottom: Inspissated bile in a mucocele.



Colour: black

Substance causing black discoloration of tissue include melanin, oxidized hemoglobin such as from digested blood, carbon particles, and the excrement of trematode parasites.



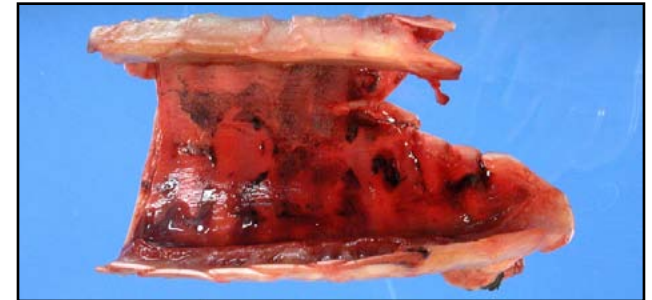
Above: Bovine lung with a lobular distribution of melanosis, which represents unusual pigment deposition of no significance.

Below: Melanotic and amelanotic tumours. Dog lung, metastases of oral malignant melanoma.



Above: The colon contains black tarry content, which is melena as a result of digestion of blood originating from a small intestinal tumour. Dog.

Below: Tiny black-red nodules in the cerebrum. Dog, metastatic hemangiosarcoma.



Above: Black carbon (soot) on the tracheal mucosa of a horse that died in a barn fire. This finding indicates the animal was alive and breathing at the time of the fire.

Below: Black tracts in the liver. Sheep, *Fasciola hepatica*. Adult flukes are visible in the bottom photo.



Texture

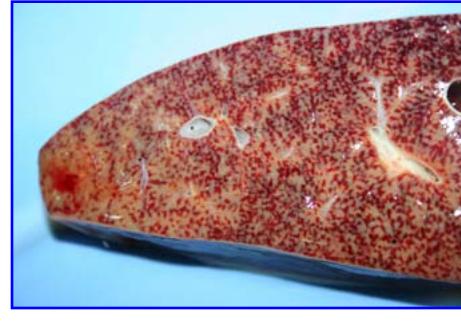
Assess firmness on a 4-point scale: soft, rubbery, firm, and hard. Dr. T. van Winkle of Penn State University has a memorable mnemonic, the facial test: the forehead is hard, the nose is firm, the lips are soft.



Above: Soft texture of subcutaneous edema. Cat, anuric renal failure with excessive intravenous fluid administration.



Above: Rubbery texture of lung affected by idiopathic interstitial pneumonia. Firmer than normal lung, but softer than liver. Dog.



Above: Firm texture of fibrotic liver. Bull, chronic right heart failure, "nutmeg liver".

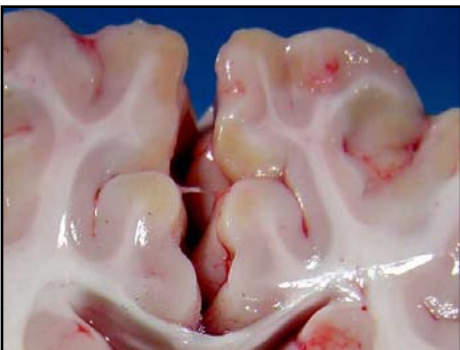


Top: Hard texture of new bone, at the edge of the joint. Cow, chronic arthritis.

Above: Deer, fracture repair.

Below: Hard texture and "playing jacks" shape of silica calculi. Dog, urinary bladder. Ouch!

Below: Yellow cortical lesions are **malacia**: softening compared to normal brain tissue. Lamb, cerebrum, polioencephalomalacia.



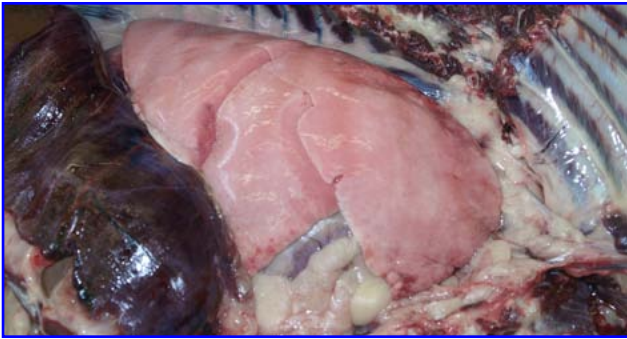
Below: Rubbery texture of a metastatic mast cell tumour. Cat, kidney.



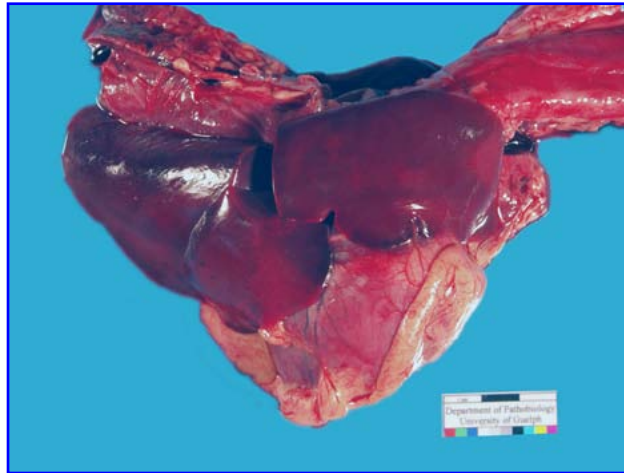
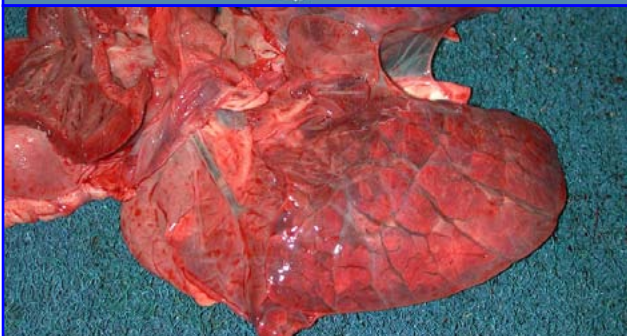
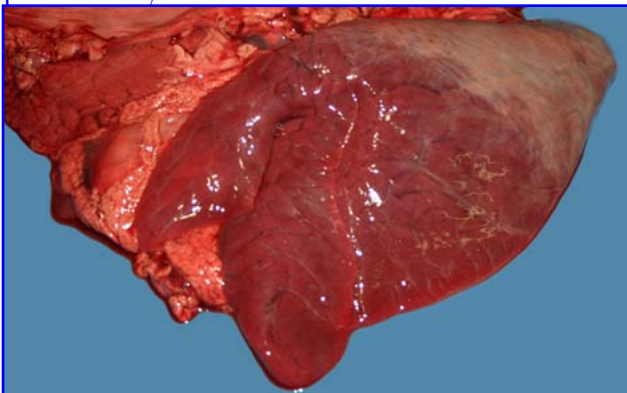
Below: Firm texture of a cholangiolar carcinoma: The neoplastic cells provoke a "schirrhous response", with formation of non-neoplastic fibrous tissue.



Texture: varying degrees of firmness, applied to the lung

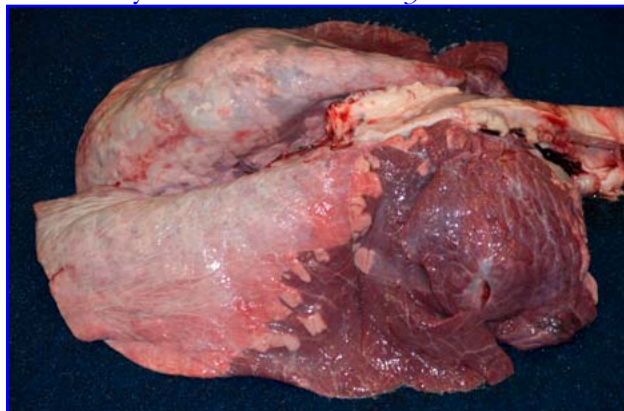


Above: Soft spongy texture. Normal dog.
Below: Soft but not spongy. Bovine, fetal atelectasis: absence of air in the lung.
Bottom: Soft but slightly rubbery. Pig, pulmonary edema due to heart failure.



Above: Rubbery texture of diffuse interstitial lung disease. Firmer than normal, but still softer and spongier than liver. Dog, post-traumatic acute respiratory distress syndrome.

Below: Cranioventral area is firm, as the air spaces are filled with neutrophils and edema. Calf, subacute bronchopneumonia, caused by *Mannheimia haemolytica*.

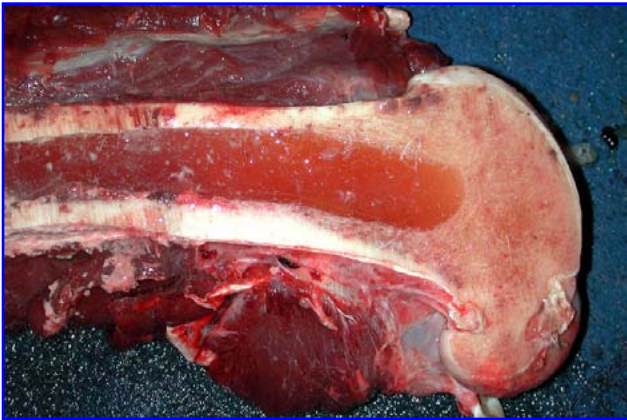


Above: The ventral areas are very firm, and cut crisply with a knife, because each individual air space is filled with polymerized fibrin. Calf, acute fibrino-suppurative bronchopneumonia, *Mannheimia haemolytica*.

Below: Cut section, same disease as above. There is a lobular pattern of variegation: fibrin and neutrophils make some lobules pale; others are purple from hemorrhage.



Texture



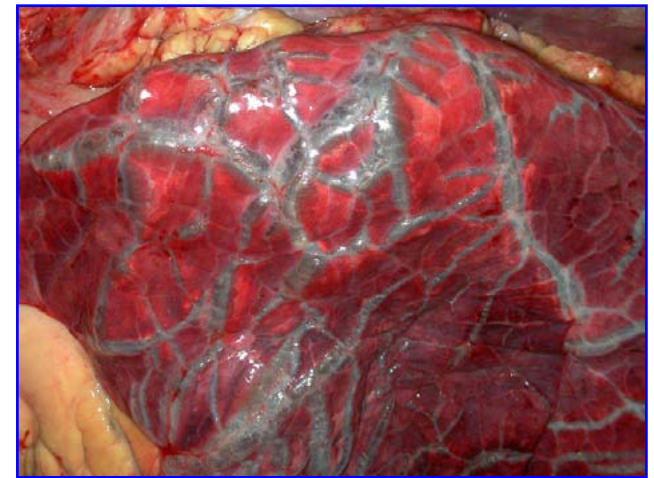
Above: Gelatinous texture of the bone marrow, due to serous atrophy of fat. The fat has been metabolized, but the remaining protein confers the jelly-like texture. Elk, starvation.

Below: Gelatinous texture of mucus-rich inspissated bile. Dog, gall bladder mucocele.



Above: Spongy texture. Cat, polycystic renal disease.

Below: Spongy texture. Cat, biliary cystadenoma. This liver tumour is formed by innumerable tiny fluid-filled cysts. The blue discoloration is surgical ink, used to identify the margins of the excision.



Above: Crackly texture of emphysema, from air bubbles in the interlobular septa. Cow, alveolar rupture due to dyspnea, a consequence of aspiration pneumonia in other areas of the lung.

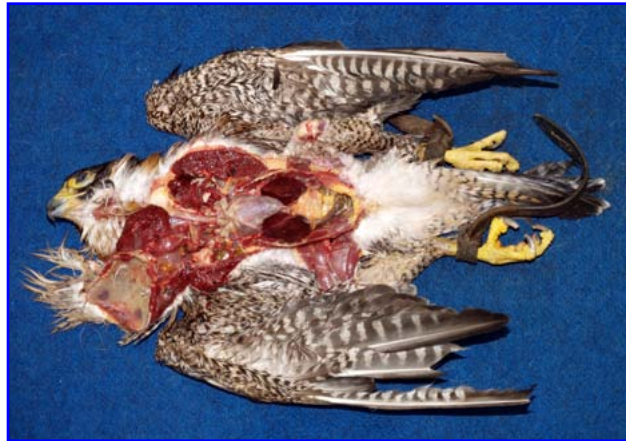
Below: Turgid texture of a fluid-filled viscus. Cat, liver, biliary cyst.



Texture



The gritty texture of tissue mineralization is detected when the white areas in this muscle are cut with a knife. Dog, widespread mineralization due to secondary hyperparathyroidism, chronic renal failure.



Urate crystals covering the epicardium have a gritty texture. Falcon, visceral gout secondary to renal failure.



Crusty texture, due to dessication of keratin covering the hyperplastic epithelial masses. Wild turkey, pox viral infection.

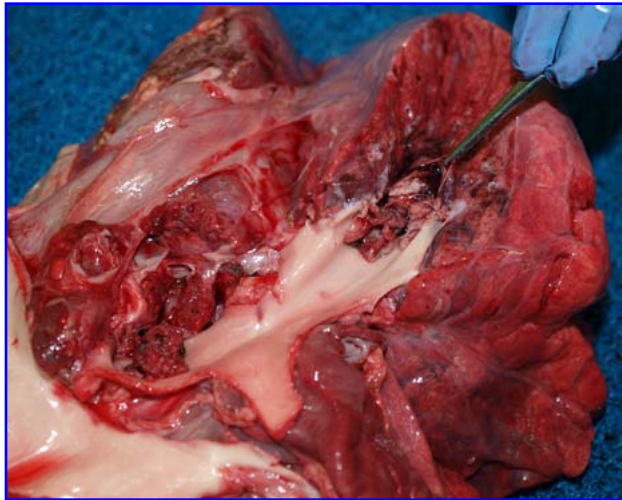
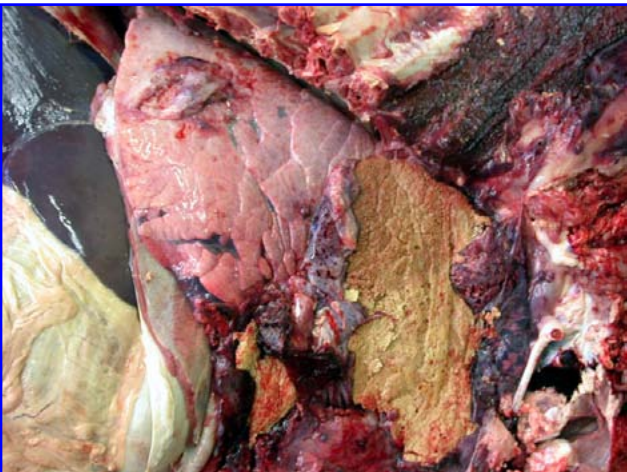


Strength



Above: Coalescing foci of caseous necrosis, each comprised of crumbly dry friable white material. The friable nature results from dissolution of the tissue stroma. Calf, lung slice, caseonecrotic bronchopneumonia, *Mycoplasma bovis*.

Below: A sequestrum of necrotic tissue occupies the cranial half of the right lung. The tissue is friable, and tears easily. Calf, *Mycoplasma bovis*.

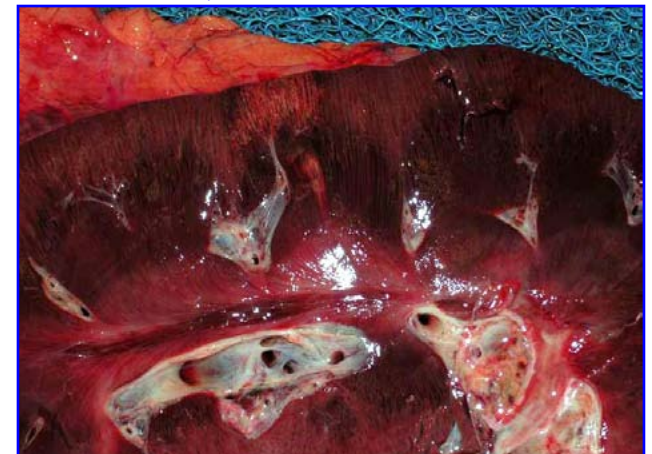


Above: A thrombus, composed of fibrin and platelets, is resilient and elastic but lacks strength. Calf, pulmonary thromboembolism secondary to bacterial endocarditis.

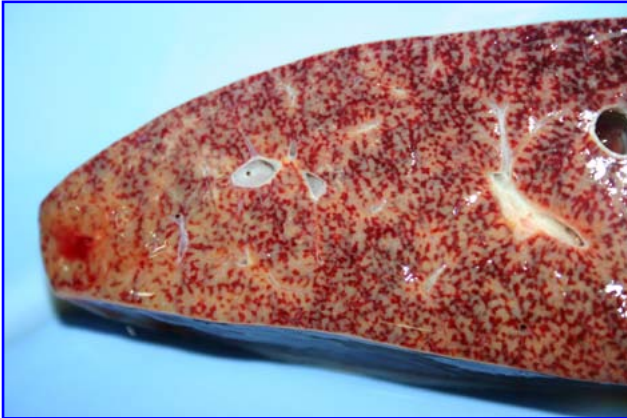


Above: Skin fragility syndrome. Loss of skin strength from weakening of dermal collagen. Cat, hyperadrenocorticism.

Below: Necrotic tissue is expected to be friable, but it is not detectably so in this pale focus of coagulation necrosis, because the tissue architecture and stroma remain intact. Horse, renal infarct.



Strength

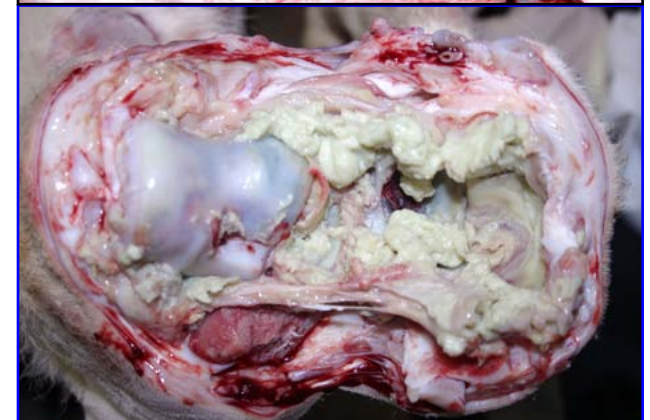
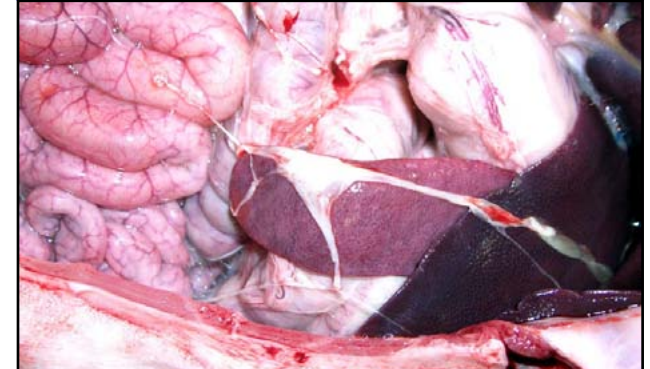
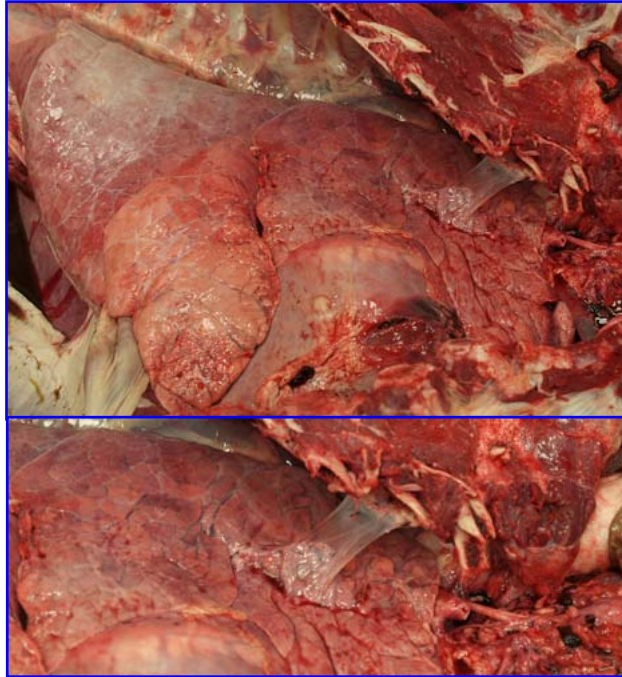


Above: Firm digital pressure is enough to crumble normal liver tissue, but this liver is remarkably tough due to fibrosis. Bull, chronic right heart failure.

Below: The pericardium (arrow), which is firmly adherent to the heart by tough fibrous tissue, cannot be peeled away without tearing the cardiac tissue. Pig, chronic fibrous tissue “organization” of an earlier fibrinous pericarditis.



Below: Fibrous adhesions of the cranial lung lobe to the parietal pleura. These tough fibrous bands represent organization of a prior fibrinous pleuritis. Cow.



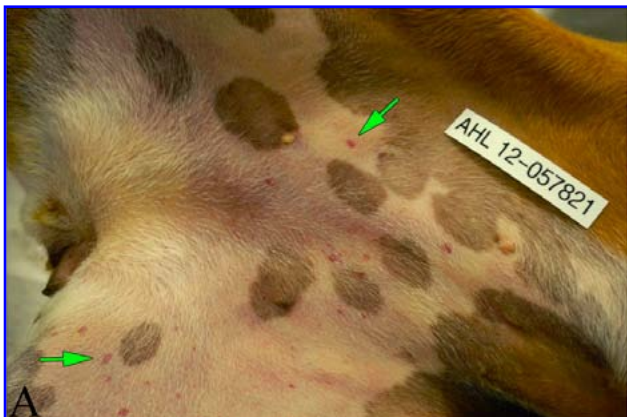
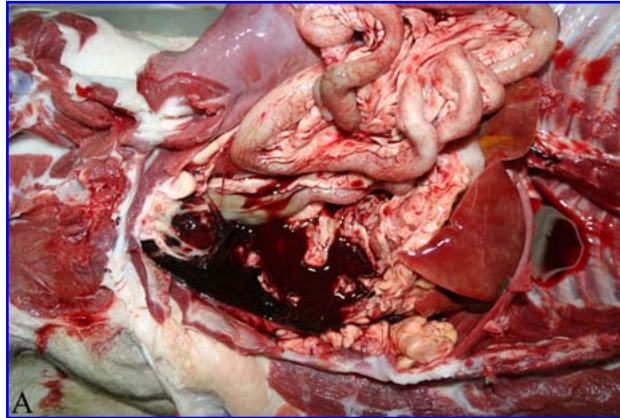
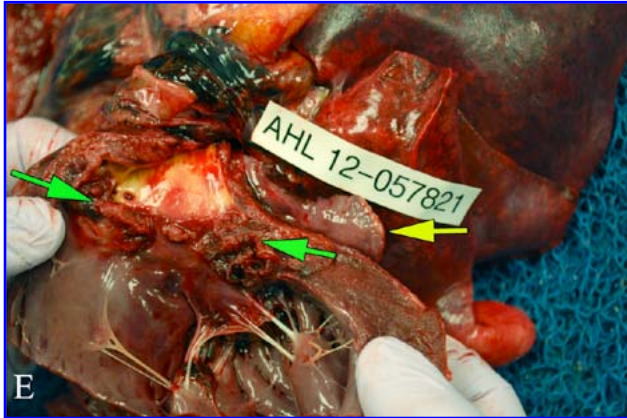
Exudates in body cavities are fluids or semi-solids that lack strength.

Top right: Tenacious liquid pus in the pastern joint of a caribou.

Middle right: Strands of fibrin, covering the spleen and liver in this case, are elastic but break easily. Pig, *Haemophilus parasuis*.

Lower right: Semi-solid mixture of fibrin and pus. The material has the texture of cheese curd, and pulls apart just as readily. Calf, carpus, *Mycoplasma bovis* infection.

Part IV. Are Necropsy Examinations Useful to Practitioners?



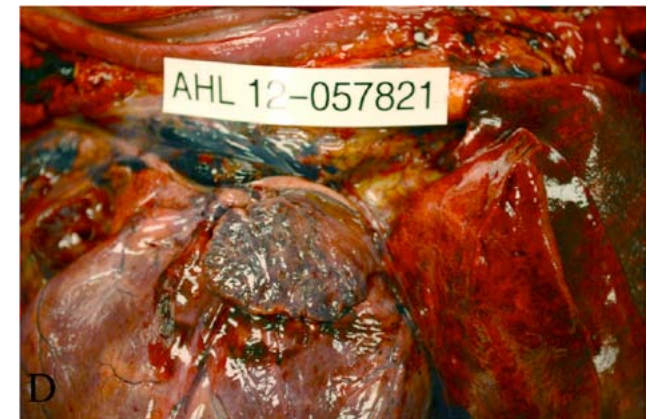
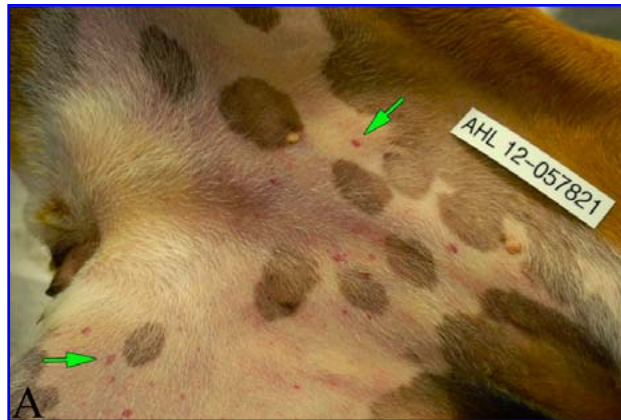
Case A: Observations

A five year old Boxer dog presented for necropsy with the following clinical history:

- April: Surgery for sialoceles removal. Meloxicam, tramadol and cephalexin were given post-operatively.
- June 18: The dog presented to the practitioner for evaluation of an acute onset of lameness in the right hind limb, with pyrexia of 40.4°C. Radiographs were non-diagnostic, but suggest an injury of the anterior cruciate ligament.
- June 19-20: The dog seemed better, and was eating and drinking
- June 21: The dog became very lethargic, and developed cutaneous and oral petechiae, jaundice. The heart stopped, restarted briefly after administration of CPR, but the dog died soon thereafter.

Examine the photos and consider the following questions, before turning the page:

1. What is the morphologic diagnosis for the lesion in figure 'E'?
2. What is the reason for petechiation?
3. What is the relationship between this lesion and clinically observed lameness?
4. Would clinical diagnosis have changed the course of therapy?



Orientation to the photos:

A: Ventral abdomen. B: Lips. C: Pluck. The left atrium is visible, and the aorta is deep to it, along the trajectory indicated by the asterisks. D: Left atrium. E: Opened left ventricle. Green arrows—aortic valve. Yellow arrow—left atrium.

AHL case# 12-057821

Case A: Interpretation

What is the morphologic diagnosis for the lesion in figure 'E' ?

Severe chronic endocarditis of the aortic valve (\pm diffuse or multifocal)

1. What is the reason for petechiation?

Probably sepsis due to bacteremia, leading to (a) endothelial damage or (b) disseminated intravascular coagulation with consumption of platelets and coagulation factors.

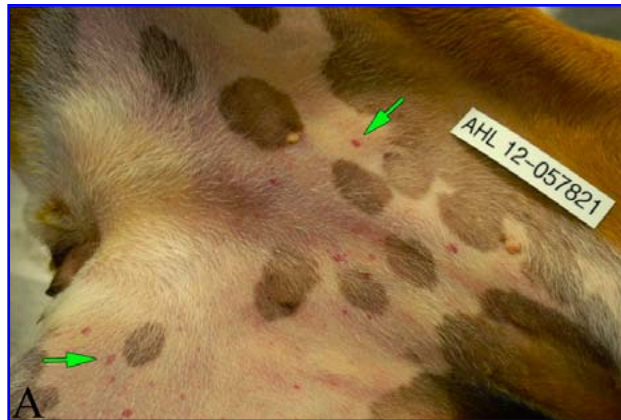
2. What is the relationship between this lesion and clinically observed lameness?

Bacterial endocarditis causes intermittent bacteremia, and localization of bacteria in the highly vascular synovium causes synovitis/arthritis and lameness.

3. Would clinical diagnosis have changed the course of therapy?

Yes, cases of bacterial endocarditis should be treated with antibiotics, preferably preceded by blood cultures to guide antibiotic selection. Nonetheless, many cases respond poorly to therapy.

Could you have performed this necropsy and made the diagnosis yourself? Yes! Necropsy examination of your own cases is a great method of continuing education.



Orientation to the photos:

A: Ventral abdomen. B: Lips. C: Pluck. The left atrium is visible, and the aorta is deep to it, along the trajectory indicated by the asterisks. D: Left atrium. E: Opened left ventricle. Green arrows—aortic valve. Yellow arrow—left atrium.

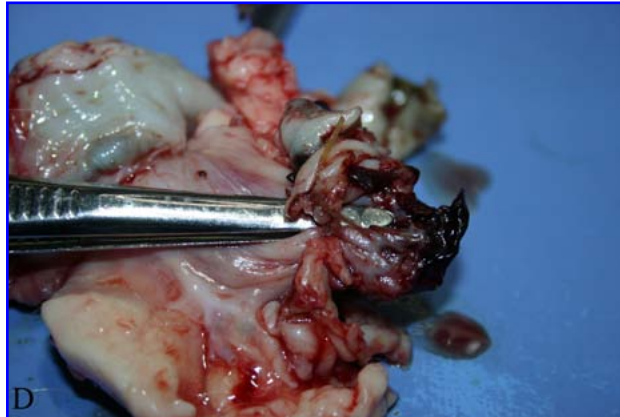
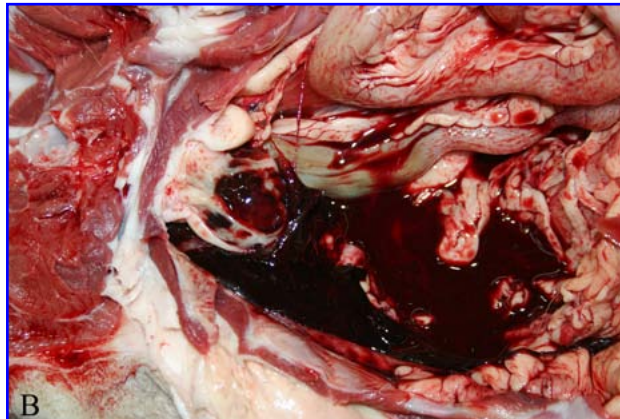
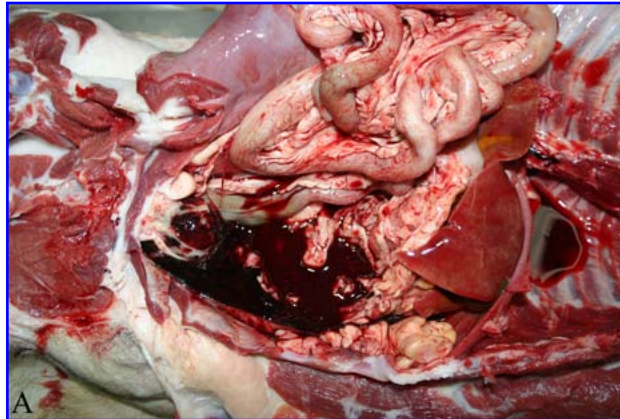
AHL case# 12-057821

Case B: Observations

These photos are from a 6 month old dog that died soon after an ovario-hysterectomy surgery. The cadaver was submitted to the diagnostic laboratory by the veterinarian, with no indication of a lawsuit, and we think that the veterinarian simply wanted an independent determination of what happened.

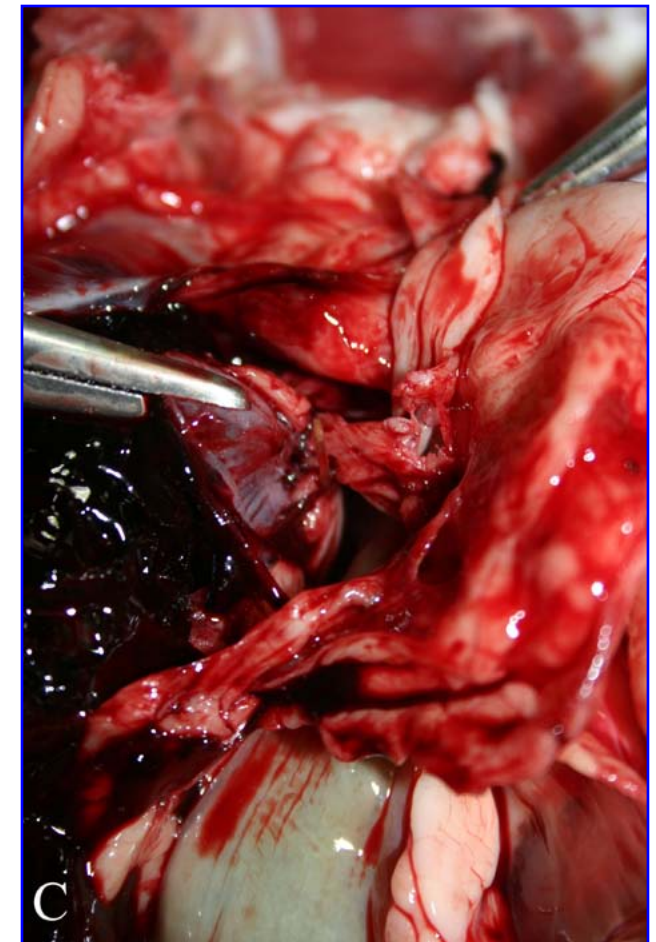
Examine the photos and consider the following questions, before turning the page:

1. What is the morphologic diagnosis for the lesion in figure 'B' ?
2. Is it plausible that this lesion was responsible for the death of the dog?
3. Based on the findings shown in figures C & D, what is the likely sequence of events in this case?
4. Is it the fault of the surgeon?
5. If you were the surgeon, what lesson would you take from this?



Orientation to the photos:

A: Opened abdomen, head to the right. B: Caudal abdomen. C & D: Cervix / uterine stump with intact ligature.



UCD Case# 13077

Case B: Interpretation

1. What is the morphologic diagnosis for the lesion in figure 'B'?

Severe hemoabdomen. The clinical history suggests this was an acute process. We have no reason to doubt this, but it cannot be objectively verified.

2. Is it plausible that this lesion was responsible for the death of the dog?

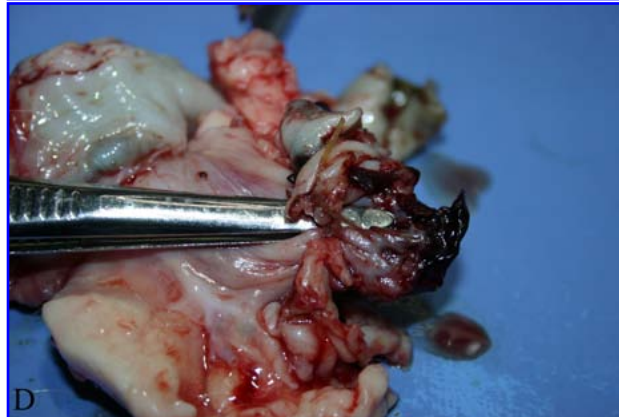
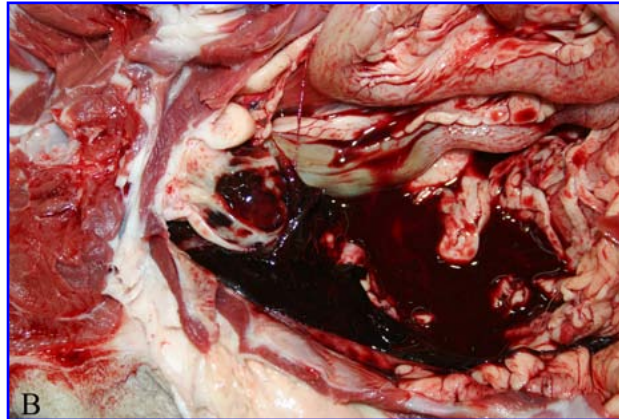
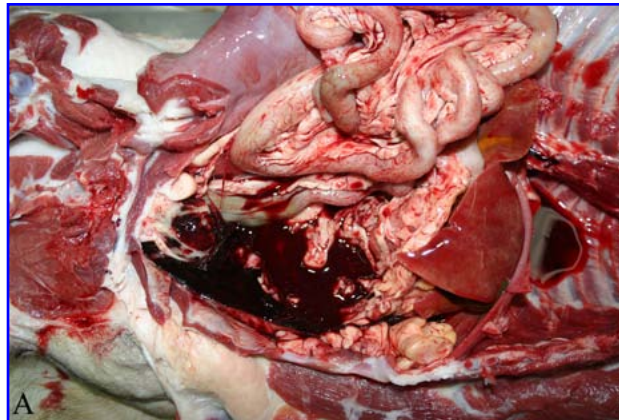
Yes. The abdomen contains about 700 mL of blood. The body weight was 15 kg. Blood volume is ~ 8.6% of body weight (1.3 litres). Loss of 30-40% of blood volume (0.4-0.5 litres) is expected to be fatal.

3. Based on the findings shown in figures C & D, what is the likely sequence of events in this case?

The ligature blood clot was adherent to the cervical stump, and this is the likely source of hemorrhage that led to death.

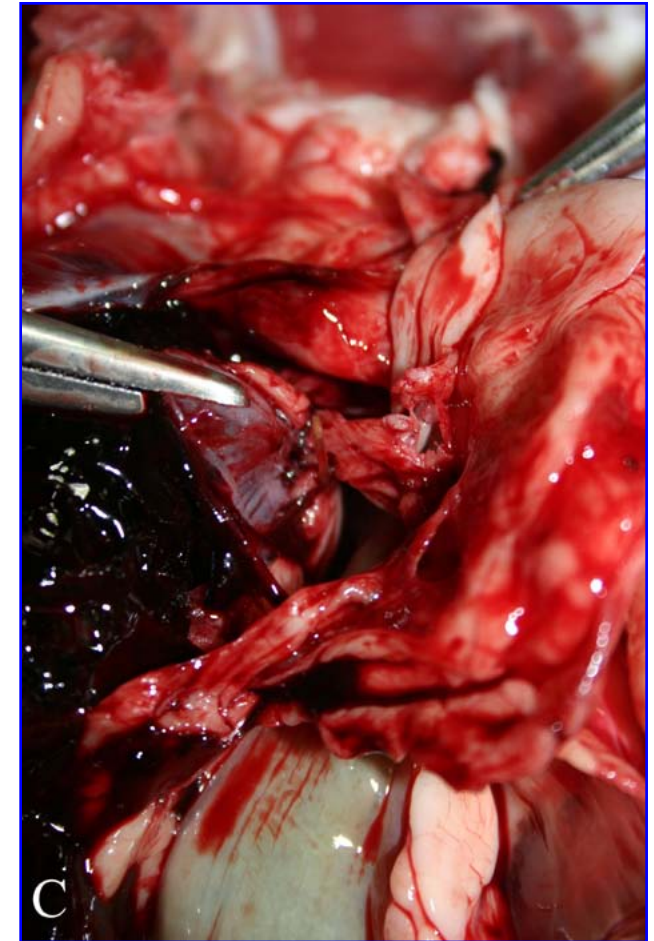
4. Is it the fault of the surgeon?

The ligature is intact, but it is very loose. We cannot rule out that some of this laxity occurred after death, or after resolution of swelling of the tissue from surgical trauma. However, it is at least as plausible that the ligature was not placed tightly enough.



If you were the surgeon, what lesson would you take from this?

Although the spay surgery is “routine”—performed daily—it is a major surgery. Work with care, on every case.



UCD Case# 13077

Case C: Observations



*H*istory of the case.

Caption.

AHL case#

Case C: Interpretation



*H*istory of the case.

Caption.

AHL case#

Case D: Observations



*H*istory of the case.

Caption.

AHL case#

Case D: Interpretation



*H*istory of the case.

Caption.

Part V. Pathologic Interpretation: Case Examples



Case 1: Questions

*H*istory of the case.

Caption.

Case 1: Answers



*H*istory of the case.

Caption.

Case 2: Questions

*H*istory of the case.

Caption.

Case 2: Answers



*H*istory of the case.

Caption.

Part VI. Appendices



Appendix 1: The vocabulary of pathology

By the end of the course, you should use and be familiar with the meaning of these terms.

- Abscess
- Adenoma
- Agenesis
- Amyloid
- Anaplastic
- Aneurysm
- Aplasia
- Apoptosis
- Ascites
- Atelectasis
- Atresia
- Atrophy
- Autolysis
- Bacteremia
- Biopsy
- Cachexia
- Carcinoma
- Cardiac tamponade
- Catarrhal
- Cellulitis
- Coagulation (coagulative) necrosis
- Congestion
- Consumptive coagulopathy
- Consolidation
- Choristoma
- Cyst
- Debridement
- Desmoplastic
- Dysplasia
- Dystrophic mineralization or calcification
- Ecchymotic haemorrhage
- Ectopic
- Edema
- Effusion
- Embolism
- Empyema
- Erosion
- Fistula
- Focal
- Friable
- Gangrene
- Granuloma
- Granulomatous
- Hamartoma
- Hematoma
- Hemorrhage
- Hemosiderin
- Hemostasis
- Hyaline
- Hyperchromasia
- Hyperemia
- Hyperplasia
- Hypertrophy
- Hypoplasia
- Hypoxia
- Iatrogenic
- Icterus
- Idiopathic
- Induration
- Infarct
- Inspissation
- Intussusception
- Involution
- Ischemia
- Ischemic necrosis
- Jaundice
- Karyolysis
- Karyorrhexis
- Lesion
- Leukemia
- Lipofuscin
- Liquefactive necrosis
- Malacia
- Malignant
- Melena
- Metaplasia
- Metastasis
- Mycosis
- Necrosis
- Neoplasm
- Organization (of fibrin)
- Papillary
- Pedunculated
- Papilloma
- Paraneoplastic syndrome
- Petechial hemorrhage
- Pleomorphic
- Polyp
- Purpura
- Purulent
- Pus
- Pyknosis
- Recanalization
- Regeneration
- Repair
- Sarcoma
- Scirrhus
- Septicemia
- Serous
- Sessile
- Stricture
- Suppurative
- Telangiectasia
- Teratogen
- Thromboembolism
- Thrombus
- Torsion
- Tumor
- Ulcer
- Vegetative
- Virulence
- Viscid (viscous)
- Volvulus
- Zoonosis

Appendix 2: Some anatomic prefixes

Just add “-itis” or “-osis” to construct part of the morphologic diagnosis ...

<u>Word Root</u>	<u>Organ/Tissue</u>
Arter-	artery
Arth-	joint
Bronch-	bronchi
Cellul-	subcutaneous connective tissue, fascia
Cholecyst-	gall bladder
Conjunct-	conjunctivae
Cyst-	urinary bladder
Dermat-	skin
Encephal-	brain
Encephalomyel-	brain and spinal cord
Endocard-	endocardium
Enter-	intestine
Gastr-	stomach
Gloss-	tongue
Hepat-	liver
Kerat-	cornea of eye
Lamin-	sensitive laminae of hoof
Laryng-	larynx

<u>Word Root</u>	<u>Organ/Tissue</u>
Laryngotrache-	larynx and trachea
Lymphaden-	lymph node
Mast-	mammary gland
Mening-	meninges
Meningoencephal-	brain and meninges
Metr-	uterus
Myel-	spinal cord or bone marrow
Myocard-	myocardium
Myos-	muscle
Nephr-	kidney
Neur-	nerve
Odont-	tooth
Omphal-	umbilicus
Ophthalm-	eye
Orch-	testes
Oste-	bone
Osteomyel-	bone marrow ± bone
Ot-	ear
Pericard-	pericardium

<u>Word Root</u>	<u>Organ/Tissue</u>
Periton-	peritoneum
Pharyng-	pharynx
Phleb-	vein
Pleur-	pleura
Pneumonia	lung ** note, we do not use “pneumonitis”
Pododermat-	skin of foot
Proct-	rectum/anus
Rhin-	nose
Splen-	spleen
Stomat-	mouth
Synov-	synovium
Tendin-	tendon
Tonsil-	tonsil
Trache-	trachea
Typhl-	cecum
Typhlocol-	cecum and colon
Vagin-	vagina